# World Journal of

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# World Journal of *Orthopedics*

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## FLYLEAF

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*EDITORIAL*

# How to approach the pediatric flatfoot

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## Abstract

The most difficult aspect regarding treatment of the pediatric flatfoot is understanding who needs surgery, when it is necessary, and what procedure to be done. A thorough history, clinical examination, and imaging should be performed to guide the surgeon through an often complex treatment path. Surgical technique can be divided in three categories: Soft tissue, bony, and arthroereisis. This paper will describe the joint preserving techniques and their application to treat the pediatric flatfoot deformity.

Key words: Flatfoot; Flexible; Arthroereisis; Pediatric; Planovalgus; Rigid; Pes planus

Core tip: This paper discusses the authors' approach to treating the pediatric flatfoot based on the their extensive clinical and surgical experience.

## INTRODUCTION

Pes planovalgus is a common condition in children. Despite being typically idiopathic, it may be associated with neuromuscular diseases, tarsal coalitions, and the accessory navicular syndrome. A common mistake that is made by surgeons is to consider the pediatric flatfoot as a small version of the adult flatfoot deformity. Indeed, the etiology and management of the deformity may be quite different. Often children are asymptomatic, and the main concern is the foot shape or the parents' concerns for future impairment. The most important challenge for the physician is to distinguish a condition that may have a benign natural history from those that may cause disability if left untreated. The treatment to correct the flat foot deformity can be nonsurgical or surgical. We can divide the surgical techniques used to correct this deformity into three categories: Soft tissue, bony (osteotomies and arthrodesis), and arthroereisis[1]. It is unlikely that a soft tissue procedure alone can successfully correct the deformity. For such reason

**Figure 1 Weightbearing alignment of the foot: Note the left midfoot abduction (A); note the left foot heel valgus and the "too many toes" sign (B).**

the addition of bony procedures and/or arthroereisis is warranted[2]. Subtalar arthroereisis has been introduced in recent years to treat flatfeet in the pediatric population. Arthroereisis is a joint-sparing technique that allows for correction of the deformity through proprioception and mechanical impingement[3,4].

## HISTORY AND EXAMINATION

As crucial as taking a good history is, it may be difficult to obtain information from young patients. They may refuse to cooperate or even minimize their symptoms. In these circumstances feedback from the parents may play an important role. History-taking should include pain, location, intensity, timing, functional problems, and alleviating/aggravating factors. A history of trauma or recurrent ankle sprains should also be specifically questioned[5].

Patients must be examined both sitting and standing. Flatfoot deformity may be a dynamic deformity that requires weight bearing to be unmasked during clinical examination. The patient must be observed walking barefoot to assess instability and gait asymmetry. It is also important look at the young patient's shoes to identify asymmetric wear of the soles. When the patient is weightbearing the physician should assess the presence (or absence) of the medial longitudinal arch, the prominence of the navicular, midfoot abduction (*i.e.*, "too-many-toes" sign)[6], and heel alignment (Figure 1). Children will often present with a hindfoot valgus. Heel rises and manipulation of the calcaneus can confirm flexibility or rigidity of the deformity. A flexible flatfoot is one whose heel valgus can be corrected into neutral or slight varus. Fixed forefoot supination should also be evaluated with manual reduction of the hindfoot deformity, and be addressed in surgical planning by performing a plantar flexion osteotomy of the medial cuneiform.

Finally, Achilles and gastrocnemius contractures must always be identified. As the hindfoot deforms into valgus the Achilles complex is deviated laterally and shortened, leading to contracture that, in turn, aggravates the deformity.

Over time flexible feet in pediatric patients will become more rigid. This may occur in early adolescence or young adulthood. Adaptive changes inevitably take place in the hindfoot that alter its relationship with the forefoot. In order to keep the foot plantigrade, as the hindfoot everts and the calcaneus moves into valgus, the forefoot has to supinate. The Achilles tendon moves laterally with the calcaneus, and the axis of force on the subtalar joint changes, increasing the likelihood of a contracture of the gastrocnemius-soleus. As these structural changes take place, rigidity increases, consequently making the treatment more challenging.

## IMAGING

Routine standard radiographs are not essential for diagnosis. However, they should always be ordered to more precisely assess uncharacteristic pain, decreased flexibility, and for surgical planning[7]. Weight-bearing anteroposterior (AP), lateral, and oblique views of the foot and the ankle should be obtained. Ankle radiographs may demonstrate signs of ankle instability or even overload and compromise of the medial physis. Hindfoot alignment may also be evaluated with Saltzman-view X-rays[8].

In case of an accessory navicular syndrome, an internal rotation oblique view is recommended in addition to the abovementioned views. A calcaneo-navicular coalition is best seen on the external rotation 45° oblique view, while a talonavicular coalition best visualized on the axial view[4,9].

The most commonly used radiographic measurement is Meary's angle (or talar-first metatarsal angle): The angle formed by a line through the long axis of the talus and navicular in relation to the first metatarsal axis. A flatfoot demonstrates a negative Meary's angle (apex plantar). On the lateral foot view one can measure the lateral talocalcaneal angle, the talometatarsal angle, and calcaneal pitch. On the AP foot view one can assess the talometatarsal angle and the talonavicular coverage angle[6].

Computed tomography (CT) scan and magnetic resonance imaging (MRI) are not necessary unless in

patients with uncommon causes of flatfoot deformity[4].

The CT scan (possible weightbearing) represents the gold standard for the assessment of tarsal coalitions. While many patients with a tarsal coalition show some radiographic evidence (*i.e.*, the "C" sign, talar beaking, or osseous bridging), these may sometimes be absent or unclear on standard X-rays[6]. Similarly, MRI provides additional information on fibrous coalitions as well as in cases of accessory navicular syndrome where the posterior tibial tendon could be compromised[5].

## TREATMENT

### Nonsurgical management
Young patients and their parents should be reassured that most flexible flatfeet are normal in childhood, and that the foot arch elevates over the first 10 years of age[10]. Although orthotics are diffusely prescribed to alleviate symptoms, to date there is no evidence supporting the use of orthotics to correct the deformity[11]. As a matter of fact some authors even suggest that insoles could cause more harm, leading to dependency and long-term negative psychological effects[11-13].

Orthotic supports and bracing may be appropriate for children who are symptomatic, although shoe wear modifications and other inexpensive modalities are quite appropriate for initial management. Custom molded orthotics or custom shoe wear should only be reserved for those that fail the aforementioned modalities. Stretching of a contracted Achilles tendon and physical therapy may offer symptomatic relief as well. The judicious use of nonsteroidal anti-inflammatories is a useful adjunct.

### Surgical management
Indications to surgery and the type of surgery to be performed for the pediatric flatfoot continue to represent a challenge for surgeons. Surgical management is recommended in patients complaining of pain and dysfunction. While clinical and radiographic measurements can help stage a deformity, there are no guidelines that help orthopedic surgeons navigate through the different types of surgical procedures. Some patients may present with mild pain but severe deformity, while other may show mild deformity with severe pain.

The techniques available to correct flatfoot deformity can be divided into three procedure categories: Soft tissue, bony (osteotomies and arthrodesis), and arthroereisis.

Soft tissue procedures usually involve the Achilles tendon and/or the gastrocnemius, the posterior tibial tendon, and the peroneal tendons. The aim of these procedures is to balance the deforming forces. A gastrocnemius contracture is almost always present in children with a flatfoot and must be addressed with a gastrocnemius recession. The Silfverskiold test is a useful method to distinguish between a gastrocnemius contracture and an Achilles tendon contracture (the latter requiring a tendo Achilles lengthening, open or

percutaneous). In children, the posterior tibial tendon is typically involved in the accessory navicular syndrome and requires an advancement following a modified Kidner procedure. As for the peroneals, we rarely intervene on them in the pediatric population. Only severe cases of flatfoot deformity with significant midfoot/forefoot abduction could require a peroneus brevis to peroneus longus transfer to allow good realignment and to prevent recurrence.

## ARTHROEREISIS
Arthroereisis should only be used to correct hindfoot valgus. Treatment results for children undergoing arthroereisis have been excellent, provided that the talonavicular joint is not significantly uncovered. The procedure seems to work very well in younger children who have predominantly heel valgus, presumably because they have more capacity for remodeling and adaptation of the forefoot. Once the talonavicular joint sags, particularly as seen on the lateral radiographic view, these feet seem to require more correction of the pronation deformity than a medial displacement calcaneal osteotomy can provide. If there is abduction deformity of the foot, with uncovering of the talonavicular joint, then neither the arthroereisis nor the medial displacement osteotomy are likely to be successful. The pediatric patient typically adapts to the arthroereisis very well, and the incidence of implant failure is low in this age-group. By contrast, in our experience with use of arthroereisis as an adjunctive procedure in a group of carefully selected adult patients, sinus tarsi pain warranted implant retrieval in approximately half of the cases. In children, however, implant removal has been necessary in less than 10% of the cases, probably because the foot adapts as it matures.

One cause for failure of the implant regardless of the age of the patient is inadequate correction of the forefoot. When the hindfoot is restored to a neutral position with the implant, some supination of the forefoot occurs. If the forefoot is able to compensate by increased plantar flexion of the first metatarsal, then a plantigrade foot is maintained. If the supination exceeds this adaptive ability, however, then in order to maintain the forefoot in a plantigrade position, the hindfoot has to evert during the foot flat phase of gait. This increased eversion then compresses the subtalar implant, causing pain. For this reason, an opening wedge osteotomy of the medial cuneiform is necessary if supination is excessive.

Intraoperatively, always start with the smallest trial sizers to get a feel for the position, location, and size of the tarsal canal. The range of motion of the subtalar joint should be carefully assessed with each incremental increase in the size of the dilator. The dorsiflexion of the foot now occurs more directly through the ankle joint, rather than in an oblique direction with a combined motion of dorsiflexion and eversion through the subtalar joint. If too large a prosthesis is inserted, motion of the

**Figure 2 Weightbearing lateral view X-ray of the left foot prior (A) and post (B) subtalar arthroereisis.** Note correction of Meary's angle as well as correction of the hindfoot valgus.

subtalar joint will be limited. An important point here is that the goal of this operation is simply to limit excessive eversion of the hindfoot. If the prosthesis is too small, correction of hindfoot valgus will not be obtained, and dorsiflexion of the foot through the subtalar joint will persist. The appropriate sizer should limit abnormal subtalar joint eversion and allow for a few degrees of remaining eversion only.

Once the ideal size has been determined, the definitive implant is inserted to rest between the middle and the posterior facets. On the anteroposterior view of the foot, the lateral edge of the prosthesis should be 4 mm medial to the lateral edge of the talar neck.

The range of motion of the subtalar joint, especially eversion with the foot in neutral dorsiflexion, must be reassessed. In most young patients treated for a flexible flatfoot deformity, insertion of the implant is enough to provide appropriate correction (Figure 2). The forefoot should be plantigrade, and no excessive supination of the forefoot should be present after hindfoot correction. If fixed forefoot supination is present, an opening wedge osteotomy of the medial cuneiform is an excellent procedure to correct any residual forefoot supination after correction of the hindfoot.

## MEDIAL DISPLACEMENT CALCANEAL OSTEOTOMY

The initial concept of mechanically altering the axis or position of the calcaneus to better normalize deformity

was first described by Gleich[14] in 1893. However, it was Koutsogiannis who first recognized that sliding the calcaneus medially improves outcomes in flexible pes planus[15].

The medial displacement calcaneal osteotomy (MDCO) is a powerful procedure to correct hindfoot valgus. The procedure not only restores the mechanical tripod of the heel with respect to the forefoot, but also medializes the insertion of the Achilles tendon relative to the axis of the subtalar joint[16,17].

The MDCO requires approximately 10 to 12 mm of translation (about 50% of the calcaneal width). While a dorsal translation must always be avoided, a mild plantar translation of the posterior tuberosity is often desirable to increase the calcaneal pitch angle. Once the displacement has been completed, the choice of fixation is dependent on skeletal maturity. If the physis is closed or reaching skeletal maturity, the construct can be stabilized with one 6.5-mm cannulated screw. If skeletally immature with significant growth remaining, the osteotomy can be stabilized with smooth pin fixation. Once the hindfoot is corrected, attention is turned to the forefoot. Depending on the amount of deformity, additional procedures may be added.

## CORRECTION OF THE ACCESSORY NAVICULAR SYNDROME

A painful accessory navicular is almost always associated with a flatfoot of variable degree. The symptoms asso-

**Figure 3  Postoperative weightbearing anteroposterior (A), lateral (B), and hindfoot (C) views of the left foot in a patient with a painful accessory navicular syndrome treated with a medializing calcaneal osteotomy and fusion of the accessory navicular with a screw.** Note correction of the talonavicular uncoverage (A), Meary's line (B), and hindfoot valgus (C).

ciated with this condition result from the disruption of the synchondrosis between the navicular and the accessory bone. As the synchondrosis is stressed, disruption of the attachment of the accessory navicular and thus of the posterior tibial tendon occurs. Another source of pain comes from pressure in the shoe secondary to an uncorrected pronated flatfoot.

Various degrees of deformity and flexibility of the hindfoot are associated with the accessory navicular. A painful accessory bone almost always requires surgical treatment. In addition to addressing the abovementioned condition, additional procedures are often required to correct the foot alignment. Such procedures may include a MDCO, lateral column lengthening, subtalar arthroereisis, medial cuneiform osteotomy, or Achilles tendon lengthening/gastrocnemius recession.

We prefer treating the painful os naviculare with a modified Kidner procedure and advancement of the posterior tibial tendon on the navicular using a suture anchor. However, large accessory bones can be treated with resection of the synchondrosis and fixation with a screw. This has the advantage of preserving the insertion of the posterior tibial tendon on the bone, thus providing quicker recovery and stronger repair (Figure 3). Nonetheless, the disadvantage is the potential for continued swelling on the medial aspect of the foot as well as nonunion. These complications can be decreased by generously shaving both the os naviculare and the medial pole of the navicular, to decrease the bulk of the bone on its medial aspect and expose bleeding subchondral bone.

Intraoperatively, the accessory navicular must be completely excised, taking care not to injure the posterior tibial tendon and the underlying spring ligament. Next, the medial border of the navicular must be resected until flush with the anterior edge of medial cuneiform to decrease the medial bulk. Once the bones have been modeled, the posterior tibial tendon is advanced with the foot in mild overcorrection (plantarflexion and inversion). In young children the tendon can be anchored into the bone using a sharp needle inserted directly into the navicular bone, the cuneiform, or both.

In the older children and adolescents, the use of a suture anchor is preferable. In our experience most patients require additional procedures to correct the foot. These include a gastrocnemius recession, an arthroereisis or a MDCO. These procedures should be done prior to the modified Kidner, as they will affect the tension on the posterior tibial tendon. Conversely, a cotton osteotomy to correct the fixed forefoot supination (often required in our experience) can be performed before or after the modified Kidner.

## LATERAL COLUMN LENGHTENING

Sangeorzan *et al*[18] presented a cadaveric study in 1993 using the Evans procedure and found significant improvements in talonavicular coverage, talometatarsal angle, and calcaneal pitch angle.

The indications for lengthening of the lateral column (LCL) are quite specific and include a flexible foot that is amenable to correction. In this context, correction implies that the talonavicular joint can be covered with the procedure. The lateral column lengthening procedure does not work well if the foot is stiff.

The hindfoot alignment can be corrected with either a MDCO (to correct the heel valgus) or a lateral column lengthening calcaneus osteotomy. The latter will not only correct the midfoot abduction, but also push the heel medially. A LCL through a calcaneocuboid fusion is not recommended in children.

We make a short incision over the sinus tarsi. The osteotomy is made 1 cm posterior to the calcaneocuboid joint. The position of the osteotomy is marked with a guide pin and checked fluoroscopically. Osteotomy cuts are then made on either side of the guide pin and completed through the neck of the calcaneus. A common mistake is to make the osteotomy too far posterior, causing subtalar impingement. With the osteotomy distracted, the position of the talus relative to the navicular is checked clinically and radiographically, and once positioning is corrected, the appropriate-size auto/allograft is prepared. The size of the graft in children is about 8 to 10 mm on the lateral aspect of the graft, and

**Figure 4  Preoperative anteroposterior (A) and lateral (B) views weightbearing X-rays in a child with a flexible flatfoot; on the anteroposterior view, note about 50% of talonavicular uncoverage; postoperative anteroposterior (C) and lateral (D) weightbearing views following a lateral column lengthening and cotton osteotomy.** Note the excellent correction of the talonavicular uncoverage (C) and Meary's angle (D).

should be trapezoid shaped as opposed to triangular (Figure 4). Fixation of the graft is not necessary, unless grossly unstable. Potential complications of LCL include lateral foot pain, nonunion, sinus tarsi impingement (typically when the osteotomy is too posterior), and a slight dorsal subluxation of the distal calcaneus (creating prominence of the anterior process of the calcaneus subcutaneously).

## OPENING WEDGE OSTEOTOMY OF THE MEDIAL CUNEIFORM (COTTON OSTEOTOMY)

The opening wedge medial cuneiform osteotomy is an excellent adjunct to many hindfoot correction procedures, including lateral column lengthening, MDCO, excision of an accessory navicular, and placement of an arthroereisis implant. Determining the exact indications for this procedure is not easy, because the capacity of the forefoot for plantar flexion subsequent to the calcaneus osteotomy cannot be predicted. As a general rule, if the forefoot is supinated more than 15 degrees, we add a cotton osteotomy.

The incision is made along the dorsal margin of the medial cuneiform A K-wire is inserted from dorsal to plantar in the middle of the cuneiform, directed slightly proximally. There is a tendency to make the saw cut too vertically and not along the axis of the cuneiform. If this placement is exaggerated, the osteotomy may enter the metatarsocuneiform joint. The osteotomy should be completed up to the base of the cuneiform without violating the plantar cortex that will act as a hinge. Once the cut is completed, a laminar spreader is inserted into the osteotomy, and as it is distracted, the first metatarsal is plantarflexed, correcting the metatarsal declination angle. A structural bone graft (allograft or autograft) is then carefully tamped into the osteotomy. Contrarily to the LCL graft which should be trapezoid shaped, the cotton osteotomy graft should be triangular. Most times the graft measures between 5 and 7 mm across at the dorsal base of the graft. The osteotomy is very stable once the graft is wedged into place, and fixation is not necessary.

## TARSAL COALITIONS

Tarsal coalitions can determine a rigid flatfoot deformity. Historically, coalition resection was indicated for coalitions inferior to 50% of the middle facet, whereas fusion was indicated for coalitions greater than 50%. We disagree with this philosophy and always try to perform a complete resection of any coalition. The decision is guided by the age of the patient, the severity of the deformity, the degree of stiffness, and the presence of arthritis. A CT scan (possibly weightbearing) is always indicated not only to assess the coalition, but also to identify other coalitions which are present in almost 50% of patients. The most common cause of a rigid flatfoot in a child is a talocalcaneal coalition of the

middle facet. The senior author recently presented a new technique to precisely excise the coalition[19]. A 5-cm incision is created inferior to the posterior tibial tendon, over the coalition. The coalition is identified in the interval between the flexor digitorum longus and flexor hallucis longus. The soft tissue and periosteal flap over the coalition must be elevated away from the coalition to ensure adequate visualization. Next, a 1-cm incision is made over the sinus tarsi soft spot. A guide pin (part of a system for subtalar arthroereisis) is inserted through the tarsal canal and pushed between the coalition and the posterior facet. Then, the coalition is exposed by inserting the arthroereisis sizing device over the guide wire in a lateral to medial direction through the sinus tarsi. The arthroereisis sizer will open up the coalition as it is inserted. In cases of a solid, complete coalition, a fracture occurs along the margins of the coalition. Resection can then be carried out using osteotomes and rongeurs. As the coalition is resected, the sizing guide can be advanced, further opening the subtalar joint and the coalition. With the arthroereisis guide in place, the coalition can be fully resected, allowing visualization of articular cartilage around the resected coalition.

## CONCLUSION

Pediatric flat foot deformity should be classified as rigid *vs* flexible. A combination of soft tissue and bony procedures is almost always necessary to correctly realign the foot and prevent recurrence. Too often surgeons ignore the power of a gastrocnemius recession and a cotton osteotomy when performing reconstructive surgery. A thorough examination of the foot both preoperatively and intraoperatively will help unmask a gastrocnemius contracture and/or a fixed forefoot supination.

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# Football injuries of the ankle: A review of injury mechanisms, diagnosis and management

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## Abstract

Football is the most popular sport worldwide and is associated with a high injury rate, most of which are the result of trauma from player contact. Ankle injuries are among the most commonly diagnosed injuries in the game. The result is reduced physical activity and endurance levels, lost game time, and considerable medical cost. Sports medicine professionals must employ the correct diagnostic tools and effective treatments and rehabilitation protocols to minimize the impact of these injuries on the player. This review examines the diagnosis, treatment, and postoperative rehabilitation for common football injuries of the ankle based on the clinical evidence provided in the current literature.

Key words: Management; Soccer; Football; Ankle; Injury

Core tip: Injury prevention is paramount in optimizing function and decreasing time lost from sport in footballers. Early recognition of foot and ankle injuries allows implementation of conservative measures aimed at improving function and reducing the risk of re-injury or development of concomitant pathologies. Treatment, whether conservative or surgical, requires an understanding of the mechanical component of injury (e.g., ligament tear, osteophytes), while additionally addressing the biological components affecting healing. This includes restoration of normal proprioceptive pathways through physical therapy programs while also treating the catabolic biochemical environment through

selected use of biological adjuncts including platelet-rich plasma and bone marrow aspirate concentrate.

# INTRODUCTION

Football is the most popular sport in the world, while also being associated with a high injury rate both at professional and amateur levels[1-3]. Elite soccer players experience between 13 and 35 injuries per 1000 competitive player-hours, with up to 74% resulting from direct player contact. When cause is analyzed, approximately 80% are traumatic in origin and 20% are overuse injuries[4,5]. The lower limb is most commonly affected with the ankle accounting for up to a third of all injuries[1-3,6]. At the 2004 Olympics, foot and ankle injuries were encountered in football proportionally more than any other team sport[7]. During the 2010 FIFA world cup, ankle sprains were among the most prevalent diagnoses and of these, approximately 50% prevented participation in training or competition[8]. Additionally a recent study of an English Premier League (EPL) club revealed over a four year period, 20% of injuries were of the foot and ankle with a resultant mean return to sport time of 54 d[9].

The consequences of ankle injuries include reduced physical activity and endurance levels, lost game time, and considerable medical cost[3,10,11]. Due to the frequency and debilitating nature of these injuries it is critical for trainers, therapists, and team physicians to correctly diagnose injuries as early as possible and apply the most effective treatments to return athletes to the field expeditiously.

This article reviews the mechanisms of injury and highlights appropriate examination, diagnostics, treatment, and postoperative rehabilitation for common soccer injuries of the ankle.

# ANKLE SPRAINS AND ANKLE INSTABILITY

Ankle sprains are the most common pathology accounting for up to 67% of all soccer related ankle injuries[12,13]. Analyzing ankle sprains in players from the English football league over a 2-year period, Woods *et al*[13] found the majority were sustained during player contact (59%) except for goalkeepers in whom 79% occurred during non-contact situations. In addition, Jain *et al*[9] showed a 28.6% recurrence in anterior talofibular ligament (ATFL) injury in their EPL cohort. In a typical sprain, forced ankle inversion-supination precipitates tearing of the ATFL to varying degrees. Video analysis of ankle injuries in professional soccer players has shown that direct contact with a laterally directed force on the medial aspect of the lower leg just before or at foot strike can causes the player to land with the ankle in this vulnerable inverted position[14]. The injury tends to be more severe if the affected foot is planted and weight-bearing at the time of impact[6]. The peroneal tendons are also at risk in a combination of mechanical stretch and overload as they attempt to evert the foot back into neutral alignment.

Mechanical instability occurs when ligaments fail to remodel to normal length, allowing motion beyond normal physiological limits. The ankle joint capsule and soft tissues about the joint are often stretched or torn at the time of injury, disrupting the proprioceptive nerve fibers that run through them. This can produce a functional instability where the player may be mechanically stable but unable to maintain balance when in unilateral foot stance[15]. Both mechanical and functional instability may be present independently or in combination in any player and if untreated can potentiate additional sprains and the development of chronic ankle instability[16].

Clinical examination may be difficult in the immediate period following an acute injury. If there is concern for a ligamentous ankle injury, consideration can be given to delaying a definitive examination for up top 5 d in the off season as this permits the partial resolution of swelling and inflammation. van Dijk *et al*[17] have reported a diagnostic sensitivity of 96% with a delayed assessment protocol. The anterior drawer and valgus stress test can be useful in the delayed or chronic setting, however, these tests have been shown to have limited sensitivity and significant variability in differing examiners hands[18]. The modified Romberg test can demonstrate proprioceptive deficiencies of the ankle, indicating the presence of functional instability[19].

During the active playing season consideration should be given for early and accurate diagnosis. Clinical diagnosis will direct further diagnostic tests including plain radiographs, magnetic resonance imaging (MRI) and computed tomography (CT). Ankle sprain is not a benign injury and up to 75% of these injuries will have an associated soft tissue pathology[20]. MRI has the sensitivity required to detect these associated injuries including osteochondral lesions (OCL's), tendinous and syndesmotic tears, and associated fractures. Plain radiographs may miss up to 50% of OCL's of the talus following ankle sprain and CT scan, while useful in evaluating bony injury, lacks the sensitivity for diagnosis of soft tissue pathology[21].

Specific clinical tests can direct the radiographic diagnostic intervention. The so called "high ankle sprain" or syndesmotic injury is typically identified by pain over the anterior inferior tibiofibular ligament (AITFL) and interosseous membrane. More specific and sensitive evaluations are the squeeze test of the mid-fibula and resisted external rotation test respectively[22,23].

**Figure 1  Plain radiographs of the left (A) and right (B) ankles of a single patient in the coronal plane.** Both ankles are under eversion stress. The right ankle was symptomatic. Only subtle syndesmotic gapping and widening of the medial clear space can be appreciated on the right compared to the left ankle.



**Figure 2  Coronal fast-spin echo proton density magnetic resonance images of the right ankle seen in Figure 1.** Disruption and remodeling of the anterior inferior tibiofibular ligament (A; yellow circle) and interosseous ligament (B; yellow circle) can be appreciated.



**Figure 3  Axial fast-spin echo proton density magnetic resonance images of the right ankle seen in Figures 1 and 2.** Disruption and remodeling of the anterior inferior tibiofibular ligament (yellow arrow) can be appreciated.

Augmenting clinical assessment and subtle injuries diagnosed by plain radiograph with MRI is recommended to fully evaluate the injury with a reported accuracy of 97% (sensitivity: 100%; specificity: 97%) (Figures 1-3)[22,24].

The peroneal tendons are injured in up to 25% of acute ankle sprains acting as secondary stabilizers[20]. Resisted eversion is useful to assess the integrity of the peroneal tendons[25]. MRI is again useful in detecting any peroneal pathology and dynamic ultrasound can be a useful adjunct in detecting subtle peroneal injury[26,27].

The so called "low ankle sprain" or pain in the sinus tarsi secondary to a torn interosseous ligament in the acute phase and scar formation in the chronic phase can be examined by direct local pressure in the sinus tarsi while inverting the mid-tarsal joint. Radiographic evaluation including MRI and ultrasound are often less sensitive and specific for the low ankle sprain than for other ankle pathology and a small local anesthetic injection to the area can often be helpful in elucidating the source of pain.

The majority of simple ankle sprains heal with non-operative treatment, however, there is no consensus on the ideal rehabilitation protocol[20]. Early mobilization followed by phased rehabilitation is advocated by most authors as beneficial in minimizing time lost[28-30]. A multicentre study of 584 patients suggested there is faster recovery with a short period of immobilization in a below the knee cast or removable boot when compared to treatment in a compression bandage[31]. It must be noted that no information was provided on additional interventions, the study utilized a postal questionnaire and there was a 17% drop-out rate. But overall, the results indicate initial immobilization can be beneficial. Conversely, prolonged immobilization of greater than 2

**Figure 4  Illustrations of the Brostrom (A) and Modified Brostrom-Gould (B) surgical technique for lateral ligament reconstruction of the ankle.** Reproduced, with permission, from Prisk *et al*[108].



**Figure 5  Illustration of the hybrid anatomic lateral ligament reconstruction**[27]. A tendon autograft taken from the peroneus longus has been docked in the talus and distal fibula and remaining anterior talofibular ligament fibers have been sutured over the reconstruction, theoretically allowing proprioceptive fibers to aid in regaining functional stability. Illustration copyright of and reproduced with permission from Kennedy JG, MD. Reproduction without express written consent is prohibited. Reproduced, with permission, from Kennedy *et al*[40].

wk has a detrimental effect on muscles, ligaments, and joint surfaces and may result in longer return to play time[32].

A rehabilitation protocol should be divided into specific stages: Acute and subacute pain and swelling control; range of motion and strengthening exercises; soccer specific functional training; and prophylactic intervention with balance and proprioception stimulating exercises. Upon return to play, continued proprioceptive training is vital to minimize recurrence[33-36]. Semi-rigid orthoses and air-cast braces may help prevent ankle sprains, especially in athletes with a history of recurrent instability[37-39]. Bracing has a mechanical advantage over simple taping, as tape loses its ability to restrict inversion and eversion approximately 20 min after starting activity[39].

Surgery is indicated in patients with chronic mechanical instability. Traditionally, two forms of repair are considered: An anatomic reconstruction such as the Brostrom or Gould modification (Figure 4), or a non-anatomic checkrein tenodesis such as the Chrisman-Snook procedure[20,40]. Anatomical repairs appear to produce better outcomes and there is additional concern that some checkrein procedures can restrict subtalar motion and prevent normal agility on playing surfaces by altering hindfoot biomechanics[38,41-43]. As over 90% of patients with chronic ankle instability have additional intra-articular lesions, arthroscopic ankle evaluation, and treatment where necessary, can be performed at the same time as open lateral ankle ligament repair[44,45].

With acute ankle ligament instability the traditional treatment paradigm of triple phase physical therapy and avoiding surgical intervention has been recently questioned. A report from van Dijk demonstrated superior results with acute surgical repair in a cohort of athletes[46]. It consisted of a subgroup analysis in which only a single surgeon series of acute operative repair was conducted. Objective instability, as defined by a positive talar tilt on stress radiographs or positive anterior drawer sign, was significantly less when compared to non-operative treatment. Because increased objective instability is a predictor for future ankle sprains, an acute reconstruction may be preferred in professional athletes[47,48]. The outcome of a recent consensus meeting also suggested a role for selective operative treatment in athletic populations[43].

Surgical outcomes in acute and chronic lateral ankle ligament repairs are generally good and most athletes are able to return to their pre-injury level of function[20]. Kennedy *et al*[40] reported an anatomic repair augmented with a portion of peroneus longus used as an ATFL checkrein in 57 athletes, including 11 soccer players (Figure 5). Although all patients achieved mechanical stability, five patients did not return to their pre-injury level for reasons unrelated to their ankle. Maffulli *et al*[49] recently reported outcomes of ankle arthroscopy and Broström repair in 38 athletes at an average 8.7 years follow-up. Laxity grade, American Orthopaedic Foot and Ankle Society Ankle-Hindfoot scores, and Kaikkonen scales all improved significantly at last follow up. Return to high contact sport was allowed six months postoperatively. Fifty-eight percent of patients returned to their pre-injury level of activity, 16% decreased their

**Figure 6 Coronal fast-spin echo proton density magnetic resonance image demonstrating an uncontained osteochondral lesion of the medial talar dome.**



**Figure 7 Intraoperative photograph of an autologous osteochondral graft transplanted into the medial talar dome.** Access was achieved *via* a medial malleolar osteotomy. The graft was gently tamped into position until flush with the surrounding surface.

activity but maintained activity in less demanding sports, and 26% abandoned sport participation but remained physically active. Arthroscopic assisted techniques are emerging and may play a role in some cases, but its exact role has yet to be determined and open techniques are currently considered the gold standard[50].

Acute syndesmotic injuries (high ankle sprains) are less common than lateral ankle sprains and if missed can potentiate persistent morbidity and early degeneration of the ankle joint[51,52]. Mild syndesmotic sprains can be managed conservatively with protected weight-bearing and functional rehabilitation. Compared with lateral ankle sprains, syndesmosis injuries typically require longer rehabilitation programs[52]. A recent study of National Football League players, however, did indicate an earlier return to play (4-6 wk) is possible with milder injuries[53]. Due to the potential for prolonged rehabilitation, some clinicians advocate surgical intervention in professional athletes with mild sprains to expedite a return to play[52].

Surgical intervention is necessary in severe acute injuries where there is tibio-fibular diastasis. Screw fixation has traditionally been utilized with most physicians advocating screw removal between 7 and 12 wk postoperatively[51,52]. Non-absorbable suture-button fixation devices have more recently emerged as a fixation technique[54]. They have the advantage of obviating hardware removal and can allow earlier weightbearing[55]. Chronic syndesmotic injuries can be treated with screw fixation, arthrodesis and arthroscopic debridement. While there are no papers specifically assessing outcomes in an athletic populations, a recent systematic review and meta-analysis reported screw fixation as the most successful treatment option[51].

## OCL's

OCL's of the ankle have an incidence between 50% and 70% of all acute ankle sprains and fractures[56-59]. Unfortunately, cartilage injuries have a poor spontaneous healing response. Therefore the role of surgical management involves the repair an acute lesion when possible, initiating fibrocartilage formation with

marrow stimulation techniques in small lesions, with consideration given to cartilage transplant or other modalities for larger lesions. The ultimate aim is to return the athlete to their pre-injury level of play.

Symptoms of an OCL may be mechanical and include clicking or locking of the ankle. Typically however, talar OCL's present localized deep joint pain without any mechanical symptoms. Physical examination may reveal swelling, localized tenderness along the joint line, and limited motion. Plain radiographs may miss up to 50% of OCL's and CT is only useful in obtaining the degree of bone injury as both are unable to assess overlying cartilage[21,60]. Magnetic resonance imaging is recommended for a definitive diagnosis and T2 Mapping sequences can be useful with increased sensitivity to cartilage change (Figure 6)[61].

Lesion size and location determine the most appropriate treatment strategy. Most OCL's will not heal with conservative treatment and therefore surgery is typically recommended[62]. Reparative surgical techniques such as bone marrow stimulation for lesions of less than 15 mm in diameter yield good functional outcomes in short term follow-up[56,63]. Larger lesions are best treated with replacement strategies including autologous osteochondral transplantion (AOT) (Figure 7) or other techniques including autologous chondrocyte implantation[64-66].

Both reparative and replacement strategies have been evaluated in regards to return to soccer however the evidence is limited in both groups. Saxena and Eakin[57] evaluated functional activity after surgical management of OCL's in 44 athletic subjects of which 18 were considered high level, including six soccer players. At medium term follow-up (32 mo), 17 of 18 high level athletes had returned to their pre-injury level and the other subject had not returned for personal reasons. The mean time to return to sport was 15 wk after marrow stimulation and 19 wk after bone grafting (not AOT's). More recently, Paul *et al*[64] assessed sports activity after talar AOT's in 131 patients with a mean age of 31 years. Seventy percent were either very satisfied or satisfied

by the procedure and 85% required no analgesia when currently involved in sports. While 20% were involved in competitive sports preoperatively, only half of these were still competing postoperatively. Age at time of injury may be a factor in a person's ability to return to sport. For example, Hangody et al[65] reported a combination of talar and knee OCL's in 354 competitive athletes including 67 soccer players and four Olympians. They found that 63% were able to return to their same activity/sporting level although most were under the age of 30 years. Overall, 9% had to give up sports completely.

The long-term outcome of cartilage repair surgeries has prompted significant interest in biologic augmentation of cartilage healing in the sports world. Concentrated bone-marrow aspirated (CBMA), typically obtained from the players iliac crest at the time of surgery, is a source of mesenchymal stem cells and growth factors. This can be delivered directly to the OCL during arthroscopy and may potentially enhance the healing environment. When combined with microfracture in a large animal model, the resultant cartilage of the CBMA group demonstrated greater type-Ⅱ collagen, proteoglycan and glysosaminoglycan content consistent with a more normal cartilaginous architecture[67]. At this time no long term data exists to support the use of CBMA and early return to sport following talar OCL surgery.

Platelet-rich plasma (PRP) is an autologous blood product that contains many growth factors that may promote cartilage repair. A recent review of PRP basic science literature found that it promotes chondroctye and mesenchymal stem cell proliferation, type Ⅱ collagen deposition, and proteoglycan deposition[68]. Results also indicated that PRP may increase chondrocyte viability, promote migration and chondrogenic differentiation of mesenchymal stem cells, as well as inhibit the effects of inflammatory cytokines. Mei-Dan et al[69] recently compared intra-articular injections of PRP and hyaluronic acid (HA) for non-operative management of talar OCL's in a randomized trial of 30 patients. Fifteen patients were randomized into each group, treated with three consecutive injections of either PRP of HA, and followed for 28 wk. American Orthopaedic Foot and Ankle Society Ankle-Hindfoot scores, visual analogue scale (VAS) pain, VAS stiffness, VAS function, and subjective global function scores all improved significantly more in the PRP group than the HA group. Current clinical studies are limited for PRP, and formal randomized human trials are required to determine the true *in-vivo* effect.

Postoperative care is dependent on the chosen treatment. After marrow stimulation, the patient is initially non-weight-bearing in a soft leg cast for two weeks during which ankle pump exercises are performed three times daily. At two weeks, the patient is placed in a CAM boot and encouraged to start range of motion exercises. At six weeks, patients commence weight-bearing starting with 10% of their body weight, increasing by 10% daily

until full weight-bearing is achieved. Formal rehabilitation concentrates on balance, joint proprioception, and stabilization. Strengthening and sport specific exercises begin at 10 wk. After an AOT procedure, patients are non-weightbearing for six weeks followed by two weeks of progressive weight-bearing as above. At eight weeks, formal physical therapy commences and sport-specific training introduced at 12 wk. Return to soccer is typically at six months following surgery.

## ANTEROLATERAL IMPINGEMENT

Anterolateral impingement syndrome (ALI) typically manifests as chronic anterolateral ankle pain following an ankle sprain. It is thought to result from the entrapment of hypertrophic soft tissues or torn and inflamed ligaments in the lateral gutter and anterolateral ankle joint[70]. Following tears of the ATFL, AITFL, and/or CFL, repetitive motion after incomplete healing can lead to inflammation and subsequent synovitis with scar tissue formation[71]. Mild sprains with minimal capsular tearing may also produce an intraarticular hematoma, the reabsorption of which by synovium in the lateral gutter may induce a reactive synovitis[71,72].

Players typically present with chronic ankle pain, limited dorsiflexion, and swelling after activity[73,74]. Tenderness on palpation of the anterolateral gutter is characteristic. ALI can be distinguished from anteromedial impingement (AMI) if pain is elicited on palpation lateral to peroneus tertius[74,75]. Symptoms must also be differentiated from sinus tarsi syndrome and a diagnostic injection of local anesthetic is useful in deducing this.

While standard radiographs are effective in diagnosing the presence of anterolateral osteophytes, MRI can reveal soft tissue impingement[74-76]. However, as false-negative results have been reported with MRI, arthroscopy is generally advocated as the definitive diagnostic and therapeutic modality[71,77].

Initial treatment involves physical therapy modalities with deep tissue massage and other techniques to reduce inflammation. If after one month of therapy no improvement is noted an ultrasound guided injection to the soft tissue impingement with a combination of low dose steroid and local anesthetic may be useful. Surgical treatment is reserved for recalcitrant cases. During the playing season players can be treated with conservative modalities and surgery may be delayed until the off season. Surgical management includes arthroscopic excision of a pathologic fascicle of the ATFL if present as well as hypertrophic synovium in the lateral gutter (Figure 8). At the end of the procedure range of motion of the ankle is checked under direct arthroscopic visualization to ensure no impingement remains during full dorsiflexion[71].

Following surgery, the player should remain non-weightbearing for two days. Dorsi- and plantarflexion exercises should commence one day postoperatively to minimize arthrofibrosis. Weight bearing is increased as tolerated. Physical therapy begins after one week and

**Figure 8 Arthroscopic image of soft tissue impingement the later gutter of the ankle joint.** ALI: Anterolateral soft tissue impingement including cicatraized tissue, entrapped lateral ligaments, and hypertrophied synovium; T$_A$: Talar dome; T$_C$: Tibial cartilage.

soccer-specific training is recommended at 2-3 wk.

There is no published outcome data in a soccer-specific cohort following surgical management for anterolateral ankle impingement; however, reported outcomes in general athletic populations are considered very good[78,79]. In a series of 11 elite dancers, Nihal *et al*[80] reported postoperative improvement in all performers with 9 returning to full function at a mean of 7 wk. DeBerardino *et al*[79] reported a series of 60 athletes with chronic anterolateral soft-tissue impingement following ankle inversion trauma treated with arthroscopic debridement. At a mean of 27 mo follow-up, 85% of patients had an excellent outcome on the West Point Ankle Score. Average functional results of the 12 m single-leg hop test was 94% of the time obtained compared to the unaffected side. Eighty-three percent of patients experienced good to excellent subjective pain relief.

## ANTEROMEDIAL IMPINGEMENT

First termed footballer's ankle by McMurray[81] in 1950, AMI is a common cause of ankle pain in soccer players. This is generally a bony rather than soft-tissue impingement and causative factors include direct trauma, recurrent microtrauma, and chronic ankle instability[73,81-83].

Recurrent stressing of the ankle at the extremes of motion was previously thought to induce bone spur formation and soft tissue proliferation due to traction of the capsule[74,75]. Current thoughts on the actual pathogenesis are less clear. Cadaveric analysis has found the anterior joint capsule to be attached more proximally than the site of the tibial spur, and arthroscopic evaluation generally identifies the osteophytes to lie within the joint capsule[84-86]. Microtrauma from recurrent impact of a football on the anterior ankle may also contribute to osteophyte formation[82]. Thickened soft tissue can be compressed between talar and tibial osteophytes (kissing lesions) with ankle dorsiflexion causing focal inflammation and pain[75,85].

AMI commonly presents as anteromedial ankle pain, swelling after activity, and sometimes limited dorsiflexion[73]. Tenderness with palpation medial to the tibialis anterior tendon is considered indicative of AMI[75]. Forced hyperdorsiflexion does not always provoke the players typical pain[75].

The diagnosis of AMI is usually confirmed by plain film radiology[87]. Anterior tibial osteophytes can be seen with standard lateral views. An oblique AMI view (45° craniocaudal, 30° external rotation of the leg with the ankle in full plantarflexion) is recommended when plain radiographs are negative as it can provide specific visualization of anteromedial osteophytes (Figure 9)[75]. CT may aid in confirming the diagnosis and MRI can identify soft-tissue impingement, soft tissue injuries, or OCL's[76].

Ultrasound-guided corticosteroid injections can reduce the patients symptoms temporarily, however, definitive treatment is typically achieved surgically[84]. This is in contrast to ALI which will often respond to conservative therapy. Operative treatment involves removal of any cicatrized soft tissue and synovial hyperplasia from the joint capsule and a thorough osseous resection of the anterior distal tibia and talar neck. Careful resection to the anterior border of the medial malleolus is vital. Range of motion assessment under arthroscopic visualization is advised to confirm an adequate debridement. The postoperative treatment course is the same as previously described for ALI.

The largest reported series for surgical management of AMI involved 41 patients of which 16 were active soccer players and one retired professional player[88]. Overall, 93% were satisfied with their outcome. For the athletic population, return to play was 7 wk on average, with longer recovery times in those that required additional procedures, specifically lateral ligament reconstruction (15 wk) and microfracture for OCL's (14 wk). All but one of the athletic cohort (33 of 34 patients) returned to their same level of play indicating an expectation of return to play following this procedure[40].

## POSTERIOR ANKLE IMPINGEMENT

Posterior ankle impingement (PAI) is characterized by posterior ankle pain with plantarflexion. Causative pathologies include a prominent posterior talar process (os trigonum), fracture of the lateral tubercle of the posterior talar process, compression of posterior soft tissues, traction on the posterior talofibular ligament (PTFL) and posterior capsule, and flexor hallucis longus (FHL) tendonitis or tenosynovitis. Repetitive plantarflexion, as seen with ball striking, can precipitate overuse injury[89-91].

Pain from ankle hyperplantarflexion is due to compression of the soft tissue or bony structures between the posterior aspect of the distal tibia and the calcaneus. Zwiers *et al*[75] described a simple clinical test where rapid passive hyperflexion of the ankle was combined with a rotational grinding motion at maximal plantarflexion.

**Figure 9  Standard lateral views about anterior tibial osteophytes.** Lateral plain radiograph of a right ankle with clinical suspicion of anteromedial impingement (A). An anteromedial radiographic view with 30 degrees of external rotation of the same ankle demonstrated an osteophyte on the anteromedial aspect of the distal tibia (B) that could not be appreciated on the lateral view.



**Figure 10  Lateral plain radiograph of a right ankle revealing an os trigonum, which was clinically symptomatic, as well as anterior tibial and talar osteophytes.**

They found a negative test could exclude PAI. Injection of local anesthetic can further aid in confirming the diagnosis.

Maximal ankle dorsiflexion can also reproduce pain due to tension within the posterior joint capsule and PTFL. Depending on the exact underlying pathology, pain can be elicited with focal palpation of the PTFL, transverse tibiofibular ligament, posterior inferior tibiofibular ligament (PITFL), CFL within the posterolateral gutter, or FHL.

Plain film radiology can help confirm PAI. The lateral ankle view is most useful in detecting osteophytes, calcifications, loose bodies, chondromatosis, and retro-calcaneal bursitis (Kager's triangle) (Figure 10). To differentiate between a hypertrophied posterolateral talar process and an os trigonum, a lateral radiograph with the foot in 25° of external rotation in relation to the standard lateral radiograph is useful[75]. Further assessment with MRI may identify associated flexor tendon injury, most commonly FHL[92,93].

Conservative treatment is initially recommended for most patients with additional consideration given to ultrasound-guided corticosteroid injections for more severe cases[94]. Surgery can be offered for recalcitrant conditions. If bone impingement is determined as the underlying cause, we advise a single diagnostic injection of local anesthesia and caution against multiple injections as they are ultimately associated with pro-longed postoperative recovery[95]. Elite soccer players require prompt return to play, and surgery should be considered early in the treatment algorithm[96,97].

While arthroscopic procedures can permit earlier rehabilitation than traditional open procedures there is concern regarding the potential risk of neurovascular injury. Several studies have shown that the rate of nerve injury in patients treated with posterior ankle arthroscopy is lower than that found in anterior arthroscopy as long as care is taken to avoid structures medial to the FHL tendon[97-99].

Following posterior ankle arthroscopy players remain non-weightbearing in a compression bandage for 24-28 h. Weight bearing is subsequently increased as tolerated until full-weight bearing by one week[95,100]. Early range-of-motion exercises help prevent cicatrization and stiffness. Physiotherapy is focused on restoring strength and range of motion.

A recent current concepts review illustrates the technique for posterior ankle arthroscopy and mana-gement of posterior impingement and co-existing pathologies[98]. As for anterior impingement syndromes, arthroscopic treatment is very effective at restoring an athlete's ability to return to competitive sport with a very low incidence of postoperative complications[95,96,99,100]. Calder *et al*[95] reported on hindfoot arthroscopy for 27 professional soccer players experiencing bone and/or soft tissue impingement. Overall, the mean time to return to training was 34 d and return to playing was 41 d. Recovery, measured as return to training, was faster in cases of soft tissue impingement (mean, 28 d) compared with osseus injury (mean, 40 d). The authors reported only one non major complication of portal leakage which resolved with two weeks rest[95].

## CONCLUSION

Preventing injuries and minimizing lost playtime is the main goal for physicians and trainers and can be

achieved *via* accurate diagnosis, effective treatment, and proper rehabilitation or postoperative course.

While most injuries in soccer occur as a result of player-to-player contact, a significant number of non-contact injuries also occur. The risk of suffering from non-contact lower extremity injuries may be lowered by implementing in-season neuromuscular training programs aimed at enhancing ankle and knee proprioception. Effective warm-up programs with an emphasis on stretching, regular cool down at the end of training or competition, sufficient recovery and rehabilitation programs, proper equipment, and good field conditions can all contribute to injury prevention[9].

Cleated shoe wear provides traction on the field while running and cutting but high shoe-surface traction has been suggested as a culprit for many ankle injuries. Longer cleats should be avoided due to the increased shoe-surface traction[101,102]. There are inherent advantages and disadvantages to both natural grass and artificial turf. Higher frictional resistance and shoe-surface traction, which is the case in turf fields, correlates with increased performance, but also increases the incidence of injuries[103]. Artificial turf also increases plantar pressures, potentially mediating metatarsalgia and stress fractures[104,105]. Natural grass has a lower friction coefficient than artificial turf but certain species are associated with increased risk of injury[106]. Warm conditions also harden the ground, resulting in increased shoe-surface traction and risk of injury[106]. Preventive measures including selecting appropriate cleats length and softening the field through watering should be considered.

Age and gender represent important risk factors for soccer players[107]. As players age, they should devote more time to appropriate warm-up, stretching and strengthening routines in order to prevent injury. Adhering to a well-designed training program throughout the season and treating injuries promptly and adequately will allow the soccer player to make the most out of the season and minimize lost play time.

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# Mechanical and cellular processes driving cervical myelopathy

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## Abstract

Cervical myelopathy is a well-described clinical syndrome that may evolve from a combination of etiological mechanisms. It is traditionally classified by cervical spinal cord and/or nerve root compression which varies in severity and number of levels involved. The vast array of clinical manifestations of cervical myelopathy cannot fully be explained by the simple concept that a narrowed spinal canal causes compression of the cord, local tissue ischemia, injury and neurological impairment. Despite advances in surgical technology and treatment innovations, there are limited neuro-protective treatments for cervical myelopathy, which reflects an incomplete understanding of the pathophysiological processes involved in this disease. The aim of this review is to provide a comprehensive overview of the key pathophysiological processes at play in the development of cervical myelopathy.

Key words: Cervical myelopathy; Cervical spine; Neck pain

Core tip: The pathophysiology of cervical myelopathy involves a combination of mechanical static and dynamic factors, triggering a cascade of biomolecular changes to include ischemia, excitotoxicity, neuroinflammation and apoptosis. Development of targeted neuro-protective treatment strategies, specifically modulating these molecular pathways, may optimize neurological recovery following surgical decompression. The aim of this review is to provide an overview of the pathophysiological processes at play in the development of cervical myelopathy.

## INTRODUCTION

Cervical myelopathy is the most commonly reported spinal cord pathology globally in the > 55 years age cohort[1-3]. Twenty-five percent of spinal cord dysfunction in the Unitd Kingdom is caused by cervical spondylotic myelopathy[4]. The exact biostatistics in relation to the disease remains unknown, however there exists a male preponderance at a ratio of 2.7:1[5]. Originally detailed by Stookey[6] in 1928, compression of the cord by cartilaginous degenerative disc nodules was the primary pathogenic mechanism attributed to this disorder. The key neuropathological characteristics of cervical myelopathy include cystic cavitation, gliosis, Wallerian degeneration of descending and ascending tracts, and loss of anterior horn cells[7,8].

Clinically, cervical myelopathy presents as progressive spinal cord impairment. Symptoms include distorted proprioception, weakness and paresthesia of the hands, spasticity of the lower limbs with resultant gait disturbance, and pyramidal and posterior cord dysfunction[9]. In addition to motor and sensory sequelae, neuropathic pain and functional limitations can be devastating, resulting in significant physical and socioeconomically restrictions for previously healthy individuals. Thus, there exists an urgent requirement to clearly define the pathobiology of the cervical myelopathy to assist discovery of translational interventional strategies.

Researchers have previously postulated that a narrowed spondylotic spinal canal causes compression of the enclosed cord potentially causing local neural ischemia and neurological impairment. The aetiology of cervical cord compression is multifactorial with contributions from disc herniation alone; and osteophytic spur overgrowth in the spinal canal referred to as spondylosis. A decrease in vertebral canal diameter as a consequence of age-related degenerative changes of the joints, intervertebral discs, and ligaments of the cervical vertebrae, significantly contributing to cord compression[1]. Infolding of the ligamentum flavum and facet joint capsule can distort spinal canal anatomy and foraminal dimensions[10]. However, this simple anatomic model has been challenged by falling short of explaining the array of clinical presentations in cervical myelopathy, specifically development of neurological impairment in the absence of static spinal cord compression[11]. Whilst the aetiology of cervical myelopathy is thought to be multifactorial including contributions from age-related degeneration, mechanical stress and biochemical factors, a genetic predisposition has been revisited, due to recent evidence of familial clustering in population studies[12].

Despite advances in the surgical management of cervical myelopathy in addition to earlier diagnosis facilitated by advances in diffusion tensor magnetic resonance imaging (MRI) and kinetic MRI, a significant proportion of patients suffer residual neurological sequele as a consequence of irreversible cord injury[13-17]. Thus, implementation of neuro-protective interventions as an adjunct to surgical decompression may optimise patient outcomes for cervical myelopathy.

## THE ANATOMIC BASIS OF CERVICAL SPONDYLOTIC MYELOPATHY

In the context of progressive age-related degenerative changes, clinically significant cervical spondylotic myelopathy typically presents in late adulthood. These changes include cervical disc degeneration, osteophytic spur formation and transverse bar formation and osteoarthritic facet hypertrophy. Age-related degenerative changes with respect to supporting ligaments include posterior longitudinal ligament calcification and ligamentum flavum thickening[18-22]. The concept of dynamic stenosis represents progressive impingement on the spinal canal, resulting in transient spinal cord compression during physiological cervical range of motion. However, in some cases, dynamic stenosis may evolve into static compression of the spinal cord, manifesting clinically as classic cervical spondylotic myelopathy.

## MECHANISMS OF CERVICAL MYELOPATHY

Although the specific pathophysiological mechanisms contributing to cervical myelopathy remain ambiguous, it is considered a manifestation of long-tract signs resulting from multifactorial compression on the cervical spinal cord[10]. Key factors in the development of cervical myelopathy are categorized as either static or dynamic mechanical factors, resulting in direct injury to neurons and glia, which in turn triggers a cascade of secondary cellular mechanisms (Table 1)[1,23].

### *Static mechanical factors*

**Spondylosis and disc degeneration:** Progressive cervical spondylotic changes are a key feature in the pathogenesis of cervical myelopathy due to an increase in the compressive extrinsic canal forces (Figure 1). With advancing age, the intervertebral discs cannot bear load due to a combination of factors to include medial splitting of the disc and gradual loss of the nucleus pulposus. It is this disc degeneration that stimulates a cascade of progressive changes resulting in cervical canal stenosis and the development of cervical spondylotic myelopathy[24]. Anterolateral uncovertebral process flattening increases the load imposed on vertebral articular cartilage. This promotes the

| **Table 1  Pathophysiological factors involved in cervical myelopathy** |
| --- |
| Static factors |
|   Spondylosis |
|   Disc degeneration |
|   Ossification of the posterior longitudinal ligament |
|   Ossification of ligamentum flavum |
|   Congenital stenosis |
|   Other acquired congenital pathology, *e.g.*, tumors, calcification |
| Dynamic factors |
|   Changes in neck flexion/extension - narrow spinal canal |
| Biomolecular factors |
|   Ischemic injury - compression of spinal cord vasculature |
|   Glutamate - mediated excitotoxicity |
|   Oligodendrocyte and neuronal apoptosis |



**Figure 1  Degenerative changes that contribute to extrinsic compression of the spinal cord in cervical myelopathy.** OPLL: Ossification of the posterior longitudinal ligament.

development of osteophytic spurs. A combination of osteophytic spur overgrowth ventrally and buckling of the ligamentum flavum posteriorly, can lead to a circumferential mechanical compression of the spinal cord inducing cervical myelopathy[25]. Additionally, the conformational change in bony structures can compromise the integrity of the vertebral artery and spinal nerve leading to demyelination of ascending and descending tracts and chronic pain[1]. Radiological assessment is key, as it assists differentiation of disk-related neck pain, radiculopathy, and myelopathy. Imaging, in the context of pre-operative planning, also aids localization of the site-specific disease[26]. Compared with other radiological studies MRI provides an overview of both bony and soft tissue architecture including intervertebral discs, supporting ligaments, and neural structures. Dynamic weight bearing (kinetic) MRI has been hailed as the gold standard modality for cervical spondylotic myelopathy[15,27]. Myelopathy is represented radiographically with increased cord signal on T2-weighted MRI and a decreased signal on T1-weighted imaging[28]. Diffusion tensor imaging (DTI) improves pathologic specificity by measuring directional diffusivities, which quantify water diffusion parallel and perpendicular to the white matter tracts[16,29]. A recent study of the role of DTI in cervical spondylotic myelopathy suggested that DTI may elucidate pathology of the spinal cord before the development of T2 hyperintensity imaging and thus may be a superior imaging modality in the future[13].

**Ectopic ossification of spinal ligaments:** Ectopic ossification and calcification of spinal ligaments has also been attributed to the development of spinal canal stenosis and the onset and aggravation of myelopathic symptoms[1,30]. Ossification of the posterior longitudinal ligament (OPLL) is a common pathology[20,31]. Pathological compression by OPLL commonly presents with severe myelopathy and can lead to quadraparesis[32]. The natural course of OPLL suggests a degenerative process as a consequence of environmental factors such as accumulative extrinsic loading on the spine and genetic predisposition. *In vivo*

studies involving the Yoshimura mouse, which develops ossification of the posterior ligaments akin to human OPLL, has identified a mutation within the nucleotide pyrophosphatase (*NPPS*) gene[31]. Defective NPPS results in reduced production of pyrophosphatase, permiting ectopic ossification of spinal ligaments. Recent genetic studies involving the human NPPS locus have identified female sex, age (< 35 years) and severe ossification (> 10 ossified vertebra) correlated with susceptibility to and severity of OPLL[33]. Further studies involving the Zucker fatty rat have propose a role of a missense mutation in the leptin receptor gene in the promotion of ectopic ossification[34]. Thus, it is credible that NPPS and leptin receptor genes function in synergy in the pathogenesis of ectopic ossification and myelopathy. In light of these novel findings, there is a requirement to investigate this association in human OPLL.

It has been postulated that stenosis and subsequent cord compression may occur as a consequence of ossification of ligamentum flavum (OLF)[21]. The major diagnostic difference is grossly anatomical. In OLF, calcifications allow ligamentum flavum to fuse with adjacent lamina, whereas in OPLL the ligament adheres to the posterior aspects of the vertebral bodies and intervertebral discs. Despite this anatomical disparity, studies report the role of bone morphogenic protein-2 and transforming growth factor-β in matrix hyperplasia and ossification in both OPLL and OLF[35].

**Congenital spinal canal stenosis:** It is reported that cervical stenosis has an incidence of approximately 4.9% of the adult population[36]. Cervical stenosis is influenced by two factors: (1) degenerative cervical spondylosis; and (2) a developmentally narrow canal. Recent cadaveric studies have revealed that females patients and individuals over 60 years old, have narrower canals[37]. The presence of congenital or developmental canal stenosis is highly predictive of later myelopathic cord dysfunction[1]. The normal anteroposterior dimenions of the subaxial canal has been reported as 17-18 mm between C3 and C7[38,39]. Numerous MRI studies have concluded that a congenital sagittal spinal

**Figure 2 Determination of the anteroposterior compression ratio in patients with cervical myelopathy.** AP = b/a × 100. Reprinted with permission. AP: Anteroposterior compression ratio.

canal diameter of < 13 mm is predictive of development of cervical spondylotic myelopathy (CSM)[18,40]. The shape and cross-sectional area of the cord are important independent predictors of the development of CSM and specifically, of neurologic deficit. A transverse area of cord < 60 mm$^2$ and a banana-shaped cord have both been correlated with the clinical presentation of myelopathy[41,42]. The anteroposterior compression ratio (AP) is calculated by dividing the anterior-posterior diameter of the cord by the transverse diameter of the cord (Figure 2)[43]. It has been suggested that an AP ratio < 40% is representative of substantial flattening of the cord and strongly predictive of significant neurologic dysfunction[43]. MRI has been reported as the most accurate method to quantify spinal canal diameter as it assesses both the bony and soft tissue components when estimating spinal canal diameter. This is particularly relevant in the context of CSM whereby central stenosis is often multifactorial to include both bony and soft tissue pathology, *e.g.*, osteophytic spurring, OPLL and posterior disc protrusion[44,45]. Spinal MRI for assessment of CSM should include imaging sets obtained in the axial and saggital planes using a combinations of T1-weighted, and T2-weighted sequences. Fast spin-echo MRI is the best modality to diagnose disc fragments and osteophytes[46].

### Dynamic mechanical factors

**Dynamic canal space narrowing:** It is intuitive that the extent of dynamic mechanical compression of the spinal cord could be significantly manipulated by movement of the cervical spine. During extremes of flexion and extension, a dynamic canal space of < 11 mm has been reported as a critical level for spinal functioning and is strongly predictive of cervical myelopathy[38,47]. Hyperextension of the neck causes canal narrowing by inducing buckling the ligamentum flavum and shingling the laminae. This "pincer effect" induces spinal cord compression between the inferior surface of one vertebra and the lamina or ligamentum flavum of the adjacent vertebra[23,47]. In flexion, the spinal cord lengthens and takes a more anterior position in the canal. If the cord impinges against a disc or vertebral body anteriorly, this will induce a higher intrinsic pressure, resulting in increased axial tension and

potential ischemic injury[19].

**Effects of stretch and shear:** Studies suggest that stretch-associated injury is a major contributor to myelopathy. This claim is supported by evidence in numerous experimental models to include neural injury, tethered cord syndrome, and diffuse axonal injury[11,48,49]. During flexion/extension movements of the spine, elongation of the canal results in longitudinal strain of the spinal cord[50]. This is consistent with Euler's Theorem, which implicates distraction of the complex portion of a structure that is placed in a flexion mode. The cord can stretch up to a quarter of its length which can correspond to an elongation of 17.6 mm measured at the level of the cervical spine during neck flexion. The increased stretch at this level results in a significant cord compression, translating to increased stress in the white matter and higher stress in gray matter[51]. This is further compounded by craniocervical flexion, which results in longitudinal transmission of stretching force and increases in intramedullary pressure in the lower cervical spine[48]. The rapid occurrence of low-grade mechanical stretching on neural tissues can exceed the biomenchanics of the tissue. This can lead disruption of the tissue and may result in transient or permanent neurological injury. Dynamic forces induced by flexion and extension of the spinal column contribute to axial cord strain with potentially detrimental stretch-induced axonal injury[11]. Cadaveric studies have demonstrated that ongoing longitudinal strain, even within physiological limits, will eventually surpass its material tolerance thereby permitting neurological injury[19]. In canine models, the elastic modulus of the spinal cord tissue decreases with increasing load. The canine spinal cord stripped of pia begins to rupture at a strain close to that developed in the cervicothoracic spinal cord of humans. Deleterious stresses to the spinal cord should be viewed in aggregate and in the context of movement. In the absence of repetitive movement, viscoelastic relaxation of the spinal cord mitigates stress resulting from deformation[52]. In a dynamic formulation, cord deformation form an osteophytic spur or calcified disc herniation adds abnormal plane loading and shear to strain resulting from

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┌─────────────────────────────────┐
│  Chronic spinal cord compression │
└─────────────────────────────────┘
                 │
                 ▼
┌─────────────────────────────────┐
│     Hypoxia/ischemia cascade     │
└─────────────────────────────────┘
         │               │
         ▼               ▼
┌──────────────────┐ ┌──────────────────┐
│ Neuronal/oligo-  │ │ Endothelial cell │
│ dendrocyte       │ │ dysfunction      │
│ apoptosis        │ │                  │
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│ Neuroinflammation│ │ BSCB disruption  │
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│ Functional neurological sequelae │
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**Figure 3  Cellular mechanisms involved in cervical myelopathy.** The hypoxia/ischemia cascade results from chronic progressive compression of the cervical spinal cord. This induces extracellular (TNF-$\alpha$) and intracellular pro-apoptotic pathways (p-53, JNK), induces neuroinflammation and ultimately, neuronal and oligodendrocyte destruction. Additionally, compromise of the BSCB results from ischemia-induced endothelial cell loss. Increased BSCB permeability alters the cellularity of the spinal cord microenvironment and is thought to further potentiate neuroinflammation. These mechanisms contribute to the upper limb dysfunction, spasticity, and gait disturbances observed in human cervical spondylotic myelopathy. BSCB: Blood-spinal cord barrier; TNF-$\alpha$: Tumor necrosis factor-$\alpha$.

flexion at the craniocervical and cervicothoracic junctions, significantly increasing overload stress.

The neurobiology of stretch-associated axonal and myelin injury is now understood as a result of work in primates and other animals and from studies of diffuse axonal injury in the human brain. Numerous studies have reproducibly demonstrated that axonal injury frequently occurs at sites of maximal mechanical stress and occurs as a series of recognized events: Myelin stretch injury, altered axolemmal permeability, calcium entry, cytoskeletal collapse, compaction of neurofilaments and microtubules, disruption of anterograde axonal transport, accumulation of organelles, axon retraction, bulb formation, and secondary axotomy[11,53,54].

There is clearly support for the contention that myelopathy increases in the presence of abnormal or excessive motion in the neck[55-57]. It should also be noted that stress is multifactorial and may include elements of gliosis within the spinal cord, acute and chronic changes in cord compliance, and altered gray and white viscoelastic relaxation gradients[51]. Understanding the deleterious effects of abnormal/excessive neck motion highlights the need to eliminate deformation of the spinal cord by minimizing abnormal mobility, and restoring normal sagittal spinal cord contour.

### Cellular mechanisms involved in cervical myelopathy

Sufficient pathophysiologic parallels between traumatic spinal cord injury (SCI) and cervical myelopathy have been identified to warrant a brief discussion of these novel mechanisms[58]. It has been proposed that primary mechanical injuries caused by static and dynamic forces to include compression, shear and distraction, induce a secondary injury at the molecular level. Several cellular mechanisms pertaining to these secondary injuries are discussed briefly to include ischemia, glutamatergic toxicity, neuroinflammation and apoptosis (Figure 3)[59-65].

**Ischemic injury:** Recent studies propose that progressive ischemia of the spinal cord may be central to the origin of cervical myelopathy[36]. The association of ischemia and myelopathy was first hypothesised from the observation of hyalinization and hypertrophy of the walls of the anterior spinal artery upon post-mortem histologic evaluation[66]. Later post-mortem studies of symptomatic cervical myelopathy patients demonstrated further abnormal histologic findings, to include spinal cord ischemic necrosis. Further evidence to implicate spinal cord ischemia in the pathogenesis of cervical myelopathy is that the lower cervical spinal cord, the commonest site of cervical myelopathy, also has the most vulnerable blood supply[67].

Ischemia of the cord secondary to compression was later proposed following experimental observations in canine models. These studies exposed the vulnerability of corticospinal tracts in ischemia conditions and the development of intramedullary cavitation secondary to peripial arterial plexus ischemia[66,68-70]. The use of balloon catheters to assess spinal compression in monkeys revealed hypoperfusion through the transverse arterioles originating from the anterior sulcal arteries and intramedullary branches in the central gray matter, upon anterior and posterior compression[71]. At the cellular level, there is evidence to suggest that oligodendroglia may be hypersensitive to ischemic injury and can apoptose after acute trauma[61,72,73]. These mechanisms may contribute to the demyelinating process observed in chronic cervical myelopathy[74]. The role of ischemic injury has also been proposed with evidence of edema and gliosis represented on MRI. T2-weighted MRI can detect high-intensity signal change, and demyelination and necrosis detected as a hypoenhancement on T1-weighted MRI[75]. MRI changes are an important prognostic indicator. The high intensity signal change seen on T2 images are associated with pathologies that are potentially reversible whereas the low intensity changes seen on T1 images are not. However, it has been proposed that all signal change on MRI is indicative of significant underlying pathology as it is representative of extensive neuronal hypoplasia, glial cell substitution of stroma, and white matter degradation[76].

**Disruption of the blood-spinal cord barrier:** It has been postulated that the ongoing cord compression observed in cervical myelopathy results in remodelling of the spinal cord[36]. This conformational change may result in endothelial cell loss and dysfunction of the local vasculature[7]. Damage to endothelial cells from resultant ischemia may also impact the integrity of the blood-spinal cord barrier (BSCB). BSCB dysfunction is accompanied by a disruption in vascular permeability leading to cord

edema. BSCB permeability in neurotrauma is viewed as a detrimental event to the central nervous system (CNS), secondary to entry of blood-borne inflammatory mediators and increased edema. However, CSM animal models examining mechanisms of BSCB dysfunction and neuroinflammatory responses, remain incomplete.

**Glutamatergic toxicity:** Glutamate is the major excitatory neurotransmitter in the human CNS. Excitotoxicity is the capacity of excitatory neurotransmitters to control apoptosis of neuronal and oligodendrocyte cells. In the clinical setting these cellular changes have been observed in multiple neuropathologies to include cerebrovascular accidents, traumatic spinal cord injuries and seizural activity[77]. Secondary excitotoxicity refers to neuronal and oligodendrocytic dysfunction mediated by glutamate. It is activated by fluctuations in neuronal metabolism, and has been associated with an array of chronic neurodegenerative diseases[77,78]. Researchers have proposed two structural properties specific to motor neurones that may increase their susceptibility to neurodegeneration. Calcium-mediated toxic events increase their susceptibility to neurodegeneration due to underexpression of both calcium binding proteins and GluR2 AMPA receptors. Based on these findings, future innovations may focus on targeting glutamate receptors in chronic neurodegenerative diseases.

**Apoptosis:** Apoptosis is defined as programmed cell death, recognised following ischemic and traumatic injury to the CNS[72,79-81]. Decreased perfusion and subsequent ischemia may be important pro-apoptotic events in cervical myelopathy, given the hypersensitivity of neurons and oligodendrocytes to ischaemic injury. In traumatic SCI, there is thought to be a cascade of degenerative changes at the lesion epicentre and demyelination of tracts distant to the injury[61]. The delayed degeneration of anterior horn cells in cervical myelopathy may reflect the effects of apoptosis. It is postulated that axonal degeneration is preceded by oligodendrocyte apoptosis in cervical myelopathy. This series of cellular events has been observed in histological analysis of *in vivo* models of spinal cord compression, whereby intact demyelinated axons have been observed in the presence of apoptotic oligodendrocytes[58,82]. This is of major therapeutic significance given recent evidence supporting the anti-apoptotic role of novel pharmocolgic inhibitors of the calcium-activated calpain, c-Jun N-terminal kinase and Fas-mediated apoptotic pathways[2,83,84]. Targeting these pro-apoptotic receptors in patients with cervical myelopathy may be employed as neuro-protective treatment strategies in attempt to to diminish the degree of neural degeneration.

**Neuroinflammation:** Although neuroinflammation is considered integral to wound healing in the setting of neurotrauma, recent evidence suggests that the inflammatory response is a key player in the pro-apoptotic pathway following this event[85]. It is becoming apparent that the innate and adaptive immune responses to acute spinal injuries and chronic cervical myelopathy are dissimilar. Unique to cervical spondylotic myelopathy is a slow inflammatory process, driven by chronic progressive inflammation[86]. Recruitment of neutrophils, monocytes/macrophages, and lymphocytes has been demonstrated in a human model of CSM[2]. However, the identity of the beneficial and detrimental inflammatory mediators in this process remains unknown. Future modulation of this inflammatory cascade has the potential to provide a basis for development of therapeutics for chronic spinal compression disorders.

**Effect of neuromodulators on CNS microvasculature:** Studies regarding the neurovascular complications of cocaine (benzoylmethylecgonine) report that its use exacerbates and accelerates the natural history of neurological pathology. Cocaine is a serotonin-norepinephrine-dopamine reuptake inhibitor that acts as a powerful CNS stimulant. The compound produces vasoconstriction of the CNS microvasculature, including the anterior spinal artery. Additionally, cocaine primes the responsiveness of platelets to arachidonic acid, leading to an increase in thromboxane release and platelet aggregation. Both these mechanisms independently contribute to infarction of the CNS microvasculature, including the spinal cord.

Recent studies have demonstrated a positive correlation between cocaine abuse and CNS infarction of the middle cerebral artery, vertebrobasilar artery territories, anterior spinal artery, and lateral medulla. However, there is a paucity of data relating to the chronic sequelae of cocaine use on the neurological microenvironment. Studies have demonstrated moderate and persistent alterations in cerebral and spinal blood flow and increased incidence of cerebral vasculitis among cocaine users. A recent study sought to establish the effect of chronic cocaine use on post-operative neurological recovery following surgical decompression of the cervical spine in a cohort of 95 patients diagnosed with symptomatic cervical spondylotic myelopathy[87]. The ability of the spinal cord to heal after surgical decompression is based on the intrinsic ability of the spinal cord to heal itself. Thus, the pre-operative health of the cord is paramount to post-operative improvement. This study revealed that chronic cocaine users had poorer post-operative neurological recovery than non-users and adds further credence to the negative impact of cocaine on spinal cord integrity. These data may influence pre-operative counseling and patient selection in attempt to optimize outcomes following surgical decompression for cervical myelopathy.

Smoking has been identified as a common risk factor for spinal degenerative diseases[88]. A recent *in vitro* study investigating the effects of nicotine exposure on nucleus pulposus proliferation and extracellular matrix synthesis demonstrated a significant anti-proliferative effect[89]. This suggests that nicotine may contribute to the pathogenesis of vertebral disc degeneration and the

development of cervical myelopathy however, further *in vivo* studies are required to elucidate its role at the molecular level.

### Genetic predisposition to cervical myelopathy

The role of heredity in the development of cervical myelopathy was first suggested by Bull *et al*[90] in 1969. Upon evaluation of several hundred cervical spine radiographs, the authors observed a higher prevalence of simultaneous CSM among twins. MRI of the spines of 172 monozygotic and 154 dizygotic twins revealed that heritability accounted for almost three-quarters of all findings, among a cohort of patients with severe CSM[91].

Flourishing interest in the role of heredity has been supported by evidence of families with a high incidence of CSM[92,93]. The authors' imply that these cases may represent extreme cases of genetic influence or it may depict the presence of a separate entity classified as "familial cervical spondylosis".

Previous studies have suggested a correlation between variants of collagen gene expression and degenerative intervertebral disc disorders[94,95]. Collagen IX, a structural component of nucleus pulposus and annulus fibrosis of the intervertebral disc, acts as a bridge between proteins in these tissues. Collagen IX is essential to the proper formation of the collagen II/IX/XI heteropolymer[96]. Recent studies have suggested that collagen tryptophan IX genetic material influence lumbar disc degeneration in patients with herniated nucleus pulposus. Interestingly, it has recently been demonstrated that patients with collagen IX polymorphisms who are smokers have a significantly higher predisposition to developing cervical myelopathy[88]. This landmark study suggests that smoking abstinence is important for reducing cervical myelopathy risk in patients with a genetic predisposition.

Despite the paucity of conclusive evidence to clearly delineate the aetiology of CSM, a multifactorial aetiology to include age-related degeneration, biomechanical factors and heredity is supported.

## FUTURE INNOVATIONS

Whilst acute SCI models have unveiled some cellular mechanisms involved in the pathobiology of CSM, this unique condition and its specific pathomechanisms are poorly defined. A limitation in advancing our knowledge of CSM has been a paucity of reproducible *in vivo* models to replicate CSM. Several models of acute cord compression have been developed, but relatively little work has focused on the development of an *in vivo* chronic, graded, cervical cord compression[64,92,97].

However, a recently developed animal model of CSM, employed a chronic compression device on the cervical spine of Sprague-Dawley rats, to achieve chronic and progressive cord compression[4]. This model has the potential to reproduce quantifiable neurobehavioral, neurophysiological, and neuropathological deficits akin to human CSM. Ultimately, this innovation may act as an important catalyst in the translation of targeted therapeutic strategies for cervical myelopathy[4].

Regarding technological advancements, innovations in neuro-imaging will continue to play a key role by facilitating timely diagnosis of soft tissue and osseous pathology in CSM, assist in optimal patient selection for surgical intervention and provide prognostic information in the post-operative period. In addition to advances in kinetic magnetic resonance imaging and DTI, metabolic neuroimaging, has been compared with functional assessments following clinical examination and findings on MRI, of patients selected for surgery for CSM. FDG-PET findings were highly sensitive displayed strong correlations with pre- and postoperative scores, and postoperative rehabilitation[98,99]. The major limitation of this technology is the poor resolution of PET scans. Despite this, future innovations in PET imaging may allow identification of early spinal cord damage and provide indications for surgical intervention.

## CONCLUSION

The cervical spine adapts to the challenges of gravity and the effects of mechanical loading through both structural and biochemical chnages. These adaptations may result in significant physical disability, and in turn stimulate altered biochemical pathways. The pathophysiology of CSM involves a combination of static and dynamic mechanical factors, which induce cellular changes to include neuroischemia, excitotoxicity, neuroinflammation and apoptotic events. There exists an urgent requirement for the development of novel neuro-protective treatment strategies to inhibit neural degeneration and optimize neurological recovery following surgical decompression for CSM.

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*MINIREVIEWS*

# Role of negative pressure wound therapy in total hip and knee arthroplasty

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## Abstract

Negative-pressure wound therapy (NPWT) has been a successful modality of wound management which is in widespread use in several surgical fields. The main mechanisms of action thought to play a role in enhancing wound healing and preventing surgical site infection are macrodeformation and microdeformation of the wound bed, fluid removal, and stabilization of the wound environment. Due to the devastating consequences of infection in the setting of joint arthroplasty, there has been some interest in the use of NPWT following total hip arthroplasty and total knee arthroplasty. However, there is still a scarcity of data reporting on the use of NPWT within this field and most studies are limited by small sample sizes, high variability of clinical settings and end-points. There is little evidence to support the use of NPWT as an adjunctive treatment for surgical wound drainage, and for this reason surgical intervention should not be delayed when indicated. The prophylactic use of NPWT after arthroplasty in patients that are at high risk for postoperative wound drainage appears to have the strongest clinical evidence. Several clinical trials

including single-use NPWT devices for this purpose are currently in progress and this may soon be incorporated in clinical guidelines as a mean to prevent periprosthetic joint infections.

**Core tip:** The application of negative pressure wound therapy (NPWT) in arthroplasty has generated much interest. Its proposed mechanisms of action include macrodeformation and microdeformation of the wound bed, fluid removal, and stabilization of the wound environment. There is little evidence to support the use of NPWT as an adjunctive treatment for surgical wound drainage. However, there appears to be strong clinical evidence for the prophylactic use of NPWT after arthroplasty in patients that are at high risk for postoperative wound drainage. Several clinical trials involving single-use NPWT devices for this purpose are currently in progress.

# INTRODUCTION

The use of negative pressure for wound healing, also referred to as vacuum-assisted closure, is a well-established practice that dates back to the 1940s[1-3]. While this technique was originally intended for flaps, skin grafts, and radical neck and groin dissection, its success has led to a rapid expansion of indications with over 700 articles describing its use[4]. Current evidence-based indications for the use of negative pressure on wound healing are broad and include chronic, acute, traumatic, subacute and dehisced wounds, partial-thickness burns, ulcers (such as diabetic or pressure), flaps and grafts[4-12].

The rationale behind the use of subatmospheric pressure for wound healing is based upon a wide array of mechanisms that ultimately result in wound contraction, mechanical stimulation of epithelial growth, and prevention of fluid collection, drainage and bacterial growth[13,14]. Despite the extensive literature, there is considerable controversy regarding its efficiency and applicability in certain clinical situations. A recent systematic review identified thirteen randomized clinical trials studying the use of negative pressure wound therapy (NPWT) and suggested that there is still little evidence to support its use in the treatment of acute and chronic wounds[15].

Periprosthetic joint infection (PJI) can be a devastating complication after joint arthroplasty. With the potential to not only treat but also prevent wound complications, there has been some interest surrounding the use of NPWT in the setting of joint arthroplasty. Patel *et al*[16] have shown that specific patient characteristics are associated with prolonged wound drainage following total hip arthroplasty (THA) and total knee arthroplasty (TKA). Prolonged wound drainage (*i.e.*, greater than five days postoperative) puts a patient at 12.7 times higher risk of PJI[17]. The ability to preoperatively determine candidates at higher risk for prolonged wound drainage (and hence, PJI) would enable the surgeon to consider NPWT in those arthroplasty patients who could benefit most from its use. To date, however, there are no specific guidelines, indications, or reviews on the use of NPWT after TKA and THA. This review will provide a brief introduction on the history and rationale of NPWT, its basic mechanisms of action, current evidence in the field of TKA and THA, contraindications, complications, risk factors and perspectives for future research in this area.

# BRIEF HISTORY AND RATIONALE OF NPWT

Despite meticulous hemostasis and tissue-handling techniques, any operation that requires separation of tissue planes and extensive tissue manipulation will cause some amount of fluid collection within the surgical wound. Fluid build-up can be detrimental to wound healing as it impedes normal blood flow and eventually leads to the formation of dense fibrous tissue. Prior to NPWT, common strategies to deal with this problem included devices such as Penrose drains and pressure dressings[18]. Numerous cases of debilitated patients with chronic, dehisced, and often infected wounds not amenable to closure led to the implementation of subatmospheric pressure systems[19]. This treatment modality provided not only complete coverage of the wound, but also constant interstitial fluid removal and mechanical stimulation of surrounding tissues. Sheppard[1] was the first to report the use of sealed drainage over surface wounds. However, the application of continuous subatmospheric pressure to this drainage system was first described by Raffl[2]. While initially intended for chronic, non-healing wounds on debilitated patients, the indications for NPWT expanded to include subacute and acute wounds[20-24].

There are five basic components to any NPWT system: The foam, tube, drapes, pump and canister. The foam is placed in direct contact with the wound and can be tailored to its specific geometry. Typically, the foam is made of polyurethane ether and is composed of highly interconnected cells of size ranging between 400-600 $\mu$m in diameter[25,26]. The so-called open-pore foam allows the pressure to be evenly distributed throughout

its entire surface. A non-collapsible tube is embedded in the foam and connected to a vacuum pump. The ideal pressure applied by the pump may vary according to the fragility of the surrounding tissues but optimal granulation tissue formation has been reported with a subatmospheric pressure of 125 mmHg[27]. Semioc-clusive adhesive drapes cover the surface of the wound containing the foam and these ensure an airtight seal. Finally, the proximal end of the tube leads to a canister that functions as a remote storage recipient for effluent fluid[19].

The first commercially available device that achieved widespread usage, in the early 1990s, was the Vacuum-assisted wound closure device and technology (V.A.C.) [Kinetic Concepts Inc. (KCI), San Antonio, TX]. Land-mark publications by Argenta *et al*[19] detailing the basic mechanism of NPWT and Morykwas *et al*[14] describing its clinical utility contributed to increasing acceptance of the NPWT. Since then, significant advances have been made to the device to improve safety and functionality. First, the incorporation of computerized alarm systems made it possible to detect inadequate seal, excessive fluid output and bleeding. Second, the development of compact, lightweight devices allowed patients to remain ambulatory throughout the duration of treatment[28]. Third, newer models also allow the instillation of fluids without loss of negative pressure in an attempt to continuously remove particulate and bacterial matter. These have been particularly useful in the setting of deep, contaminated wounds[29,30]. The recent use of silver coated foam is intended to provide a local antibacterial effect[31,32].

There is some concern regarding the overall quality and conflict of interest associated with the published studies supporting the use of NPWT in various clinical settings. One contributor to this problem is the hetero-geneity of published articles in terms of wound types, comparisons and outcome variables[15]. Conflict of inte-rest in NPWT-related research is also a matter of concern since the main research sponsors are the two leading device manufacturers. Despite the great commercial success, there is still a lack of data supporting the benefits of NPWT on wound closure[24].

## MECHANISMS OF ACTION

The application of NPWT on wound beds has direct and indirect effects on wound healing. There are four main direct mechanisms by which NPWT has been suggested to work: (1) macrodeformation; (2) microdeformation; (3) fluid removal; and (4) stabilization of the environ-ment[33]. Numerous indirect effects of NPWT on wound healing have also been proposed, including the modulation of inflammation[34], angiogenesis[35], granulation tissue formation[36,37], peripheral nerve response[38,39] and alteration in bioburden[7]. This section will focus mainly on the four direct mechanisms as they are more broadly studied and widely accepted.

The concept of macrodeformation involves the con-

traction of the foam once subatmospheric pressure is applied. Foam contraction exerts centripetal traction at the wound-foam interface resulting in approximation of the edges and decreased wound surface area[40]. The increased pressure applied to the tissue below the wound bed also contributes to the compression of capillaries, creating a localized decrease in perfusion. As a result, local upregulation of hypoxia-inducible growth factor production, including vascular endothelial growth factor, stimulates directionalized vessel sprouting towards the wound[33]. The end result of macrodeformation of the wound bed is thus a decrease in wound surface area and increased local vascularity.

When exposed to subatmospheric pressure, the porous surface of the foam induces microdeformations in the underlying tissue by creating an undulated surface of the wound bed[41]. Cell deformation leads to cytoskeletal stretch which in turn provides an independent stimulus for cell proliferation, migration, and differentiation[42]. As a consequence, the conformational changes induced in the surface of the wound by the porous surface of the foam ultimately result in increased epithelial cell proliferation as compared to normal occlusive dressings[40].

Fluid removal is an essential mechanism by which NPWT relieves the compressive effect of extracellular fluid on surrounding tissues[43] and clears the wound from toxins, exudates and bacteria[44]. Indirectly, this also reduces the amount of fluid that must be cleared by the lymphatics and induces a local increase in lymp-hatic density[45]. Less extracellular fluid build-up also translates into decreased capillary compression and increased tissue perfusion[43].

Finally, NPWT dressings have the ability to transform an open wound into a closed wound. The semiocc-lusive drapes covering the foam and surrounding skin maintain thermal stability, prevent evaporative water losses[46], stabilize osmotic and oncotic gradients at the wound surface[47] and reduce the risk of external contamination[48]. The semiocclusive aspect of the drapes also allows for limited permeability to vapor and other gases in order to maintain a moist wound environment[46].

## CURRENT EVIDENCE IN HIP AND KNEE ARTHROPLASTY

The use of NPWT was pioneered in plastic surgery and subsequently adopted by other surgical fields, including vascular, cardiothoracic and abdominal surgery. In orthopaedic surgery, there is limited literature on the use of NPWT. In a systematic review, Karlakki *et al*[49] identified 9 studies reporting the use of NPWT in orthopaedic surgery, five of which were Randomized controlled trials (RCTs). There is an even greater scarcity of published studies concerning the use of NPWT in adult reconstructive surgery. In this review, we identified eight studies reporting on the use of NPWT on either THA or TKA (Table 1).

**Table 1  Literature on negative pressure wound therapy on hip and knee arthroplasty**

| Ref. | Origin | Type | Indication | Device, negative pressure delivery | Length of use | Results | Conflict of Interest |
|---|---|---|---|---|---|---|---|
| Gomoll et al[50] | United States | Case series | Revision THA with history of multiple revisions (n = 1 NPWT), hemiarthroplasty with bladder/bowel incontinence (n = 1 NPWT) | V.A.C.®, 75 mmHg | 3 d (average) | Incisions noted to be well healed at a minimum of 3 mo | No |
| Kirr et al[51] | Germany | Case series | I and D for acute hip (n = 3 NPWT) and knee PJI (n = 2 NPWT) | V.A.C.® instill system with antibiotic instillation, amount of pressure delivery N/A | 15.2 d (average) | Eradication of infection in all cases, complete wound healing achieved in 10-14 d | N/A |
| Lehner et al[52] | Germany | Case series | I and D for acute hip (n = 2 NPWT), and knee PJI (n = 1 NPWT) | V.A.C.® instill system with antiseptic solution, 100-150 mmHg | 6 d (average) | Retention of all 3 implants with no further signs of infection at a minimum of 8 wk | N/A |
| Kelm et al[53] | Germany | Case series | I and D for acute hip PJI (n = 28 NPWT) | Periprosthetic placement of V.A.C.®, 200 mmHg for 72 h, and 150 mmHg | 6 d | Infection eradication in 26/28 cases with implant retention at a minimum of 12 mo | No |
| Howell et al[54] | United States | RCT | Primary TKA with increased risk for postoperative drainage (n = 24 NPWT vs n = 36) | V.A.C.®, 125 mmHg | 2 d | No difference in days to dry wound; 1 PJI in each group. Study stopped because of skin blisters in 63% of patients in NPWT arm | Yes |
| Pachowsky et al[55] | Germany | RCT | Primary THA for OA (n = 9 NPWT vs n = 10) | Prevena™, 125 mmHg | 5 d | Decreased seroma development in NPWT group (P = 0.021) | Yes |
| Hansen et al[56] | United States | Retrospective cohort | Persistent wound drainage at POD 3 to 4 after primary THA (n = 86 NPWT) and revision THA (n = 23 NPWT) | V.A.C.®, 125 mmHg | 24-48 h | 83 (76%) had no further surgery, 26 (24%) had further surgery. No NPWT-related complications | No |
| Pauser et al[57] | Germany | RCT | Hemiarthroplasty after femoral neck fracture (n = 11 NPWT vs n = 10) | Prevena™, 125 mmHg | 5 d | NPWT group had fewer dressing changes, less days of wound secretion, less wound care time, and less dressing material used (P < 0.05 for all) | Yes |

V.A.C.®: Vacuum assisted closure system (KCI, San Antonio, TX); Prevena™: Prevena incision management system (KCI, San Antonio, TX); THA: Total hip arthroplasty; TKA: Total knee arthroplasty; NPWT: Negative pressure wound therapy; PJI: Periprosthetic joint infection; I and D: Incision and debridement; RCT: Randomized controlled trial; OA: Osteoarthritis; N/A: Not available; KCI: Kinetic Concepts Inc.; POD: Post-operative day.

Gomoll et al[50] reported their experience in five cases in which NPWT was used as a postoperative dressing for primarily closed incisions. Indications included either a high likelihood of prolonged wound drainage or procedures performed in areas prone to postoperative swelling. This was among the first reported prophylactic use of NPWT in orthopaedic surgery. Of the five cases, two involved hip reconstruction. In one patient, NPWT was used because of large dead space and extensive soft tissue dissection. In the other case, NPWT was used because of the increased risk of contamination secondary to bladder and bowel incontinence. Treatment duration averaged three days and negative pressure was set at 75 mmHg. At three months post-operatively, the incisions were well-healed without complications. The remaining three cases involved the use of NPWT after open reduction and internal fixation for comminuted pilon, intertrochanteric, and subtrochanteric fractures which are not discussed here.

A series of papers originating from Germany reported on the therapeutic use of NPWT after acute PJI. Kirr et al[51] used a newer model of NPWT in which the direct instillation of fluids to the wound is made possible without the loss of negative pressure. The device was used on five cases after irrigation and débridement (I and D) for acute PJI in which the wound was left open. Fluids used for instillation included a local antibiotic (bacitracin with neomycin sulfate) and an antiseptic solution (polihexanide). Complete wound healing was achieved in all five patients after fourteen days of therapy. In a similar study, Lehner and Bernd[52] used the same device with instillation therapy to treat three cases of acute hip or knee PJI. Instillation was made with only a solution of polihexanide. At eight weeks, retention of all three implants was successfully achieved. Lastly, Kelm et al[53] reported on 28 cases of acute hip PJI managed with I and D followed by an internal use of NPWT. This was the first study to report the placement of the foam either periprosthetically or into the resection cavity with an outgoing transcutaneous tube. The wound was closed and inflammation parameters monitored. After a mean duration of nine days, surgical removal of the foam was performed. The foam was exchanged in cases in which there were macroscopic signs of persistence of the

infection. At a mean follow-up of 36 mo, eradication of infection was achieved in 26 out of the 28 cases. These three preliminary case series suggest a potential role for NPWT in the treatment of PJI, which requires further testing with large scale, controlled studies to support this practice.

Howell *et al*[54] conducted a RCT to establish the benefit of prophylactic NPWT after TKA in patients at high risk for prolonged wound drainage. High risk was defined as body mass index > 30 and the use of enoxaparin sodium for deep venous thrombosis prophylaxis. The trial was prematurely interrupted when a total of 60 knees were enrolled and a significant difference in blister formation was detected between the NPWT group and the control group. Among the 24 knees in the NPWT group, 15 (63%) developed linear blisters at the edges of the polyurethane ether foam, whereas only three out of 36 knees in the control group (12%) developed blisters. There was no difference in time to a dry wound or incidence of PJI between the two groups. In order to address the issue of blistering, a single fine-meshed, non-adherent film was recommended for use over unprotected skin in order to avoid direct contact with the foam[54]. This has already been incorporated in single-use, disposable devices such as Prevena™ (KCI, San Antonio, Texas) and PICO™ (Smith and Nephew, Hull, United Kingdom) and the blistering complication has not been reported in subsequent studies.

Another RCT evaluating the prophylactic use of NPWT for wound complications was conducted by Pachowsky *et al*[55]. Inclusion criteria included normal-risk THA for osteoarthritis, with nine patients receiving a single use NPWT device for five days and ten patients receiving a standard occlusive dressing. The novelty of this study was its primary end-point: The development of post-operative seromas as detected through ultrasound measurements. On post-operative day ten, a seroma was present in 44% of patients in the NPWT group as compared to 90% in the control group, with a significantly decreased seroma volume in the NPWT group (1.97 mL *vs* 5.08 mL, $P = 0.021$). Although reduction of postoperative seromas may potentially lead to increased blood flow and better apposition of the wound edges, there are no data to suggest that this is specifically linked to decreased rates of PJI and to justify the use of NPWT in normal-risk patients.

Hansen *et al*[56] investigated the therapeutic use of NPWT for persistent incisional drainage after primary and revision THA. Indication for NPWT was persistent wound drainage at postoperative days 3 to 4. Interestingly, 83 patients (76%) had complete resolution of wound drainage without further surgical intervention. Of the 26 patients who required further intervention despite NPWT, 23 (88%) had complete resolution of drainage after a single I and D. This study was the first in the field of reconstructive surgery to attempt NPWT first instead of I and D. Furthermore, it was reported that failed therapy with NPWT did not compromise the results of a

subsequent I and D. Even though this was a retrospective study, it provided important data as to the value of NPWT as primary therapy for early wound drainage.

Lastly, Pauser *et al*[57] conducted a RCT studying the prophylactic use of NPWT after hemiarthroplasties for femoral neck fractures. Eleven patients were randomized to the NPWT group and ten patients to a control group (occlusive dressing). The end-points chosen for analysis were the number of dressing changes ($P < 0.0001$), days of wound secretion ($P = 0.0005$) and wound care time ($P < 0.0001$). Statistical significance was achieved in all three end-points favoring the NPWT group. Furthermore, there was a decreased incidence of seromas in the NPWT group (36% *vs* 80%). Despite the limited sample size, this study attempted to show not only the main benefits of NPWT in terms of wound healing, but also secondary gains such as less time spent by health care professionals and less consumption of wound care resources.

Overall, there is a clear lack of high-ranking scientific evidence in the field of adult reconstructive surgery concerning the use of NPWT. Studies are limited by a high variability of clinical settings and small sample sizes. The prophylactic use of NPWT after arthroplasty in high risk candidates seems to have the strongest clinical evidence[54,56,58]. The use of NPWT as an adjunctive therapy for acute PJI after I and D is only supported by small case series[51-53]. Finally, the use of NPWT as the main therapy for postoperative wound drainage is supported by a single retrospective study[56].

## CONTRA-INDICATIONS, COMPLICATIONS AND RISK FACTORS

According to the Food and Drug Administration (FDA), due to the lack of appropriate studies, NPWT should be contraindicated in the following scenarios: (1) necrotic tissue or eschar present; (2) untreated osteomyelitis; (3) unexplored fistulas; (4) malignancy in the wound; and (5) exposed vasculature, nerves, anastomotic sites or organs[58]. These guidelines were based on two major concerns: (1) the inability of NPWT to replace surgical treatment when this is formally indicated; and (2) the mechanical strain that sub-atmospheric pressure can place upon fragile tissues.

Despite the rapid expansion in the use of NPWT across various clinical settings, the reported complication rates are surprisingly low. The most worrisome and potentially lethal complication has been exsanguination. Four fatal exsanguinations have been reported with use of NPWT and these occurred when the tube was attached to wall suction[59]. This practice is now strongly condemned and the use of safety alarms for excessive fluid drainage has been incorporated to NPWT devices. Safety alarms are also designed to detect air leaks, as this has been shown to increase wound size due to skin dehydration[27]. Fatal toxic shock syndrome has been reported in two cases, both of which had a purportedly blockage in the drainage system[60]. Retention

of foam within the wound, particularly when multiple, small fragments of foam were used, is also a known complication[33]. Lastly, blistering has been a minor complication in most studies within orthopaedic surgery, except for one[54]. This problem has largely been resolved with the addition of a protective adhesive layer between the foam and skin.

Patient-related risk factors that demand special attention when considering NPWT are: (1) high risk of bleeding and hemorrhage; (2) use of anticoagulants or platelet aggregation inhibitors; (3) patients with friable or infected blood vessels, vascular anastomosis, infected wounds, osteomyelitis, exposed organs, vessels, nerves, tendons, and ligaments, sharp edges in the wound, spinal cord injury, enteric fistulas; (4) patients requiring magnetic resonance imaging, hyperbaric chamber, defibrillation; (5) patient size and weight (increased dead space); (6) proximity to vagus nerve (with risk of bradycardia); (7) circumferential dressing application; and (8) mode of therapy (intermittent *vs* continuous negative pressure)[58]. The FDA report also stresses that the vast majority of adverse events and deaths related to NPWT has occurred either at home or in a long-term care facility. Nevertheless, despite the contraindications and risk factors, there are successful reports of NPWT in the settings of sternum osteomyelitis[61] and exposed organs[62].

## AUTHOR RECOMMENDATIONS

At our institution, NPWT is applied for the aforementioned indications in total hip and knee arthroplasty. The quantum of negative pressure applied is typically either greater than 75 mmHg (for wound depth extending beneath fascia) or less than 75 mmHg (above fascia). Variations in pressure magnitudes for certain populations (such as pediatric and geriatric patients) are made in accordance with manufacturer guidelines and clinician judgment. Placement of NPWT must be done only after ensuring that the surrounding skin is dry enough for the adhesive material to provide an effective seal. Incisional NPWT is typically discontinued 3-5 d after surgery when there is no longer any drainage from the wound. However, NPWT dressings for deep, open wounds are changed every few days until satisfactory healing is eventually achieved. If drainage persists or is excessive in quantity, further surgical management may be necessary. In order to avoid skin maceration, the authors recommend placing the foam directly on the open wound and using protective material, such as a hydrocolloid dressing, for the surrounding skin.

## PERSPECTIVES

There are currently over 60 clinical trials registered online at www.Clinicaltrials.gov, mostly concerning the prophylactic use of NPWT over high-risk closed incisions. In the adult reconstructive field, there are seven clinical trials on NPWT, all of which are evaluating its efficacy in preventing wound complications and infections. Despite the substantial lack of evidence, the prophylactic use of single-use devices such as Prevena™ (KCI, San Antonio, Texas) and PICO™ (Smith and Nephew, Hull, United Kingdom) in patients at increased risk for postoperative drainage seems to be gaining acceptance and may potentially be incorporated in clinical guidelines for PJI prevention in the near future.

The therapeutic use of NPWT for prolonged wound drainage in an attempt to avoid the need for an I and D is still unsupported. Furthermore, Jaberi *et al*[63] showed that delaying surgical intervention after the onset of drainage predicts a higher failure rate once an I and D is undertaken. The role of NPWT in the management of prolonged wound drainage or acute PJI is still controversial and should not be a reason to delay surgical intervention.

## CONCLUSION

The efficacy of NPWT in wound healing and its secondary benefits in terms of improving cost-effectiveness and comfort for both patient and caregiver is irrefutable. The fast expansion of indications and wide range of clinical scenarios in which it has been adopted has precluded standardization of protocols and large scale studies. For this reason, the use of NPWT still relies heavily on empirical data. Within hip and knee reconstructive surgery, the most commonly accepted use of NPWT is for the prophylaxis of wound complications in high-risk closed surgical wounds. There is a dire need for unconflicted, standardized and larger volume studies to validate this practice and to establish the role that NPWT may have in the treatment of prolonged wound drainage and acute PJI.

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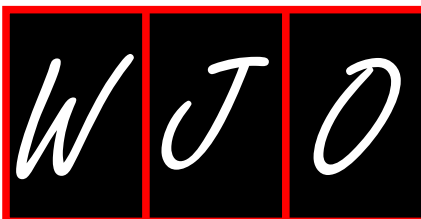
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*MINIREVIEWS*

# Direct anterior total hip arthroplasty: Literature review of variations in surgical technique

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## Abstract

The direct anterior approach to the hip has been suggested to have several advantages compared to previously popular approaches through its use of an intra-muscular and intra-nervous interval between the tensor fasciae latae and sartorius muscles. Recent increased interest in tissue-sparing and minimally-invasive arthroplasty has given rise to a sharp increase in the utilization of direct anterior total hip arthroplasty. A number of variations of the procedure have been described and several authors have published their experiences and feedback to successfully accomplishing this procedure. Additionally, improved understanding of relevant soft tissue constraints and anatomic variants has provided improved margin of safety for patients. The procedure may be performed using specially-designed instruments and a fracture table, however many authors have also described equally efficacious performance using a regular table and standard arthroplasty tools. The capacity to utilize fluoroscopy intra-operatively for component positioning is a valuable asset to the approach and can be of particular benefit for surgeons gaining familiarity. Proper management of patient and limb positioning are vital to reducing risk of intra-operative complications. An understanding of its limitations and challenges are also critical to safe employment. This review summarizes the key features of the direct anterior approach for total hip arthroplasty as an aid to improving the understanding of this important and effective method for modern hip replacement surgeons.

Key words: Anterior hip arthroplasty; Anterior supine intramuscular approach; Total hip arthroplasty; Direct anterior approach

Core tip: This review captures the most important concepts of direct anterior total hip arthroplasty as described by numerous surgeons' experiences with the procedure. It compares variations in surgical exposure and arthroplasty techniques, while identifying key elements of the anterior hip anatomy for performance of safe and efficient surgery. The review divides anterior hip arthroplasty into six distinct elements, citing the

most relevant pearls and pitfalls of previous publications and the most relied upon surgical methods. This concise summary can be beneficial to any level of surgeon desiring to enhance their understanding of direct anterior total hip arthroplasty.

## INTRODUCTION

The direct anterior approach (DAA) to the hip was first described by Carl Hueter in 1881. However Marius Smith-Petersen is frequently credited with developing this technique due to his prolific use of the technique throughout his career, after initially publishing his description in 1917. Modern day literature frequently refers to this method interchangeably as both the Hueter and Smith-Petersen approach when identifying the anterior based incision that utilizes the interval to the hip joint through the tensor fasciae latae (TFL) and the sartorius muscles[1]. Light and Keggi[2] published their extensive experience using this approach for hip arthroplasty in 1980, and the Judets described the procedure with use of a fracture table in 1985[1,2]. The modern day desire to perform hip reconstruction through less invasive and tissue sparing methods was the key driver in the newfound interest of the anterior approach. This has led to a surge in its proliferation over the past 15 years[3]. During this time, numerous authors have described variations of the technique and key concepts to safe and successful performance of hip arthroplasty. Although many consider the DAA appropriate exclusively for primary joint replacement, several authors have noted routine use of this technique for complex revision surgery and bipolar hemiarthroplasty for hip fractures[4-6]. This review seeks to summarize the published literature on the direct anterior total hip arthroplasty procedure with a focus on comparative key pearls and pitfalls.

## INDICATIONS AND CHALLENGES

Several authors have recommended using the DAA in patients of nearly all body habitus and hip conditions[4,7]. The ideal patient has been described as a flexible, non-muscular patient with valgus femoral neck and good femoral offset. It is reasonable to initially develop skills to perform the approach in slender patients with a body mass index of less than thirty[8]. As achievement of appropriate exposure is gained with experience using the technique, it has also been suggested that lack of appropriate instrumentation designed for anterior supine intramuscular approach is a contraindication[8]. Some anatomic features of the native hip and pelvis

are recognized to make the DAA more difficult. A wide or horizontal iliac wing limits access to the femoral canal for broaching and femoral component placement. Acetabular protrusio brings the femoral canal closer to pelvis and obstructs access to femur. A neck shaft angle with decreased offset positions the femoral canal deeper in the thigh, and anatomy associated with obese muscular males limits the space available to place components[9]. One disadvantage of the anterior approach is diminished access to the posterior column. If the patient has a deficient posterior acetabular wall from previous hardware or trauma, or if posterior acetabular augmentation is contemplated, the anterior exposure may be unsuitable[10].

## SURGICAL TECHNIQUE

### Patient positioning

The vast majority of authors describing the DAA position the patient supine on a fracture or regular table. Michel *et al*[11] also proposed performing anterior total hip arthroplasty (THA) using lateral decubitus positioning. When using a regular table, the patient is positioned with the pelvis located over the table break, which can be angled to allow hyperextension at the hip joint (Figure 1A). A bump may be used placed under the sacrum, centered at the anterior superior iliac spine (ASIS) to further elevate the pelvis[3]. Kennon *et al*[12] recommends orienting the table at right angles to the walls for accurate referencing and anatomic orientation. The contralateral leg is frequently draped into the field, and an arm board may be placed alongside to allow for abduction during femoral exposure[8]. Obese patients should have the pannus retracted with adhesive tape to avoid interference with exposure[7]. With use a fracture table (Figure 1B), the peroneal post should be well-padded to avoid peroneal nerve neuropraxia[13].

### Surgical approach

The descriptions for skin incision vary by surgeon, however most authors rely on the ASIS and greater trochanter as anatomic landmarks for reference (Figure 2). An oblique incision is made originating 2-4 cm distal and lateral to the ASIS to a point a few finger breaths anterior to the greater trochanter[4,7,8,11,14,15]. A cadaveric anatomy study showed that the zone immediately distal to the intertrochanteric line formed an anatomic barrier to protect neurovascular structures. Incision extension distal to this point risks damage to branches of the lateral femoral circumflex artery (LFCA) supplying the proximal quadriceps muscles and femoral nerve divisions to the vastus intermedius and lateralis[16]. The incision is generally oriented in line with the TFL, which can also be delineated by a line from the ASIS to the patella or fibular head, or in line with the femoral neck[17]. Fluoroscopy may be used to assist in identifying the femoral neck and midpoint for the incision[18].

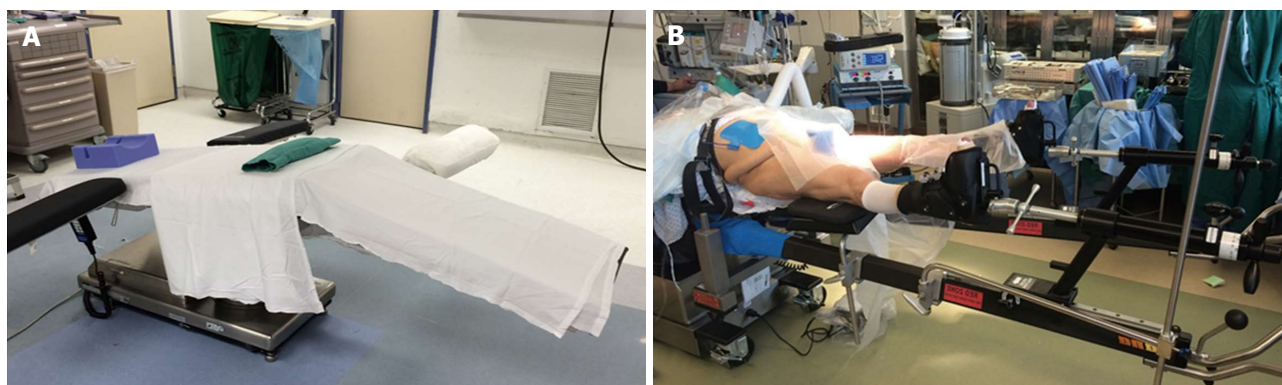A well-recognized complication of this approach is the proximity of the incision to the lateral femoral

**Figure 1 Patient positioning.** A: Use of a regular table with bump under the sacrum and ability to lower the distal end of the bed down to afford better femoral exposure. An extra arm board can be placed on the contralateral distal end of the bed to support the contralateral leg while accessing the operative femoral canal; B: Patient positioning on a fracture-type table (Hana table, Mizuho Orthopedics Systems, Inc.).

cutaneous nerve (LFCN). Though it is commonly believed that remaining lateral to the TFL/sartorius interval reduces risk for injury to the nerve, a cadaveric study of LFCN arborization showed the gluteal branch crossed the anterior margin of the TFL at 44 mm from the ASIS; the femoral branch also crossed this margin in half of specimens, at an average of 46 mm distal to the ASIS[19,20]. A 10% variation in branches was found in a study of 60 cadavers[21]. De Geest *et al*[22] found a decreased incidence of LFCN injury by further lateralizing their incision. Blunt dissection through the subcutaneous fat is recommended to further minimize risk of nerve injury[7]. Damage to the medial subcutaneous fat pad should be avoided to prevent injury to the trunk of the LFCN, which can result in meralgia paresthetica[8].

The interval between the TFL and sartorius is entered by incising the fascia over the medial TFL muscle belly, retaining an adequate sleeve of tissue for closure and offering protection to the LFCN[4,15,23]. Care should be taken to ensure the appropriate interval, as dissection through the lateral TFL and not in the intramuscular portal may result in damage to the motor branch of the superior gluteal nerve[7]. If the exposure is too posterior, blood vessels will be seen entering the fascia and the fascia becomes denser as it overlies the gluteus medius, which should prompt recognition of the improper interval[15]. Conversely, if the plane is developed too medially, dissection in to the femoral triangle will occur, risking injury to the femoral neurovascular bundle[19]. Blunt dissection separates the TFL muscle belly from the fascia and facilitates entry into the interval for proper exposure of the hip capsule.

### Hip exposure

A sharp retractor may be placed around the greater trochanter and the rectus femoris can be retracted medially with a rake or Hibbs retractor[8]. The ascending branches of the LFCA usually lie in the distal portion of the approach, though can be somewhat variable in location and extent; these vessels should now be visualized and ligated with electrocautery or hand suture tie. Some surgeons have employed bipolar sealing technology

for the purposes of vessel coagulation and hemostasis throughout the procedure. The instrumentation is replaced with a curved retractor over the superior capsule to retract the TFL superiorly. A second cobra or Hohmann can be placed in a "soft spot" proximal to the vastus lateralis, on the medial of the neck to retract the rectus femoris and sartorius medially. Overzealous retraction should be avoided to minimize damage to the TFL and rectus, as well as to avoid neurovascular traction.

Specialized retractors with extra-depth blades and curved sides (Figure 3) are also available for minimally invasive surgery to facilitate gentle soft tissue handling[19]. In muscular patients, the rectus femoris and TFL insertions near the ASIS may be elevated to facilitate exposure[7]. A capsulotomy or capsulectomy of the anterior capsule have both been described. Some authors advocate removal of the capsule to facilitate exposure. However, retaining a medial portion may provide a sleeve of tissue between the iliopsoas tendon and acetabular rim to reduce irritation[13,24,25]. Kennon *et al*[12] advised removal of a thick, contracted anterior capsule to prevent impingement possibly contributing to posterior dislocation. Positioning the operative leg in a figure-four position on a regular table can assist in release of the anterolateral and inferior capsule in the calcar region[15,23]. Release of the superior capsule has been shown in a cadaveric study to be the most crucial in allowing elevation of the femur, which was not increased with release of the posterior capsule[26]. Following capsular release, attention is then turned to the femoral neck and head.

The visible labrum and anterior osteophytes may be excised to assist in removal of the femoral head, but this is often not necessary. Medial and lateral retractors are repositioned within the capsule around the femoral neck. The head may be removed by performing a femoral neck cut and placing a corkscrew in the head, or by first excising a "napkin ring" section of the neck. Placement of a hip skid along the anterior acetabulum with a slightly distracted femoral head, *via* traction to the limb or instrumentation in the head, can facilitate head removal and transection of the ligamentum teres[13,27]. Gentle
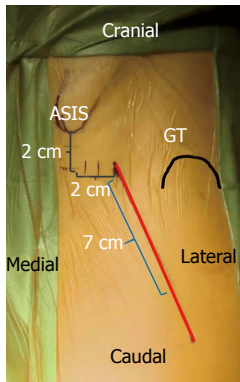
**Figure 2 Surface anatomy for the direct anterior approach.** A 6-8 cm oblique incision is typically used by the authors. This incision may be extended proximally and distally as needed along the Smith-Petersen interval for adequate femoral and acetabular exposure.



**Figure 3 Selected retractors used for direct anterior approach.** From left to right, hip skid for ceramic head reduction, greater trochanteric retractor/elevator, femoral elevator for medial/calcar exposure (front and side views), medial acetabular wall retractor, posterior acetabular wall retractor, and tensor fascia lata retractor.

external rotation can help with dislocation. If not already done so, the femoral neck cut based on pre-operative planning is performed, with completion of the cut near the greater trochanter finished using an osteotome to reduce the risk of fracture by an oscillating saw.

*Acetabulum*
A sharp retractor may be inserted at the ventral acetabular rim, keeping the retractor immediately adjacent to the bone to avoid femoral nerve compression[19]. Some surgeons use a light-mounted retractor in this position to improve visualization. Additional retractors are frequently placed at the posterior acetabulum and at the level of the transverse acetabular ligament. Remaining labrum and obstructing osteophytes are removed from the acetabulum, and reaming is commenced. Most surgeons performing this approach advocate use of specialized offset instruments. However, a straight reamer can be used with adequate exposure and careful attention to avoid leverage of the anterior acetabulum to prevent eccentric anterior reaming. Post *et al*[3] also recommend positioning the reamer head first and then attaching the handle for challenging access. If performing the procedure in the lateral decubitus position, visualization of the anterior acetabulum is difficult and can complicate reaming and cup placement.

Assessing the native pelvis to assist proper cup positioning may be accomplished by palpating the anterior superior iliac spines[19]. Although there is a tendency towards over-anteverting the cup with this approach by holding the cup positioner too vertical, an advantage of the supine DAA is the ability to utilize fluoroscopy intra-operatively[19]. Many surgeons recommend reaming and cup placement using image guidance, particularly in initial adoption of the technique. A press-fit acetabular cup may be inserted with a target abduction angle of 35-45 degrees and anteversion of 10-20 degrees.

Of note, a study of fluoroscopy-guided anterior hip arthroplasty found an early higher rate of dislocation using a goal of 10-30 degrees of anteversion, which was improved by adjusting the target angle to 5-25

degrees[28]. Computer aided navigation has also been described to improve accuracy of cup placement. A study of computed tomography-based hip navigation comparing mini-anterior and mini-posterior found an accuracy of 2.0 degrees for abduction and 2.7 degrees for anteversion of cup placement with the anterior approach. Surface registration took one minute longer in the anterior approach but operative time was not significantly different[29]. A review of 300 DAA hips, half performed with computer aided navigation, showed decreased operative times with navigation and greater accuracy of abduction angles[30]. Following placement of the cup, an acetabular liner is inserted and acetabular retractors are removed.

*Femur*
The femur may be exposed on a fracture table by dropping the limb spar to the floor, with all traction removed, by a non-scrubbed assistant, along with external rotation and adduction of the limb. Adequate soft tissue capsular releases about the proximal femur should be performed prior to this maneuver. Matta *et al*[27] utilized a scrubbed assistant to provide additional external rotation force at the femoral condyles using to reduce the stresses generated by the traction boots across the ankle, which can subject patients to iatrogenic ankle fracture. A retractor should be located at the calcar region, and a second retractor at the lateral greater trochanter during this maneuver. Release of the posterosuperior capsule will aid in clearance of the greater trochanter from behind the acetabular rim[8].

Using a regular table, this exposure is performed by dropping the distal end of the table and by placing the bed in a Trendelenburg position, forming an inverted V-shape of the body for hyperextension at the hip. Trendelenburg positioning may alternatively be established at the onset of the case; however, patients undergoing general anesthesia may be at higher risk for gastrointestinal reflux[13]. The contralateral leg may be placed on a mounted arm board or padded Mayo stand

to allow for adduction of the operative limb under the contralateral leg in a figure-of-four position on a regular table[24]. For lateral decubitus positioning, the operative limb is abducted, hyperextended, externally rotated and flexed at the knee, with the foot positioned into a sterile bag posterior to the patient.

Elevation of the femur may be accomplished using a hydraulic lift hook or manual placement of a hook just distal to the vastus ridge around the posterior femur. Tension on the femur can be appreciated through tactile and visual feedback of the retractor behind the greater trochanter. In cases where the femur is unable to be appreciably exposed using these maneuvers, Moskal *et al*[9] described sequential releases of soft tissue along the medial greater trochanter and femoral neck under tension, progressing through the release of hip capsule, piriformis, gemelli, and obturator internus. Posterior circumflex vessels should be identified and cauterized with these releases[12]. The obturator externus provides hip stability through the most medial pull of the femur to the pelvis and should not be released unless necessary[9]. Adequate entry to the femur should be verified with removal of interfering bone or tissue by rongeur or box cutter to prevent varus stem positioning[13]. A high-speed burr may also be used as necessary.

Offset hand instruments may be preferred for broaching and stem placement. An alternative method to providing access utilizes a separate stab wound proximally in line with the femoral canal. This technique may be useful in large or muscular patients and for revision arthroplasty, and can prevent the need for extensive posterior releases[12]. As femoral perforations are a known early complication of this approach, Post *et al*[3] recommended identifying the trajectory of the canal through use of a guide wire on a T-handle. The femur is then broached and the trial component inserted. Reduction of the hip is performed by reversal of the steps utilized in exposing the femur. Fluoroscopy can be used to assess the adequacy of components, and a stability assessment is performed, emphasizing careful attention to extremes of external rotation and extension. Leg lengths may be compared directly on a regular table or by utilizing radiographic comparison to the contralateral limb with a fracture table[13-15]. Final components are then placed in a similar fashion, and a final stability examination is performed.

### Closure
The wound is thoroughly irrigated, and closure is performed according to surgeon preference. If capsulotomy was made, the flaps may be approximated. Hematoma prevention requires adequate hemostasis, as there is higher predilection for hematoma formation with less inherent gravity pressure over an anterior wound compared to other approaches. Furthermore, the risk for hematoma to track deeply exists as the only layer routinely closed is the tensor sheath[31]. Suturing of the tensor fascia latae should be performed with care to avoid damage to the LFCN medially. The subcutaneous

tissue and skin are closed in a standard fashion. Many surgeons choose to leave a drain in place beneath the fascial layer; however, a study of 120 patients comparing drain utilization found that patients without drains had an earlier dry surgical site and were discharged from the hospital on average one day earlier. There was a non-significant trend toward high pain scores on post-operative day one, with increased thigh swelling on post-operative day two. There were no difference in transfusion requirements between the groups[32].

## CONCLUSION
All approaches to the hip have been shown to be safe and effectives with proper training and meticulous technique. The DAA to the hip has gained significant popularity recently, and can be a valuable tool for hip replacement in most patients. This review examines the published variations of direct anterior THA, showing that several facets of the procedure can be tailored to a given surgeon's preference or the particular needs of the patient anatomy. Familiarity with the surgical anatomy and understanding the limitations of the anterior hip approach are key to successful execution. The growing desire for less invasive arthroplasty with improvement in functional results makes this approach an attractive choice for surgeons.

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*MINIREVIEWS*

# Total elbow arthroplasty is moving forward: Review on past, present and future

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## Abstract

The elbow joint is a complex joint, which, when impaired in function, leads to severe disability. In some cases however, an arthroplasty might be an appropriate treatment. In the past four decades, large steps have been taken to optimize this treatment in order to achieve better post-operative outcomes. To understand these progresses and to discover aspects for upcoming improvements, we present a review on the past developments, the present state of affairs and future developments which may improve patient care further.

Key words: Total elbow arthroplasty; History; Future; Improvements; Review

**Core tip:** Total elbow arthroplasty (TEA) is a relatively uncommon surgical procedure, performed in selected cases of incapacitating elbow diseases. In the past four decades, TEA has evolved from an experimental procedure to a reliable option, which is still more frequently performed. We believe it is necessary to understand the history of the development of TEA in order to accomplish further improvements. In this review we focus on the evolution of the elbow arthroplasty, from a historic overview, up to the present and address issues that could improve the clinical outcome in today's practice.

## INTRODUCTION

From an anthropologic point of view, the upper extremity in humans has evolved into an instrument capable of achieving a large range of motion in order to perform highly complex tasks. This "open kinetic chain"

demands different anatomic structures in comparison to the "closed kinetic chain" of the lower extremity.

Consequently, pathological conditions differ between the two extremities. Pathology of the lower extremity generally results in reduced mobility of the patient. In the upper extremity however, pathologies here restrict the patient from performing simple activities in daily life. In this situation, the problem cannot be managed by the help of external aids (*e.g.*, wheelchairs or crutches)[1].

The elbow is a complex joint, consisting of three independent joints which cooperate together to move in multiple axes while maintaining a high level of stability[2]. The humero-ulnar joint permits a flexion/extension motion and is additionally stabilized by the olecranon and coronoid process in extreme flexion and extension. The combination of the proximal and distal radio-ulnar joint allows a pronation/supination movement, which is restricted by ligaments to a certain degree. The flexion of the elbow is important in allowing the hand to reach above and at the level of the head in order to achieve simple, yet important day-to-day activities, such as eating and the washing of hair and face. The combination of these movements, as well as shoulder rotations, allows versatile positioning of the hand in space and is a prerequisite for the fulfillment of complex tasks.

A decreased range of motion in the elbow joint can be directly due to pathology, *i.e.,* primary osteoarthritis, or trauma. Pain, usually secondary to pathology such as rheumatoid arthritis, is another factor that may restrict elbow function as well. A total elbow arthroplasty (TEA) can improve the range of motion and can also relieve pain in selected cases. Therefore, TEA can considerably improve function of the upper limb and increase the quality of life.

Though the use of TEA has almost doubled between 1998 and 2011 in the United States, it is still a relatively uncommon orthopedic procedure. It is performed more often in women than in men[3] and is also used in relatively young patients[4,5]. The number of of TEA performed annually is 1.4 in 100000 of the population, considerably less than the 70 to 99 in 100000 of the population for total hip replacement[4,6].

The expanding practice of TEA leads to a new field in orthopedic surgery. We believe it is necessary to understand the history of the development of TEA in order to accomplish further improvements. In this review we will focus on the evolution of the elbow arthroplasty, from a historic overview to the present and address issues that could improve the clinical outcome in today's practice.

## THE PAST

The first salvage surgery by excising infected humeral and ulnar bone was performed by Ambroise Pare in the sixteenth century to prevent amputation due to an infected elbow joint[7]. In the nineteenth century, as more advanced surgical and post-operative care could be provided, creating a pseudoarthrosis by resecting the distal humerus became an option for incapacitating elbow disease. Following the developments in hip surgery, instead of resecting the joint, the idea of replacing the diseased elbow joint became a concept. It resulted in two streams; the anatomical arthroplasty, aimed to recreate native anatomical structures, and the functional arthroplasty, which covers the functionality of the elbow joint but does not resemble normal anatomical structures.

In 1925, the first attempt to replace an elbow joint by prosthetic materials was documented, when Robineau inserted an anatomically correct elbow prosthesis, consisting of metal and vulcanized rubber. In 1941, Boerema used a hinged non-anatomical prosthesis completely made of metal[7].

In 1952, Venable[8] published a case-report of a custom-made anatomical prosthesis after a comminutive fracture of the distal humerus which was not amendable for proper osteosynthesis. A short-term follow-up of 15 mo was reported with a good outcome[8].

The promising results of experimental elbow surgery led to a rush on patents for elbow arthroplasties by several inventive doctors. In 1954, a functional prosthetic elbow joint was patented by Prevo[9], but did not reach a widespread use due to frequent loosening. In 1972, Dee[10] reported his treatment of 12 patients using a functionally designed TEA. This publication initiated an increase in various TEA models in the 1970's, ranging from stemmed devices to anatomy-resembling resurfacing models[9,11-17]. However, overall post-operative complication rates including; loosening, deep infection, and ulnar nerve neuropathy were high; ranging up to 57%[18].

It has been a challenge to design a TEA, which copies the native function and stability of all three articulations in the elbow joint. A drawback of anatomical arthroplasties was the lack of intrinsic stability. The anatomical, unlinked resemblance requires the integrity of ligaments and muscles. However, these structures often become insufficient in long-standing disease such as rheumatoid arthritis. Therefore, the unlinked anatomical design has lead to a high dislocation rate[19,20].

During flexion and extension of the elbow, some degrees of valgus and varus laxity is normal[21]. However, the linked "first generation" TEA's did not offer this laxity, which resulted in frequent loosening due to stress at the implant-bone transition[18]. This problem was overcome by the "second generation" TEA, introducing sloppy hinges, which allow some varus-valgus laxity due to their semi-constrained design.

Fixation of the prosthesis proved to be challenging too, resulting in the application of a wide range of methods: Prevo[9] designed screw-threaded stems, Stevens a slide-on self-locking resurfacing arthroplasty, Schlein[11], Pritchard *et al*[13] and Dee[10] used smooth cemented stems, Roper *et al*[14] used a cemented humeral component and Amis and Miller[16] used screw fixation for the ulnar component[9,11,13-17]. Harmon[12] used two rings as a radiocapitellar joint. These models are

**Figure 1 Historical, distinctive types of total elbow arthroplasty.** A: Prevo[9] (1954); B: Schlein[11] (1974); C: Harmon[12] (1978); D: Pritchard[13] (1976); E: Roper[14] (1975); F: Dee[15] (1974); G: Amis[16] (1981); H: Stevens[17] (1970).

presented in Figure 1.

Beside improvements in materials and models, different operative techniques have arisen, each with their own advantages. In general, two approaches can be distinguished; the triceps sparing and non-triceps sparing approach. The non-triceps sparing approach, entails the triceps tendon to be split longitudinally or reflected from its insertion at the olecranon and at the end of surgery, needs to be repaired, yielding good results[22].

In the triceps sparing approach, a Chevron osteotomy of the olecranon is performed, distal to the triceps insertion, which is turned aside *en-bloc* with the triceps tendon attached. After insertion of the TEA this Chevron osteotomy is repaired. A study showed the triceps-sparing approach may result in better range of motion and a lower chance of infection compared to the triceps-detaching approach[23].

The human factor of gained experience on TEA surgery, together with improved materials, has led to positive results regarding clinical outcome and revision rates. Also larger trials and level 4 follow-up data coming from registries have enabled more thorough research on TEA, contributing to evidence-based patient care.

## THE PRESENT

In today's practice, the indications for elbow arthroplasty include all kinds of incapacitating elbow diseases, such as primary osteoarthritis, post-traumatic osteoarthritis, rheumatoid arthritis, comminutive elbow fractures, post-traumatic deformities and oncologic disease. However,

unlike in hip and knee arthroplasty, the main indication is not primary osteoarthritis. In 1997, the main indication for TEA in the State of New York, United States, was rheumatoid arthritis. However, in 2006 a shift was seen to trauma as the main indication for TEA[5].

Today, both the linked sloppy hinged and unlinked TEA's are available. Fixed hinge models are not used contemporarily. According to the patient's pathology and surgeons' preferred choice of the type of implant is often made pre-operatively. A "third generation" type of TEA is currently available, which allows the surgeon to decide during surgery to place a linked or unlinked implant.

Survival rates of different types of TEA have improved in the past four decades to around 90% after 5 years[24,25]. Cumulative revision rates after four to five years for fixed-hinge models is 13%, for sloppy hinge models 11%, and for unlinked models 13%[25]. In the short term, the main cause of failure is infection, while in the long term, the main cause is aseptic loosening by prosthetic wear[25,26]. When compared per group, the fixed-hinge models have a loosening rate of 11%, the sloppy hinged models 5% and the unlinked TEA's 10%[25].

Deep, periprosthetic infection is a serious complication in arthroplasty surgery, since it requires aggressive treatment in order to preserve the implant without removing it, as well as other problems to patients. To counter the infection rate, the use of per-operative antibiotics has become standard and maximum aseptic measures are taken during surgery, such as double gloving and laminar flow[27]. Use of antibiotic-containing bone cement has lowered the deep infection rate to

around 8%[4,28].

The use of bone cement might play a role in the aseptic loosening rate. A comparative study of cemented and uncemented ulnar components showed a lower rate of loosening in cemented components[29]. To avoid the use of bone cement and still achieve a firm bone-implant interface, several prosthetic coatings are available. These use the concept of bone ingrowth or osseointegration. The prosthesis is coated with hydroxyapatite, the molecular equivalent of bone. Human osteoclasts can dissolve the coating and attract osteoblasts to replace the coating with human bone[30]. A different concept is the ability to host osteoblasts in an optimal environment to enhance the intertwining of bone and implant. This can be accomplished with tantalum mesh or titanium beads[31-33].

To prevent metallosis, which might occur in metal-on-metal articulations, and to minimize shear stress between components, a plastic inlay is used. Depending on the type of arthroplasty, the inlay is either a polyethylene layer between unlinked components (iBP) or a bushing (Discovery, Coonrad-Morrey). These inlays are made of different materials, which aim to minimize wear of the prosthesis.

Wear debris can trigger "particle disease", which in turn leads to arthroplasty component loosening and eventually failure[34]. Analysis of loosened TEA's showed presence of wear debris (predominantly bone cement, polyethylene and metal) in surrounding tissue, due to wear of the polyethylene interface[35]. The inlay wear can be lowered by either crosslinking the polymers or adding substances, such as vitamin E[36,37]. However, no long-term follow-up results are published for elbow arthroplasty.

Patient-reported outcome scales nowadays have a more prominent role in assessing elbow function. Outcome measures have shifted from solely surgeon-opinion, to patient-oriented questionnaires, which focus on activities of daily life[38]. In a review on outcomes after TEA, the patient-reported outcomes were good or excellent in 78% of cases[25]. The function assessed by improvement of range of motion, was better in fixed-hinge models and sloppy hinged models (38 degrees and 35 degrees, resp.) than for unlinked models (20 degrees)[25].

## THE FUTURE

Considering the present issues of aseptic loosening and infective complications of elbow arthroplasty, there is obvious room for improvement. Ongoing insights in elbow kinematics might guide implant designers in refining TEA, not only by design but also by choice of material[21]. The previously mentioned third generation TEA models might provide a good choice when a pre-operative decision on linked or unlinked TEA is not yet clear. Also, restoring the radiocapitellar joint by inserting a radial head prosthesis is possible.

Because of the increasing use of elbow arthroplasties,

an inevitable problem occurs; revision arthroplasty. Because of good results, orthopedic surgeons may perform TEA's with less difficulty in incapacitated patients than several decades ago. Besides, treatment of systemic diseases, such as rheumatoid arthritis has improved, with an overall increase in the quality of life, exposing TEA to a longer period of use. Results on TEA revision are promising; in a recent study revision led to pain relief and improved range of motion after failure of primary TEA[39].

The improved overall results might also question the need of post-operative functional restrictions, such as restricted lifting activities. These movements lead to shear distracting forces on the bone-implant junction and are therefore theoretical risk factors for implant loosening. In linked TEA types the pulling forces during lifting are transferred more to the humeral component than in unlinked TEA, since unlinked TEA requires ligaments and muscles to remain stable in this situation and is not connected to the ulnar component. However, no studies on post-operative rehabilitation are published, yet high-demanding patients show worse overall implant survival compared to low-demanding patients[40]. Therefore, research on post-operative management should be conducted to determine both mechanical factors influencing implant survival and optimal functional improvement.

Furthermore, several aspects on TEA research itself should be addressed. By setting up large implant registries, trends in the long-term can be studied. In Scotland, Sweden, Norway, the Netherlands and New Zealand, data on elbow arthroplasties are reported on a routine basis[4,40,41]. If this could be expanded to more countries, larger cohort studies with better follow-ups are possible[42]. Large registries also raise the possibility to assess practical questions, for example, a recommended minimum of annual cases to retain optimal surgical results. The Scottish and Finnish arthroplasty registers show that high-volume specialized centers yield better implant survival[4,40].

Use of pre-operative plain radiographs allows to plan implant size on beforehand, to optimize concordance between the pre-operative native elbow joint and the arthroplasty. Concerning the planning of the implant size, a radiograph-based planning tool is available, with good results in hip and knee arthroplasty. However, even though the intra-observer variability is good, the predictive value of this form of planning is insufficient[43]. A three-dimensional planning tool would possibly give more accurate information on TEA placement and sizing[44].

Another question is the use of three-dimensional guiding. Creating three-dimensional structures can be seen in two ways, creating the implant itself or re-creating the diseased elbow. Firstly, unlike Venable described in 1952, patient-specific implants could be made without preceding surgery, according to preoperative CT-scans. However, on a large scale, this might be too labor-intensive to plan and too expensive to fabricate. Therefore, patient-specific implants could be

used in cases, where usual implants are not suitable.

Secondly, re-creating the diseased elbow could be of beneficial use in complex cases with severe deformation, *e.g.*, the surgeon practicing on a model beforehand. This is already a method used in maxillofacial surgery[45]. In knee arthroplasty, patient-specific cutting guides, based on pre-operative MRI- or CT-scans, are available for difficult cases[46-49].

## CONCLUSION

The knowledge on elbow arthroplasty has improved greatly in the past seven decades. With more encouraging results and a more widespread awareness, further improvements can be made. By setting up databases on implants, a structured analysis on adverse factors can be made to identify further improvable factors. Advances in materials and technical aids, such as three-dimensional printers, might improve postoperative outcomes.

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# Review of management of unstable elbow fractures

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## Abstract

Stable and painless elbow motion is essential for activities of daily living. The elbow joint is the second most commonly dislocated joint in adults. The goals of treatment are to perform a stable fixation of all fractures, to achieve concentric and stable reduction of the elbow and to provide early motion. The treatment modality for complex elbow instability is almost always surgical. The treatment objectives are anatomic reduction, stable fixation, and early rehabilitation of the elbow. The common complications of these unstable fractures include recurrent instability, stiffness, myositis ossifications, heterotopic calcification, and neurovascular dysfunction. We analyzed the management of complex elbow fractures and instabilities on the basis of recent literature and suggested possible guidelines for the treatment in this paper. In conclusion, recognition of the injury pattern and restoration of the joint stability are the prerequisites for any successful treatment of an unstable elbow injury.

Key words: Transolecranon fracture; Coronoid fracture; Monteggia injury; Radial head fracture; Terrible triad

Core tip: As the elbow joint is the second most commonly dislocated joint in adults, we aimed to analyze the management of complex elbow fractures and instabilities, on the basis of recent literature and suggested possible guidelines for the treatment in this paper.

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## INTRODUCTION

Basic elbow function requires stable and painless elbow motion. The three articulations, namely the ulnotrochlear, radiocapitellar, and proximal radioulnar joints, provide elbow flexion/extension and supination/pronation. Static and dynamic constraints create stability of the elbow joint. The ulnohumeral articulation, anterior bundle of the medial collateral ligament, and lateral collateral ligament complex form the primary static constraints. The joint capsule and radial head are among the secondary static constraints. The dynamic constraints, such as the common flexor and extensor

muscle groups, are any muscles crossing the elbow joint that exert a compressive force on the joint[1].

The elbow is the second most commonly dislocated joint in adults[2]. The dislocations may be complex or simple. When dislocations are associated with fractures, they are designated as complex. The reported annual incidence of simple and complex elbow dislocations is 6.1 per 100000 patients[3]. Radial head fractures/dislocations, coronoid fractures, terrible triad injuries, transolecranon fracture-dislocations, and Monteggia-like lesions can be listed as common causes of complex elbow injuries.

Complex elbow fractures and instability typically present with edema, tenderness, pain in active/passive movement, and restriction of motion. A fall onto the extended outstretched hand or a direct trauma to the elbow is usually described as the mechanism of the injury. Anteroposterior and lateral standard radiographs and computerized tomography scans (either standard or 3D) are needed to properly evaluate the bone injuries and to accurately plan their surgical treatment. Neurovascular examination and documentation of the injuries before and after any manipulation are of critical importance. A temporary fracture alignment with cast immobilization may be done until definitive surgery in patients with severe injuries.

The treatment modality for complex elbow instability is almost always surgical. The treatment objectives are anatomic reduction, stable fixation, and early rehabilitation of the elbow.

The common complications of these unstable fractures include recurrent instability, stiffness, myositis ossifications, heterotopic calcification, and neurovascular dysfunction.

In this review, we analyze the management of complex elbow fractures and instability on the basis of recent literature, and suggest possible guidelines for the treatment of these injuries.

## RADIAL HEAD FRACTURES

Radial head fractures are among the most common elbow fractures, occurring in up to 20% of all elbow injuries[4]. Radial head fractures are mostly associated with complex injuries like elbow dislocation, and lateral collateral ligament (LCL) and medial collateral ligament (MCL) tears. Impaction fractures of the capitellum may also be associated with radial head fractures, and can easily be overlooked[5]. Only about 5% of cases have a radial head fracture as an isolated injury[6,7]. In a large incidence study[8], the mean age was found to be 36 years and the female-to-male ratio was 47.7/53.3. A thorough physical examination is essential to diagnose associated ligament injuries. Fluoroscopy may be used to confirm an MCL injury if medial pain and ecchymosis are present. If the MCL is injured, the role of the radial head in valgus resistance increases up to 30%[9,10]. Stable anatomic reconstruction of the radial head is the primary objective of the treatment.

The Mason classification system[11] divided radial head fractures into three categories: type Ⅰ, non-displaced fractures; type Ⅱ, displaced partial head fractures; and type Ⅲ, comminuted displaced fractures involving the whole head.

Modifications to the Mason classification were introduced to guide treatment. The Hotchkiss modification[12] defines type Ⅰ fractures as non-displaced fractures (< 2 mm displacement) without mechanical blockage that do not require surgery, type Ⅱ fractures as displaced fractures (> 2 mm displacement) of the radial head or neck that lack severe comminution, may have mechanical blockage to movement, and usually require open reduction and internal fixation, and type Ⅲ fractures as severely comminuted fractures of the radial head and neck. Satisfactory reconstructions of these fractures are not possible, and therefore the radial head is either excised or replaced with a prosthesis[13]. Fragment excision is avoided in complex elbow instability to prevent valgus instability[14]. If there is an associated LCL rupture, it should be repaired after appropriate management of the radial head fracture, either by fixation or by prosthetic replacement. Suture anchors or transosseous sutures can be used for the reconstruction. The joint stability should be confirmed by dynamic fluoroscopic examination. If residual instability persists, MCL reconstruction and/or dynamic elbow fixation should be done[15,16].

## CORONOID FRACTURES

The coronoid process plays a pivotal role as an anterior buttress in providing elbow stability. Although coronoid fractures may occur in isolation, they are more commonly seen as a component of unstable elbow fractures[17].

The classification proposed by Moon *et al*[18], which defines anteromedial facet lesions, may be better for guiding the surgical management of coronoid fractures. Type Ⅰ injuries involve fractures of the coronoid tip, and are divided into two subtypes based on the fracture size. Subtype 1 fractures are smaller than 2 mm, and subtype 2 fractures are larger than 2 mm. In type Ⅱ injuries, the anteromedial aspect of the coronoid is fractured. These injuries are divided into three subtypes based on the anatomic location. Subtype 1 fractures involve the rim, subtype 2 fractures involve the rim and the tip, and subtype 3 fractures involve the rim and the sublime tubercle with or without the tip. Type Ⅲ fractures are basal coronoid fractures involving at least 50% of the height of the coronoid. They are divided into two subtypes depending on whether the fracture involves the base of the olecranon. Stable fixation and ligament repair are essential for the treatment of coronoid fractures[19,20].

## TERRIBLE TRIAD INJURIES

Terrible triad injuries have the most common complex pattern. They comprise a radial head fracture and elbow

**Table 1  Reviewed studies investigating unstable elbow fracture diagnosis and treatment**

| Author | Ring et al[16] | Chemama et al[22] | Konrad et al[26] | Mouhsine et al[29] | Zeiders et al[21] | Winter et al[13] | Mortazavi et al[30] | Strauss et al[27] |
|---|---|---|---|---|---|---|---|---|
| Year | 2002 | 2010 | 2007 | 2007 | 2008 | 2009 | 2006 | 2006 |
| Mean age (range) | 36 (17-62) | 46 (26-75) | 42.1 (21-72) | 54 (22-82) | NA | 40 (18-77) | 35 (22-58) | UHD+: 46.8 UHD-: 55 |
| *n* (female/male) | 56 (21/35) | 23 (7-16) | 63 (22/41) | 14 (8/6) | 32 (NA) | 13 (4/9) | 8 (1/7) | UHD+: 6 (2/4) UHD-: 17 (12/5) |
| Radial fracture Mason classification | Type 2: 30 Type 3: 26 | Type 1: 2 Type 2: 9 Type 3: 10 Radial neck: 2 | Type 2: 7 Type 3: 9 | NA | NA | 13 non-reperable RH fracture | Type 1: 1 Type 3: 1 | UHD+: Type 1: 1 Type 2: 2 Type 3 : 3 |
| Coronoid fracture classification R-M | NA | Type 1: 16 Type 2: 7 | NA | NA | NA | Type 1: 5 Type 2-3: 8 | Type 3: 4 | UHD+: Type 1: 4 Type 3: 2 |
| Monteggia fracture classification (BADO) | NA | NA | Type 1: 19 Type 2: 37 Type 3: 5 Type 4: 2 | NA | NA | NA | NA | Type 2: 6 (UHD+) Type 2: 17 (UHD-) |
| Time to surgery | NA | NA | First 24 h | NA | NA | 1.9 d (1-4) | 3.8 d | NA |
| Monteggia fracture Ulna treatment | NA | NA | Bado 1, 3, 4 (26 3.5 mm DCP) Bado 2 (26 plate, 11 tension band) | NA | NA | NA | NA | UHD+: Small fragment plates UHD-: Small fragment plates |
| Transolecranon fracture treatment | NA | NA | NA | K-wire and tension band: 7 3.5 mm 1/3 plate: 2 3.5 mm DCP: 1 3.5 mm recon. plate: 4 | NA | NA | 3.5 mm AO recons. Plate: 7 K-wire and tension band: 1 | NA |
| Coronid treatment | NA | None: 10 TOS: 3 Anchor: 2 Screw: 4 Plate: 1 Resection: 3 | Lag screws through the ulnar plate or indirect repositioning | NA | 32 patients repair of coronoid brachialis Complex pull-through suture tec | NA | 4 patients interfragmentary Screws through the plate | UHD+: Type 3 (lag screws) |
| Radial head treatment | Mason 2: 26 (screw), 4 (plate, screw) Mason 3: 22 (plate, screw), 4 (screw) | 13 mini screws Radial neck: 2 plate RHP: 4 PRHR: 2 TRHR: 2 | 46 luxation CR 12 RH ORIF 4 RH resection | NA | 6-intact RH 7-reconst 19-prothesis | 13 RHP | 1 type 1: Mini screws 1 type 3: Total exicision RH | Type 1, 2: mini fragment plates Type 3: 3 replacement |
| LCL repair | NA | NA | NA | None | 18 (anchor) 12 (mcl + lcl rep) | No: 4 abs suture Used for repair | NA | UHD+: 6 |
| MCL repair | NA | NA | NA | None | 2 (ancor) 12 (mcl + lcl rep) | NA | NA | UHD+: 0 |
| External fixator | NA | None | NA | None | 21 hinged ex-fix | NA | NA | UHD+: 1 hinged ex-fix |
| Follow-up | 48 mo | 63 mo | 8.4 yr (5-14) (47 patients) | 42 mo (7-84) | 3 yr (1-5) | 25 mo (15-48) | 37.4 mo (10-50) | UHD+: 28 mo (14-48) UHD-: 29 (12-60) |
| Range of motion | MFA Mason 2: 119 Mason 3: 111 | MFA: 109 MSA: 64 MPA: 70 | MFA: 97.5 MFR: 125 | MFA: 103 MSA: 76 MPA: 68 | MFA: 100 | MFA: 120 | MFA: 93 MFR: 157.5 | UHD+: MFA 95, MSA 55, MPA 50 UND-: MFA 122, MSA 67, MPA 60 |

| Mean score | Mason 2: BM: 92 Mason 3: BM: 86 | MEPS: 87 (75-100) 14 patients result | BM: 87.2 (45-100) DASH: 17.4 (0-70) | BM: 82 (78-100) | DASH: 23 (19/28) | BM: 86.5 (55-100) | BM: 88 (71-100) ASES: 89 (69-100) | UHD+: DASH 34 (0-80), BM: 73.8 UHD-: DASH 23 (0-70), BM: 83 |
|---|---|---|---|---|---|---|---|---|
| Unsatisfactory | Mason 2: 4 Mason 3: 14 | BM: 0 | BM: (9 fair) (4 poor) result | BM: 2 fair 2 poor | | 2 (stiffness, infection) | BM: 1 fair | UHD+: 2 UHD-: 7 |

NA: Not applicable; UHD: Ulnohumeral dislocation; RH: Radial head; R-M: Roger Morrey; DCP: Dynamic compression plate; TOS: Transosseous suture; RHP: Radial head prosthesis; PRHR: Partial radial head resection; TRHR: Total radial head resection; MFA: Mean flexion arch; MSA: Mean supination arch; MPA: Mean pronation arch; MFR: Mean forearm rotation; BM: Broberg and Morrey index; MEPS: Mayo elbow performance score; DASH: Disabilities of the arm shoulder and hand; ASES: American Shoulder and Elbow Surgeons assessment system; LCL: Lateral collateral ligament; MCL: Medial collateral ligament.

dislocation along with a coronoid fracture. Both medial and lateral compartments can be exposed through a posterior incision. The Kocher approach can be used for the radial head fracture. Hotchkiss type I and type II radial head fractures can be fixed with headless screws or a plate[12]. Prosthetic replacement is mandatory for comminuted radial head fractures (type III) to avoid chronic instability. There is often a comminuted type 1 fracture in the coronoid, and it can usually only be fixed with a transosseous suture. If there is an isolated fragment that is sufficiently large, fixation with K wires or screws can be done[19]. The LCL is repaired last, and elbow stability is assessed by fluoroscopy. In the presence of a residual instability, the MCL should also be repaired or a hinged external fixator should be applied[7,21,22] (Table 1).

## MONTEGGIA-LIKE LESIONS

Monteggia injuries comprise a fracture of the ulnar shaft with an associated radial head dislocation. Monteggia originally described the lesions as a fracture of the proximal third of the ulna and an anterior dislocation of the proximal epiphysis of the radius[23]. Bado[23] classified these injuries by primarily focusing on the radial component. Jupiter *et al*[24] modified this classification by defining subtypes for the posterior Monteggia lesions (Bado type 2). Ulnohumeral dislocation, radial fracture, proximal and/or distal radioulnar dislocation, and interosseous membrane lesions may also accompany the ulnar fracture and radiohumeral dislocation. Each of these must be recognized and treated. The varying combinations of these injured structures explains the complexity and diversity of the management procedures.

Anatomic reduction and stabilization of the ulna and the ulnohumeral joint is the primary objective of surgical treatment for posterior elbow fracture-dislocations[25]. The radial head fracture is addressed initially. If the radial head cannot be salvaged satisfactorily, radial head arthroplasty is preferred. To size the radial head properly, the ulnar length should be restored by a provisional fixation[17]. The coronoid process is stabilized after the ulnar shaft fracture has been addressed, and the olecranon is fixed with a dorsal plate. Finally, ligamentous components of the injury are addressed[26,27] (Table 1).

## TRANSOLECRANON FRACTURE-DISLOCATIONS (ANTERIOR OLECRANON FRACTURE-DISLOCATIONS)

The radial head is dislocated anteriorly with an associated olecranon fracture in this injury pattern[28]. Two subtypes have been described: One with a simple olecranon fracture, and one with a comminuted olecranon fracture[28]. The second subtype is more common and may be associated with trochlear and coronoid fractures. This injury pattern is distinct from the anterior Monteggia (Bado type 1) lesion, because in transolecranon fracture-dislocation, the ulnohumeral stability is lost but the radioulnar relationship remains intact. Bony disruption is the main reason for the failure of the ulnohumeral joint rather than the ligamentous structures.

Anatomic reduction with particular attention to restoring the ulnar length and greater sigmoid notch is essential in the treatment[29] (Table 1). Restoration of the ulnohumeral anatomy is crucial to prevent radiocapitellar instability or subluxation[25,30] (Table 1).

## CONCLUSION

Surgical treatments of complex elbow fracture dislocations are among the most challenging procedures for orthopedic surgeons. Interpretation of the underlying mechanisms for elbow instability and accurate identification of the injured structures are crucial for surgical planning. Stable elbow fracture fixation is important for early elbow motion and avoiding joint stiffness. Recognition of the injury pattern and restoration of the joint stability are the prerequisites for any successful treatment of an unstable elbow injury.

In this review, we have examined the diagnosis, classification, and treatment of unstable elbow fractures. Future studies should be conducted to determine the optimal management strategies, the role of ligament reconstruction, and reductions in the complication rate.

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*MINIREVIEWS*

# Calcific tendinitis of the rotator cuff

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## Abstract

Calcific tendinitis within the rotator cuff tendon is a common shoulder disorder that should be differentiated from dystrophic calcification as the pathogenesis and natural history of both is totally different. Calcific tendinitis usually occurs in the fifth and sixth decades of life among sedentary workers. It is classified into formative and resorptive phases. The chronic formative phase results from transient hypoxia that is commonly associated with repeated microtrauma causing calcium deposition into the matrix vesicles within the chondrocytes forming bone foci that later coalesce. This phase may extend from 1 to 6 years, and is usually asymptomatic. The resorptive phase extends from 3 wk up to 6 mo with vascularization at the periphery of the calcium deposits causing macrophage and mononuclear giant cell infiltration, together with fibroblast formation leading to an aggressive inflammatory reaction with inflammatory cell accumulation, excessive edema and rise of the intra-tendineous pressure. This results in a severely painful shoulder. Radiological investigations confirm the diagnosis and suggest the phase of the condition and are used to follow its progression. Although routine conventional X-ray allows detection of the deposits, magnetic resonance imaging studies allow better evaluation of any coexisting pathology. Various methods of treatment have been suggested. The appropriate method should be individualized for each patient. Conservative treatment includes pain killers and physiotherapy, or "minimally invasive" techniques as needling or puncture and aspiration. It is almost always successful since the natural history of the condition ends with resorption of the deposits and complete relief of pain. Due to the intolerable pain of the acute and severely painful resorptive stage, the patient often demands any sort of operative intervention. In such case arthroscopic removal is the best option as complete removal of the deposits is unnecessary.

Core tip: This review article discuss calcific tendinitis of the rotator cuff regarding the definition, prevalence, pathology, pathogenesis, natural history, clinical presentation, classification, diagnosis and various treatment modalities.

## INTRODUCTION

Calcium deposits within the rotator cuff tendon is a common shoulder disorder[1]. Calcium deposits may be in the form of calcific tendinitis or dystrophic calcification.

Calcific tendinitis is calcification within a viable and well vascularized rotator cuff. It occurs within the midsubstance of the cuff, 1 to 2 cm proximal to its insertion. Classically, the condition will end by spontaneous resolution and it is uncommon to see other signs of degenerative changes[2].

Dystrophic calcification is calcification within a non-viable and poorly vascularized rotator cuff. It occurs at the insertion site or at the edges of a cuff tear. Classically, the condition worsens by time and it is common to see other signs of degenerative changes[2].

## PREVALANCE

Calcific tendinitis usually occurs during the fifth and sixth decades of life. It occurs within the supraspinatus tendon in almost 50% of cases. It is more common in females (60%). It occurs more commonly with sedentary workers than with heavy labor workers (45% are house wives)[3-5].

## PATHOGENESIS AND NATURAL HISTORY

Controversy exists over the exact cause of calcific tendinitis. Burkhead[6] and Gohlke[7] proposed that it is a degenerative process that involves necrotic changes of tendon fibers that progress into dystrophic calcification.

Mclaughlin[8] believed that it proceeded from focal hyalinization of the fibers that become fibrillated and detached from the tendon, thus wounding up into rice-like bodies that later undergo calcification.

On the other hand, Uhthoff[9] pointed out that this may be true regarding dystrophic calcification. But regarding calcific tendinitis they believed that this explanation was most unlikely. They argued that calcific tendinitis occurred in viable and well vascularized tissues and thus could not be a degenerative process, instead they suggested that it was a reparative process progressing through a predictable disease cycle[9-12].

The calcium deposits may have a chalk-like consistency or a fluid consistency or a mixed one[13].

## CLASSIFICATION AND CLINICAL PRESENTATION

Many classifications have been suggested to describe calcific tendinitis. Some classified it according to the severity of the symptoms into acute, subacute and chronic[14]. Others classified it according to the radiological form into two categories. The first with localized, discrete, dense and homogenous deposits with spontaneous healing tendency and the second with diffuse, fluffy and heterogeneous deposits characterized by delayed and slow healing[15]. The French society of arthroscopy divided the condition into four types: Type A (20%) with homogenous deposits with well defined edges; Type B (45%) with heterogeneous fragmented deposits with well defined edges; Type C (30%) with heterogeneous deposits with ill defined edges and Type D which is not calcific tendinitis but degenerative dystrophic calcifications at the rotator cuff insertion[16].

Uhthoff *et al*[12] were the ones who described the complete cycle of the calcium deposits and explained the development of its natural history. They divided the condition into formative and resorptive phases. Lying within the two phases most authors motioned the presence of three stages; pre-calcification (silent), calcification (impingement) and post-calcification (acute)[17-20].

The chronic formative phase results from transient hypoxia that is commonly associated with repeated microtrauma and sometimes with a significant single trauma. This results in increased proteoglycan levels that induce tenocyte metaplasia into chondrocytes. This is followed by calcium deposits, mainly into the matrix vesicles within the chondrocytes. These deposits develop into bone foci that later coalesce.

During the acute resorptive phase the periphery of the calcium deposits shows vascularization with macrophage and mononuclear giant cell infiltration together with fibroblast formation. This produces an aggressive inflammatory reaction with inflammatory cell accumulation, excessive edema and rise of the intra-tendineous pressure. This leads to severe pain which is attributed by some to secondary impingement resulting from the increased tendon size, or due to rupture of the deposits into the subacromial space or into the bursa.

During the post-calcification stage the fibroblasts lay down collagen (mainly type II) that fills the gap. This will maturate into collagen type I within 12 to 16 mo[9,11,12].

The clinical presentation is highly variable and depends on the phase the patient is passing through. During the chronic formative phase that may extend anywhere from 1 to 6 years, the patient may be completely asymptomatic. In some cases the condition will only be discovered accidentally. Some patients may present with symptoms that mimic mild impingement. However during the acute resorptive phase, the patient usually presents with severe symptoms that may extend from 3 wk up to 6 mo. In general, the more severe the symptoms are, the shorter the duration of the condition is. The patient presents with tremendous pain all over the shoulder with tenderness over the supraspinatus insertion. Pain commonly extends to the root of the neck

with difficulty during overhead activity associated with muscle spasm. It is very difficult to perform any of the special tests due to the unbearable pain.

## IMAGING

Radiological investigations confirm the diagnosis and may even make the diagnosis in asymptomatic cases. It also suggests the phase of the condition and is used to follow its progression.

They include conventional X-ray in true antero-posterior, lateral and outlet views. Deposits within the subscapularis may be detected by anteroposterior view in external rotation. In internal rotation, the deposits within the infraspinatus and teres minor may be detected. "Skullcap appearance" indicate rupture of the deposits within the bursa[21].

Ultrasonographic examination was reported to be more sensitive in detecting the calcium deposits within the cuff[22].

Computed tomography allowed better localization of the deposits[21]. Although routine conventional X-ray allowed detection of the deposits, magnetic resonance imaging (MRI) studies allow better evaluation of any coexisting pathology. The deposits present with a low intensity signal in the T1 weighted images. In the T2 weighted images there may be perifocal low intensity signal denoting surrounding edema[23].

The thinned out cuff lateral to the deposits may be falsely interpreted as a cuff tear. MRI arthrography was more beneficial to avoid such false conclusions[23].

## TREATMENT

Various methods of treatment have been suggested. The appropriate method should be individualized for each patient depending on proper understanding of the pathophysiology and natural history of the condition, as well as proper clinical and radiological assessment of the patient, and finally accurate determination of the stage at which the patient presents.

The treatment may be "conservative" including pain killers and physiotherapy, or "minimally invasive" as needling and puncture and aspiration, or "operative" whether arthroscopic or open.

Due to the intolerable pain of the acute and severely painful resorptive stage, the patient often demands any sort of intervention despite explaining to him that the condition is probably resolving.

Since the natural history of the condition ends with resorption of the deposits and complete relief of pain, usually conservative measures are successful in most of cases, reaching 80% in some studies and even 99% in others[12,24].

During the acute stage the aim is to relief of pain. The efficacy of non-steroidal drugs may be doubtful with frequent need to narcotic medications.

### Physiotherapy
Some authors suggested physiotherapy including range of motion exercises to avoid gleno-humeral stiffness and idiopathic frozen shoulder. However there is no evidence that calcific tendinitis causes gleno-humeral capsular contracture[25].

There is no solid evidence that different physical modalities including infrared, ultrasound, or deep heat have any effect on the natural history of the condition.

### Extracorporeal shock wave
Extracorporeal shock wave (ECSW) has been used to treat symptomatic patients passing through the chronic formative phase with definite radiological evidence of calcium deposits[26].

Most authors report short term symptomatic improvement[27]. But ECSW was not free from complications, that included transient bone marrow edema and even reported cases of humeral head necrosis[28,29].

Most authors reported that the improvement is dose dependant, with better results following one or two sessions of high energy applications[30].

### Needling or puncture and lavage
Minimally invasive techniques include needling or puncture and aspiration. These techniques were suggested by many authors aiming at decompressing the deposits and thus relieving the pain. They suggested that direct puncture of the deposits would shorten the natural history of the condition and accelerate resorption in 50% of cases[31].

Since the fifties of the last century some authors recommended blind needling of the deposits with intralesional local anesthetic injection reporting pain relief in 85% of cases. They reported that the amount of deposits removed didn't affect the outcome and accordingly concluded that pierce opening of the deposits was the essential step and not the calcium removal[32,33]. In the sixties Depalma and Kruper[14] popularized blind needling of the deposits without any radiological localization, with good results. Clement reported pain relief within 24 h following repeated blind needling of the deposits (15 to 20 times), after local anesthetic and corticosteroid injection into the subacromial space. He referred the patients to ultrasonic treatment within a few days. He claimed that this would cause active hyperemia that would enhance deposit absorption[34]. Most authors reported very good results after performing needling under fluoroscopic and, or ultrasonographic guidance[35,36].

Local corticosteroid injection (whether intralesional or into the subacromial space) following the needling of the deposits, is recommended by some authors with good results and some suggested two or more injections. Many studies showed no evidence that corticosteroid injection improved the results[36,37]. Some reported that corticosteroids injection was short acting and only symptomatic. Other surgeons argued that corticosteroids

would reduce the tendon healing process[38]. Neer[39] disagreed with this claim.

Many authors recommended dual needling and lavage for cases with calcium deposits[40-42]. Needling is no new technique as it has been described over a century ago by Flint in 1913 as reported by Codman in 1934[6].

After all of the various needling techniques the patient should be instructed to rest the shoulder for a short period (1 to 2 d) followed by gradual return to daily activities.

The patient should also be forewarned that a successful full recovery may take 3 to 6 mo. During the chronic formative stage the symptoms are usually mild and no intervention is needed. Yet some authors suggested needling (whether blind needling or under radiological guidance) suggesting that direct puncture of the deposits would shorten the natural history of the condition and accelerate deposits resorption[43]. Non-steroidal drugs may be used every now and then.

The true debate concerning needling or puncture and lavage is the fact that the acute and severe symptoms are almost always associated with an expedient resolution of the condition. Thus, any form of treatment at this point will ultimately be a "success".

### Operative intervention
**Indications:** Many authors suggested that the indications for operative intervention include progression of symptoms that interferes with the daily activities after failure of conservative measures[44,45].

Neer stated that operative indications include long standing symptoms after failure of conservative measures in the presence of multiple, hard and gritty deposits. He rarely resorted to operative intervention and suggested that residual tendinopathy would follow[39].

Most authors starting from Burkhead[6], passing through Lippmann[1], to Mclaughlin[8], and up till today[45,46] agree that surgical removal of rotator cuff calcium deposits end with good permanent results. They agree that the indications include symptomatic patients after failure of conservative measures with radiological evidence of relatively homogenous calcium deposits.

During the resorptive stage, conservative measures were recommended as the natural history of the condition would end with complete resolution of the deposits and the symptoms. Yet operative intervention is to be considered upon the patient's demand due to the intolerable pain despite the conservative measures.

**Open surgery:** It is performed through a deltoid splitting incision, the site of which may be modified to allow the best access to the exact location of the deposits. The deltoid fibers are separated and the deposits are split-open along the direction of the cuff fibers. Usually the deposits are readily apparent as a bulge within the cuff. The deposits commonly burst out when opened. Open surgery has a high success rate in complete removal of the calcium deposits but with some intraoperative

complications[47-49]. Some authors suggested resuturing of the cuff if a significant gap is left behind. The benefit of this step is unclear[13,50]. This is followed by a period of shoulder rest (5 to 7 d), with gradual return to daily activities over a 4 to 6 wk period of time.

Most authors reported that complete intraoperative removal of the deposits was unnecessary as it didn't significantly affect the final clinical outcome. Thus total removal was not essential and sometimes not possible without substantial damage to the tendon[49,51]. In most cases partial removal of the deposits will finally lead to total resorption. This was reinforced by other studies[46,52].

**Arthroscopic management:** Nowadays, open surgery is rarely used to remove calcium deposits as arthroscopy offers a much better choice.

Arthroscopic calcium deposits removal was quite effective, although it may fail to completely remove the deposits compared with open surgery[47-49]. But as long as complete removal of the deposits was unnecessary, then arthroscopic removal was clearly a better option. Studies showed that the rate of full-thickness rotator cuff tears after calcium deposits removal was quite low (3.9% after a 9-year follow-up)[53]. Accordingly, rotator cuff repair following calcium deposits removal was not mandatory. However, it was found that the intraoperative status of the rotator cuff had a significant influence on the functional results at follow-up[47]. In one study, the 2 patients of the 54 cases of the study (3.7%) who needed later rotator cuff repair showed obvious degeneration of the rotator cuff during the removal of the deposits. Accordingly, it should be recommended to repair the rotator cuff after the removal of calcium deposits, whenever the cuff appears to be noticeably degenerative[46].

Arthroscopic subacromial bursectomy should be performed to allow better visualization of the rotator cuff. In cases with shoulder impingement, subacromial decompression (acromioplasty) should be performed. The calcium deposits were identified as a bulge within the cuff tendon "calcific bulging sign"[51]. Then, *via* a lateral working portal, a half-moon arthroscopy knife may be used to open up the deposits along the fibers of the cuff. After that, a 3.5-mm motorized shaver was used to remove as much as possible of each deposit, only stopping short of causing any iatrogenic damage to the cuff.

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*ORIGINAL ARTICLE*

Case Control Study

# Patient specific guides for total knee arthroplasty are ready for primetime

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## Abstract

**AIM:** To present the radiological results of total knee arthroplasty (TKA) with use of patient specific matched guides (PSG) from different manufacturer in patients suffering from severe osteoarthritis of the knee joint.

**METHODS:** This study describes the results of 57 knees operated with 4 different PSG systems and a group operated with conventional instrumentation ($n$ = 60) by a single surgeon. The PSG systems were compared with each other and subdivided into cut- and pin PSG. The biomechanical axis [hip-knee-ankle angle (HKA)], varus/valgus of the femur [frontal femoral component (FFC)] and tibia (frontal tibial component) component, flexion/extension of the femur [flexion/extension of the femur component (LFC)] and posterior slope of the tibia [lateral tibial component (LTC)] component were evaluated on long-leg standing and lateral X-rays. A percentage of > 3° deviation was seen as an outlier.

**RESULTS:** The inter class correlation coefficient (ICC) revealed that radiographic measurements between both assessors were reliable (ICC > 0.8). Fisher exact test was used to test differences of proportions. The percentage of outliers of the HKA-axis was comparable between both the PSG and conventional groups (12.28% *vs* 18.33%, $P < 0.424$) and the cut- and pin PSG groups (14.3% *vs* 10.3%, $P < 1.00$). The percentage of outliers of the FFC (0% *vs* 18.33%, $P < 0.000$), LFC (15.78% *vs* 58.33%, $P < 0.000$) and LTC (15.78% *vs* 41.67%, $P < 0.033$) were significant different in favour of the PSG

group. There were no significant differences regarding the outliers between the individual PSG systems and the PSG group subdivided into cut- and pin PSG.

**CONCLUSION:** PSG for TKA show significant less outliers compared to the conventional technique. These single surgeon results suggest that PSG are ready for primetime.

**Key words:** Total knee arthroplasty; Patient specific matched guides; Patient matched instruments; Single surgeon; Alignment; Conventional instruments; Cutting guides; Pin guides

**Core tip:** Total knee arthroplasty (TKA) is one of the most successful and commonly performed surgical procedure for the treatment of severe knee osteoarthritis with excellent 15-20 years survivorships. This article provides an analysis on patient specific matched guides (PSG) between different manufacturers and the conventional technique and between pin- and cutting guides for TKA. In addition, we compared our results with previous studies (level 1 evidence), which are generally unambiguous, and show no radiological difference. However, in this trial, we do see difference in favour of the PSG technique.

## INTRODUCTION

Total knee arthroplasty (TKA) has been developed significantly over the last decades. Many changes have been made to improve both survival and functioning. A good postoperative biomechanical axis is one of the key elements for a good implant survival. Malalignment is associated with poor implant survivorship[1-4]. Several studies reported results of postoperative malalignment using conventional intramedullary alignment rods in TKA[5-9]. Computer navigation was introduced to cope with malalignment and instability in conventionally placed prostheses[10]. These days, revolutionary changes within the elective knee arthroplasty have taken place due to industry driven interventions[11]. Patient specific matched guides (PSG) for TKA is a relatively new technique to align the knee prosthesis, using 3D rapid prototyped disposable cut or pin guides that fits on the native anatomy of the individual patient[12,13]. This perioperative guiding technique eliminates the use of intra- and extra medullar rods to make bony resections. Previous published results on PSG suggest this to be a

good alternative to conventional instrumentation with comparable results, improved radiological outcome and reduced operation time and blood loss[7,13-23].

This prospective study on PSG between different manufacturers and conventional technique for the implantation of TKA was designed to address the following research questions: Is there a significant difference in outliers in alignment in the frontal and lateral plane between PSG and conventional TKA, secondly between the four individual different PSG systems and thirdly between cut- and pin PSG? We hypothesise that there will be fewer outliers with PSG TKA compared to conventional TKA without differences between different PSG systems and cut- and pin PSG.

## MATERIALS AND METHODS

Patients were operated for TKA with PSG systems from 4 different manufactures (Table 1). In daily practice the TKA system and PSG from the company Biomet is used. Between May 2013 and April 2014, 60 consecutive patients with debilitating osteoarthritis (OA) of the knee joint, who were eligible for primary TKA were included (Figure 1). Patients who were not eligible to undergo magnetic resonance imaging (MRI) due to metal artefacts around the knee joint from previous surgery, claustrophobia, movement artefacts during MRI scanning time, pigmented villonodular synovitis, implanted electronic devices and patients that refused to consent were excluded. TKA surgery was done using PSG and consisted of guides from 4 different TKA suppliers (Table 1). The conventional TKA group consisted of 60 patients who were randomly selected from a cohort ($n \geq 500$) as a comparison group. We did not match patients (*e.g.*, body mass index, gender, age and severity of OA) to avoid selection bias.

All patients gave informed consent to participate in this prospective study and were operated by a senior knee orthopaedic surgeon (NK) with extensive experience with PSG[15,16]. Patients were not blinded to the type of alignment method used. Three patients were excluded from the study and therefore did not receive the intervention as planned. A flowchart of the study design is shown in Figure 2. There were no significant differences in baseline demographics, as summarized in Table 2.

PSG and the conventional TKA surgery are extensively described in previous published studies[15,16]. Preoperative, a virtual 3 dimensional plan was made based on the imaging protocols of the different manufacturers (Table 1). Preferred component position of the prosthesis was planned to obtain a neutral biomechanical axis [hip-knee-ankle angle (HKA)] and position of the femoral [frontal femoral component (FFC)] and tibial [frontal tibial component (FTC)] components in the frontal plane. All settings during planning in the lateral plane were similar for all PSG systems: Femoral component flexion [flexion/extension of the femur component (LFC)] and tibial component posterior slope

**Table 1  Different industries with brand names, guide type, implant name and scanning modality**

|  | Dupuy-Synthes | Smith and Nephew | Zimmer | Biomet |
|---|---|---|---|---|
| PSG | Trumatch | Visionaire | PSI | Signature |
| Guides | Cut | Cut | Pin | Pin |
| Implant | Sigma CR | Genesis Ⅱ | NexGen | Vanguard CR |
| Imaging protocol | CT[1] | MRI[2] | CT or MRI[1] | CT or MRI[1] |

[1]Scan of the hip, knee and ankle join; [2]MRI of the knee joint with long leg standing X-ray. PSI: Patient-specific instrument; PSG: Patient specific matched guides; CT: Computed tomography; MRI: Magnetic resonance imaging; CR: Computed radiography.

**Table 2  Baseline demographics per alignment method, *n* (%)**

|  | Trumatch | Visionaire | PSI | Signature | Conventional | *P* value |
|---|---|---|---|---|---|---|
| Number of patients | 15 | 13 | 14 | 15 | 60 |  |
| Mean age, yr (range) | 72 (57-90) | 72 (63-82) | 69 (52-86) | 68 (56-74) | 65 (50-83) | 0.097 |
| Male | 6 (40) | 7 (54) | 7 (50) | 7 (47) | 34 (57) | 0.967 |
| Mean BMI (range) | 30 (23-36) | 30 (23-37) | 30 (26-36) | 30 (23-38) | 28 (21-37) | 0.373 |
| Severity OA |  |  |  |  |  |  |
| Moderate | 13 (87) | 11 (85) | 13 (93) | 14 (93) | 53 (88) | 0.991 |
| Severe | 2 (13) | 2 (15) | 1 (7) | 1 (7) | 7 (12) | 0.959 |

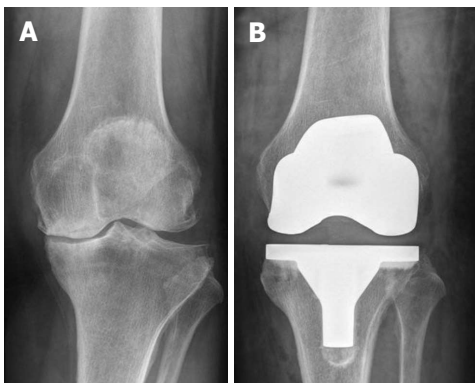PSI: Patient-specific instrument; BMI: Body mass index; OA: Osteoarthritis.



**Figure 1  Anterior-posterior radiograph of a left knee of a female patient.** A: Preoperative severe osteoarthritis; B: Postoperative with the Sigma CR, total knee arthroplasty (Depuy) *in situ*.

[lateral tibial component (LTC)] were set at 3°. The final approval of settings was done by the operating surgeon (NK). After approval, the disposable cut or pin guides (Table 1) for perioperative alignment were manufactured and used during surgery. A midline approach was used and a cemented prosthesis implemented in all cases (Table 1). The guides were designed to make contact with osteophytes and therefore it was not allowed to remove these prior to the bony cuts. The same procedure was performed in the conventional group, except for the standard conventional rods for femur and tibia with the same implant as the Signature group (Vanguard Complete Knee System, Biomet, Warsaw, INC). Conventional rods were used to align the position of the cutting blocks: LFC and LTC were set at 0°.

All patients received a multimodal pain protocol including spinal or general anesthesia and local infil-

tration analgesia without a drain and urine catheter. Postoperative procedures were the same in all TKA patients. Patients followed an enhanced recovery pathway and received subcutaneous thromboprophylaxis (Fondaparinux) once daily for 35 d, starting on the evening on the first postoperative day.

Preoperative approved planning for the femur and tibia component were compared with the postoperative achieved alignment of each component on radiographs. HKA-axis and implant position were measured with a calibrated protocol on digital images on a PACS system[15,16]. HKA angle was evaluated on standardized 1-year postoperative frontal long-leg standing X-rays. Varus/valgus position of the FFC and FTC perpendicular to the HKA angle were measured on the same frontal radiographs. Flexion/extension of the LFC, measured from the anterior femoral cortex and posterior or anterior slope of the LTC measured from the posterior cortex of the tibia, were evaluated on 1-year postoperative lateral radiographs. Deviations of > 3° between preoperative planned HKA-axis (sum of FFC and FTC) and individual components (FFC, FTC, LFC and LTC) compared to the postoperative achieved alignment on radiographs, were considered as outliers. Mean values, SD and percentages of > 3° deviation of the preoperative planned alignment and postoperative alignment were first compared between the complete PSG group and the conventional group and all PSG groups were compared with each other. A comparison between cut- and pin guides was also made (Table 1).

## Ethical approval
This study was approved by the institutional review board (IRB Atrium-Orbis Zuyd Heerlen, the Netherlands;
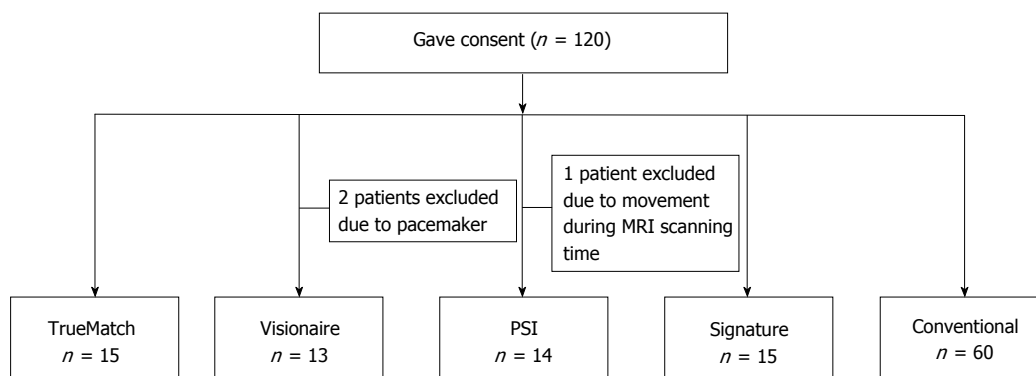
**Figure 2 Flowchart study design.** PSI: Patient-specific instrument; MRI: Magnetic resonance imaging.

**Table 3 Inter observer correlation coefficients**

|  | HKA | FFC | FTC | LFC | LTC |
|---|---|---|---|---|---|
| Inter CC | 0.811 | 0.879 | 0.883 | 0.850 | 0.943 |

HKA: Hip-knee-ankle angle; FFC: Frontal femoral component; FTC: Frontal tibial component; LFC: Flexion/extension of the femur component; LTC: Lateral tibial component; CC: Class correlation coefficient.

IRB-nr.13N09), registered online at the Dutch Trial Register (NTR4739) and was performed in compliance with the Helsinki Declaration of 1975, as revised in 2000. All patients were informed and they consented to providing data for anonymous use.

### Statistical analysis

Statistical Package for the Social Sciences V17.0 (SPSS, Inc., Chicago, IL) for Windows was used. All radiographic evaluation was performed once for each radiograph, performed by 2 independent assessors (MS and SH). Inter class correlation coefficient (ICC) was calculated to check for inter observer reliability. An ICC $\geq$ 0.7 was considered as good correlation. Statistically significant differences for radiographs were analyzed with a one-way ANOVA. The Bonferroni method for correcting for multiple comparisons was used to reduce the chances of obtaining false-positive results (type I errors). Fisher exact test was used to test differences of proportions. *P*-value was considered to be statistically significant at *P* $\leq$ 0.05 for all statistical analyses.

## RESULTS

Of the 120 patients included, 3 patients could not be scanned with MRI and were operated with use of computed tomography (CT)-based PSG (Signature, Biomet). Baseline demographics are shown in Table 2. All guides fitted well during the time of operation, there were no conversions to conventional instrumentation. All radiographic measurements of both observers were reliable and ICC's were excellent (Table 3).

With regard to the individual components, percentage of outliers of the FFC ($P < 0.000$), LFC ($P < 0.000$) and LTC ($P < 0.05$) were significantly different

**Table 4 Mean (SD) values and amount of patients and percentages of outliers of > 3° deviation of the planned alignment and postoperative alignment compared between the patient specific matched guides and the conventional group, *n* (%)**

| Outliers | PSG | Conventional | *P* value |
|---|---|---|---|
| HKA outliers | 7 (12.28) | 11 (18.33) | 0.424 |
| Mean (SD) | 179.49 (2.24) | 178.54 (2.27) | 0.015 |
| FFC outliers | 0 (0) | 11 (18.33) | 0.000 |
| Mean (SD) | 89.44 (1.73) | 88.03 (1.73) | 0.000 |
| FTC outliers | 1 (1.75) | 0 (0) | 1.000 |
| Mean (SD) | 89.87 (1.32) | 90.37 (1.38) | 0.058 |
| LFC outliers | 9 (15.78) | 35 (58.33) | 0.000 |
| Mean (SD) | 86.09 (2.86) | 86.04 (3.14) | 0.314 |
| LTC outliers | 9 (15.78) | 25 (41.67) | 0.033 |
| Mean (SD) | 92.86 (2.64) | 87.43 (2.63) | 0.000 |

PSG: Patient specific matched guides; HKA: Hip-knee-ankle angle; FFC: Frontal femoral component; FTC: Frontal tibial component; LFC: Flexion/extension of the femur component; LTC: Lateral tibial component.

in favour of the PSG group (Table 4). Regarding the individual different PSG systems, the mean (SD) HKA-axis ($P < 0.000$), the FFC ($P < 0.000$) and LTC ($P < 0.000$) alignment were significantly different (Table 5). The PSG group subdivided into cut- and pin PSG showed significant difference regarding the mean FFC ($P < 0.022$) and the LTC ($P < 0.009$) alignment (Table 6).

## DISCUSSION

This industry driven technology proved to be safe, reproducible and easy to use. This leads to a commercial success compared to other computer-assisted technologies[11]. Although, published results on PSG are contrasted, even on level I studies. Seven level I studies compared conventional instrumentation with PSG and compared different PSG manufacturers. None of them had measured a significant difference in outliers of HKA axis (Table 7). However, Pfitzner *et al*[24], recently published results comparing conventional instrumentation with CT and MRI based PSG from 2 different manufacturers, and between both PSG groups. They found a significant difference regarding the outliers in HKA-axis between MRI based PSG (Visionaire; 7%) and conventional instruments (43%), but no significant difference between

**Table 5  Mean (SD) values and amount of patients and percentages of outliers of > 3° deviation of the planned alignment and postoperative alignment compared between the patient specific matched guides groups, *n* (%)**

|  | Trumatch | Visionaire | PSI | Signature | *P* value |
|---|---|---|---|---|---|
| HKA outliers | 3 (20.00) | 1 (7.69) | 2 (14.28) | 1 (6.66) | 0.819 |
| Mean (SD) | 178.5 (2.3) | 181.3 (1.6) | 180.6 (1.6) | 177.9 (1.8) | 0.000 |
| FFC outliers | 0 | 0 | 0 | 0 | 1.000 |
| Mean (SD) | 89.9 (1.6) | 90.1 (1.5) | 89.9 (1.2) | 87.9 (1.8) | 0.000 |
| FTC outliers | 0 | 0 | 1 (7.14) | 0 | 1.000 |
| Mean (SD) | 89.3 (1.4) | 90.0 (1.2) | 89.9 (1.6) | 90.6 (1.3) | 0.081 |
| LFC outliers | 2 (13.33) | 2 (15.38) | 1 (7.14) | 4 (26.66) | 0.663 |
| Mean (SD) | 85.7 (1.6) | 85.4 (2.1) | 87.4 (1.9) | 85.8 (4.5) | 0.307 |
| LTC outliers | 2 (13.33) | 4 (30.76) | 2 (14.28) | 1 (6.66) | 0.594 |
| Mean (SD) | 92.7 (2.4) | 91.2 (3.0) | 94.8 (1.2) | 92.8 (2.7) | 0.000 |

PSI: Patient-specific instrument; HKA: Hip-knee-ankle angle; FFC: Frontal femoral component; FTC: Frontal tibial component; LFC: Flexion/extension of the femur component; LTC: Lateral tibial component.

**Table 6  Mean (SD) values and amount of patients and percentages of outliers of > 3° deviation of the planned alignment and postoperative alignment compared between the cut (*n* = 28, Trumatch and Visionaire) and pin (*n* = 29, patient-specific instrument and signature) patient specific matched guides group, *n* (%)**

|  | Cut PSG | Pin PSG | *P* value |
|---|---|---|---|
| HKA outliers | 4 (14.3) | 3 (10.3) | 1.000 |
| Mean (SD) | 179.9 (2.4) | 179.3 (2.2) | 0.342 |
| FFC outliers | 0 | 0 | 1.000 |
| Mean (SD) | 90.0 (1.5) | 89.6 (1.8) | 0.022 |
| FTC outliers | 0 | 1 (3.4) | 1.000 |
| Mean (SD) | 89.6 (1.3) | 90.2 (1.5) | 0.115 |
| LFC outliers | 4 (14.3) | 5 (17.2) | 1.000 |
| Mean (SD) | 85.6 (1.8) | 86.6 (3.5) | 0.184 |
| LTC outliers | 6 (21.4) | 3 (10.3) | 0.477 |
| Mean (SD) | 92.0 (2.7) | 93.8 (2.3) | 0.009 |

PSG: Patient specific matched guides; HKA: Hip-knee-ankle angle; FFC: Frontal femoral component; FTC: Frontal tibial component; LFC: Flexion/extension of the femur component; LTC: Lateral tibial component.

CT based PSG and conventional instruments, neither between both PSG systems[24]. This was contrary to what Victor *et al*[25] found. They compared 4 different PSG systems with the conventional technique, operated by 4 surgeons, with more significant outliers for the FTC and LTC in favour of the intra- and extramedular technique (Table 7). Even between the 4 different PSG systems, percentages of outliers of > 3° deviation of the planned HKA and LFC angle were significantly different, ranging from 6% to 45% and 20% to 82%, respectively[25] (Table 7). Published level I percentages of outliers in the frontal and lateral plane for individual components for both femur and tibia vary and are inconclusive. Outliers of the FFC for the PSG are comparable or less than the conventional intramedular technique. Only 2 authors published significant differences in favour of the MRI based PSG[17,24]. This was in contrast to the FTC (Table 7). Most of the outcomes are comparable, however, 2 articles published significant better outcome with extramedular rods[25,26]. Only Ng *et al*[22] found significant better outcome with MRI based PSG for the tibia. Level I results are very remarkable in regard to the LTC. These were significantly better with PSG than with conventional instrumentation (Table 7). Most notable are the significant differences that have been found with CT based PSG, which scored poorer outcome regarding to LTC outliers, ranging from 21% to 65%[19,25-27] (Table 7). A possible explanation for these outcomes can be the limitations in visualization and outlining of intra-articular cartilage in CT based 3D models[28-31]. Another explanation, based on our experience, is that CT based guides were more difficult to place on the bony surface compared to MRI based guides. Nevertheless, we did not reveal a significant difference between the MRI and CT PSG surgeries for HKA-axis and individual components for the different planes.

There may be some concerns regarding our radiological measurements. A wide variety of different analyses in the literature are used to objectively determine the postoperative position for both the femur and tibia implants (Table 7). Despite a good ICC for the evaluation of the frontal and lateral position of both femur and tibia implants, rotational alignment was not examined. Most of the literature use long-standing radiographs, except for 1 paper which used scout CT scan[17] and two used full-leg CT scans[22,27]. Postoperative evaluation on 3D-CT have shown to be a valuable tool to measure position and orientation of both the femur and tibia components and it is more accurate with significantly better femoral rotation alignment after use of PSG[18,22,32]. Unfortunately, a postoperative 3D-CT is not routinely performed in our clinic. On the other hand, plane radiographs are generally applicable for everyone.

This single surgeon experience with different PSG manufacturers could raise questions about the general applicability. We had the opportunity to use different types of PSG and implants. Based on the experience with TKA, the use of PSG and a possible learning curve, implementation of a new implant system may be a

**Table 7 Published level Ⅰ studies with significant percentage of outliers of > 3° deviation between the patient specific matched guides and conventional intramedular and/or extramedular alignment method for hip-knee-ankle angle axis, frontal femoral component, frontal tibial component, flexion/extension of the femur component, lateral tibial component and axial rotation of the femur and/or tibia component controlled with postoperative X-ray (long-leg standing and/or lateral X-rays) and/or computed tomography**

| Outliers (%) > 3° deviation | PSG system | Modality | Conventional femur/tibia | Control | Sample size (PSG/conventional) | Significant outliers (%) (PSG/conventional) |
|---|---|---|---|---|---|---|
| Boonen *et al*[16] | Signature | MRI | Intra | X-ray | 90/90 | LFC (49/65)[1] |
| Chareancholvanich *et al*[17] | PSI | MRI | Intra/Extra | X-ray and CT | 40/40 | FFC (0/18)[1] |
| Chotanaphuti *et al*[18] | TruMatch | CT | Intra/Extra | X-ray and CT | 40/40 | NA |
| Hamilton *et al*[19] | TruMatch | Scout CT | Intra/Extra | X-ray | 26/26 | LTC (65/50)[2] |
| Ng *et al*[22] [Outliers (%) > 2° deviation] | PSI | MRI | Intra | CT | 51/27 | FTC (27/67)[2], Femoral rotation (16/67)[2], Tibial rotation (22/95)[2] |
| Pfitzner *et al*[24] | TruMatch | CT | Intra/Extra | X-ray and CT | (30/30)/30 | HKA (30/7/43)[2] |
| | Visionaire | MRI + X-ray | | | | FTC (13/3/23)[1] |
| | | | | | | Femoral rotation (1/13 /50)[1] |
| Victor *et al*[25] | Signature | MRI, CT | Intra/Extra | X-ray and CT | (16/16/16/16)/64 | FTC (15/3)[1] |
| | TruMatch | MRI + X-ray | | | | LTC (21/3)[2] |
| | Visionaire | MRI | | | | HKA (6/25/45/19)[1,3] |
| | PSI | | | | | LFC (62/20/20/56)[2,3] |
| Kotela *et al*[26] | Signature | CT | Intra/Extra | X-ray | 49/46 | FTC (39/20)[1] |
| Woolson *et al*[27] | TruMatch | CT | Intra/Extra | CT | 22/26 | LTC (32/8)[1] |
| Current study | Signature | MRI | Intra | X-ray | (15/13/14/15)/60 | FFC (022)[2] |
| | TruMatch | CT | | | | LFC (16/67)[2] |
| | Visionaire | MRI + X-ray | | | | LTC (16/42)[1] |
| | PSI | MRI | | | | |

[1]Statistically significant different, $P \leq 0.05$; [2]Statistically significant different, $P \leq 0.005$; [3]Outliers > 3° deviation between the different PSG groups. NA: Not applicable for outliers; PSG: Patient specific matched guides; PSI: Patient-specific instrument; CT: Computed tomography; MRI: Magnetic resonance imaging; Intra: Intramedular; Extra: Extramedular; HKA: Hip-knee-ankle angle; FFC: Frontal femoral component; FTC: Frontal tibial component; LFC: Flexion/extension of the femur component; LTC: Lateral tibial component.

potential bias in the outcome[25]. However, research is mostly performed by high-volume surgeons who probably easier adapt to a new surgical technique than low-volume surgeons or residents. PSG could be an added value in less experienced surgeons due to their simplicity[19]. On the other hand, we evaluated cut and pin PSG from different manufacturers with less outliers compared to the conventional group.

Our primary goal was to investigate the accuracy of alignment between conventional and PSG and between different PSG systems compared with published level Ⅰ evidence. A comparison on perioperative and clinical outcome were not made, although there is a trend towards significant shorter operating time[16-18] and blood loss[16] with surgeries performed with PSG. However, published results on component sizing are inconclusive to come up with a statement[18,19,27].

Finally, even though this study was a consecutive series compared with a historical cohort and not a randomized trial, a potential criticism was the sample size and power of this study.

The present study illustrates that this simplified surgical technique for TKA is safe and effective with acceptable radiological outcome. The PSG group shows significantly less outliers compared to the conventional technique. Whether these differences are clinically relevant is questionable and should be investigated on the long term. Based on these single surgeon results, we conclude that PSG are ready for prime time.

# ACKNOWLEDGMENTS

# COMMENTS

## Background
Patients with knee osteoarthritis often results in debilitating function of the knee joint warranting a total knee arthroplasty (TKA). This study aims to present the radiological results of TKA with use of patient specific matched guides (PSG) from different manufacturer in patients suffering from severe osteoarthritis of the knee joint.

## Research frontiers
Patients suffering from osteoarthritis of the knee joint can be operated with use of PSG for TKA from different manufacturer. TKA with PSG has concerns regarding accurate implant alignment and the long term survival of the TKA compared to the conventional instrumentation.

## Innovations and breakthroughs
In this study, PSG for TKA from different manufacturer restored good biomechanical axis and individual implant alignment in patients suffering from moderate to severe osteoarthritis of the knee joint compared to conventional alignment.

## Applications
To summarize, PSG from different manufacturer can be an added value in daily

TKA practice in patients suffering from moderate to severe osteoarthritis of the knee joint compared to the conventional instrumentation for TKA.

### Peer-review

The authors compared the accuracy of TKA using patient-specific instruments (PSIs) with that of TKA using the conventional technique. In addition, they compared the accuracy of 4 different manufactured PSI TKAs. In conclusion, TKA using PSIs was more accurate than TKA using the conventional method, and no difference in accuracy was found between the 4 different manufactured PSI TKAs. Regarding the PSI TKA that was recently developed, more research studies, including precision, cost, operation time, blood loss, radiation exposure, and long-term survival, should be conducted in order to examine if it confers more benefits to patients than the conventional TKA. The manuscript could add new information on PSI TKA regarding its accuracy.

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*CASE REPORT*

# Surgical treatment of sacral fractures following lumbosacral arthrodesis: Case report and literature review

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## Abstract

Sacral fractures following posterior lumbosacral fusion are an uncommon complication. Only a few case series and case reports have been published so far. This article presents a case of totally displaced sacral fracture following posterior L4-S1 fusion in a 65-year-old patient with a 15-year history of corticosteroid use who underwent open reduction and internal fixation using iliac screws. The patient was followed for 2 years. A thorough review of the literature was conducted using the Medline database between 1994 and 2014. Immediately after the revision surgery, the patient's pain in the buttock and left leg resolved significantly. The patient was followed for 2 years. The weakness in the left lower extremity improved gradually from 3/5 to 5/5. In conclusion, the incidence of postoperative sacral fractures could have been underestimated, because most of these fractures are not visible on a plain radiograph. Computed tomography has been proved to be able to detect most such fractures and should probably be performed routinely when patients complain of renewed buttock pain within 3 mo after lumbosacral fusion. The majority of the patients responded well to conservative treatments, and extending the fusion construct to the iliac wings using iliac screws may be needed when there is concurrent fracture displacement, sagittal imbalance, neurologic symptoms, or painful nonunion.

Key words: Sacral fracture; Insufficiency fracture; Surgical treatment; Complication; Lumbosacral fusion; Revision surgery

Core tip: Sacral fractures following posterior lumbosacral fusion are rare. This article presents a case of totally displaced sacral fracture following posterior L4-S1

fusion. Computed tomography has been proved to be able to detect most such fractures and should probably be performed routinely when patients complain of renewed buttock pain within 3 mo after lumbosacral fusion. The majority of the patients responded well to conservative treatments, and extending the fusion construct to the iliac wings using iliac screws may be needed when there is concurrent fracture displacement, sagittal imbalance, neurologic symptoms, or painful nonunion.

## INTRODUCTION

Sacral fractures following posterior lumbosacral fusion are an uncommon complication, previously described in only a few case series and case reports. Although these sacral fractures are rarely reported, their incidence could be much higher than previously thought. One of the reasons for underdiagnosis of these fractures is that they are usually unrecognized on plain radiographs, and establishment of a diagnosis is often dependent on computed tomography (CT) or magnetic resonance imaging (MRI). Another reason is that many of these fractures can heal without intervention in a few months, which makes them difficult to be noticed by doctors.

Risk factors for developing sacral fractures following lumbosacral fusions have been identified by several authors. They include old age, female sex, obesity, smoking, postmenopausal osteoporosis, chronic corticosteroid use, prior radiation therapy, graft harvesting, multisegmental lumbosacral fusion, and abnormal spinopelvic alignment. According to the reported experience, most of these fractures occurred within 3 mo after surgery, and the majority of these patients responded well to conservative therapy. Surgical intervention may be needed when there are persistent neurological deficits, significant displacements, severe pain, or fracture nonunion.

This article presents a case of totally displaced sacral fracture following posterior L4-S1 fusion in a 65-year-old patient with a 15-year history of corticosteroid use who underwent open reduction and internal fixation using iliac screws.

## CASE REPORT

The current case was a 65-year-old overweight female (body mass index = 25.63 kg/m$^2$) who presented with a chief complaint of 5 years of progressively increased left lower extremity pain and difficulty in walking, which were refractory to conservative management.

The patient was ambulant with 4/5 lower extremity weakness. Both Hoffmann's sign and Babinski's sign were negative. The patient suffered from asthma and had a 15-year history of corticosteroid use. A bone density test of the hip (T-score to -3.7) showed poor bone quality.

Roentgenograms revealed grade 2 anterolisthesis of L5 on S1. The MRI of the spine found central lumbar spinal stenosis at the L5/S1 level and L4-S1 foraminal narrowing (Figure 1). L4-S1 posterior fusion with poly-axial pedicle screws and double rods (XIA Ⅱ, Stryker) was performed with posterolateral bone grafting using an autologous lamina/spinous process. A cage was also inserted into the L5/S1 disc.

The patient tolerated the procedure well and was walking well on the first postoperative day. On the 5$^{th}$ day after surgery, however, the patient reported a sudden exacerbation of bilateral buttock pain, left-leg radicular pain and sphincter disturbances without precedent trauma. Physical examination revealed a 3/5 weakness of the left lower extremity. A CT scan revealed a horizontal fracture at the S1/S2 level with S2 being totally displaced (Figure 2).

We tried to reduce the fracture with traction but failed. Considering the significant neurological deficits and severe S2 displacement, we performed posterior neural decompression and hardware revision with deformity reduction for the patient at 2 wk after the index operation. The fusion construct was extended to the iliac wings using iliac screws (Figures 3 and 4).

Immediately after the revision surgery, the patient's pain in the buttock and left leg resolved significantly. The patient was followed for 2 years. The weakness of the left lower extremity improved gradually from 3/5 to 5/5.

The sagittal radiographic parameters are listed in Table 1. The preoperative values indicate that she had a high PI (64.1°) and SS (37.1°).

## DISCUSSION

Sacral fractures following posterior lumbosacral fusion are rarely seen, and there has been a paucity of data on the association in the published literature. Before 2013, only 34 cases had been reported. No cohort with more than 5 cases had been published until recently. Many spine surgeons have never encountered or noticed such a type of fracture.
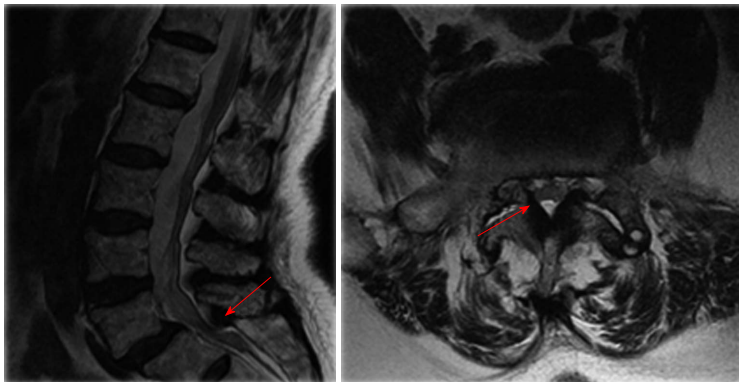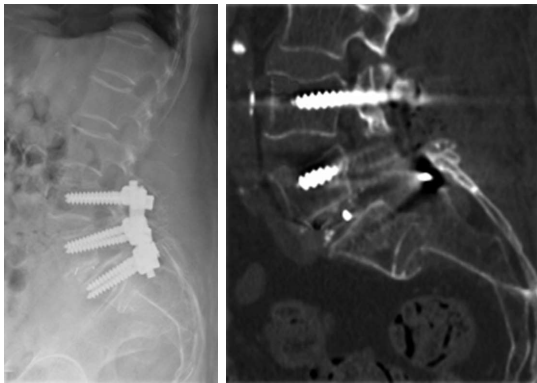
### *Incidence and diagnosis*

The incidence of postoperative sacral fractures could have been rather underestimated. Because most of these fractures are not visible on a plain radiograph, diagnosis is mostly established based on CT, MRI, or nuclear scintigraphy. There are 2 studies with a larger cohort that have been published recently. Meredith *et al*[1] included all patients undergoing posterior lumbosacral arthrodesis at their institution between 2002 and 2011. Twenty-four out of 392 (6.1%) patients

|  | Pelvic incidence | Sacral slope | Pelvic tilt | Lumbar lordosis |
| --- | --- | --- | --- | --- |
| Before index surgery | 64.1° | 37.1° | 27° | 53.9° |
| After index surgery | N/A | 36.7° | N/A | 51.3° |
| After revision surgery | N/A | 30.3° | N/A | 47.4° |
| 2 yr | N/A | 31.4° | N/A | 45.9° |

Pelvic incidence is the angle formed by the perpendicular line to the tangent line to the center of the sacral plateau and the line connecting this center of the bicoxofemoral axis. Pelvic tilt is the angle between the vertical plane and the line connecting the center of the sacral plateau and the center of the bicoxofemoral axis. The lumbar lordosis is measured between the cranial endplate of L1 and caudal endplate of L5. N/A: Not applicable because of the displacement of S1.



**Figure 1  Magnetic resonance imaging of the spine found central lumbar spinal stenosis at L5/S1 level and L4-S1 foraminal narrowing.**



**Figure 2  A computed tomography scan revealed a horizontal fracture at the S1/S2 level with S2 being totally displaced.**

presented with sacral fractures after surgery, which were confirmed by CT, MRI, or nuclear scintigraphy. However, in only one out of the 24 cases, could the sacral fracture be noticed on the postoperative radiographs. Wilde *et al*[2] reported a cohort of 23 patients who had sacral fractures after lumbosacral fusion. Similarly, the sacral fracture was noticed in only one out of 23 patients on the postoperative radiographs. As such, sacral fractures after lumbosacral fusion could have been greatly underdiagnosed. CT has proven to be able to detect most of such fractures and should probably be performed routinely when patients complain of renewed buttock pain within 3 mo after lumbosacral fusion.

### Risk factors and prevention

Old age, female sex, osteoporosis, obesity, and a long moment arm of multisegmental lumbosacral fusion are the most frequently cited risk factors for sacral fractures after posterior lumbosacral fusion. The current case had a 15-year history of corticosteroid use for her asthma. The bone density test of the hip (T-score to -3.7) showed a poor bone quality. Furthermore, abnormal spinopelvic alignment could also be a risk factor for fracture development.

To prevent the onset of postoperative sacral fractures, fixation of the iliac wings can be considered in high-risk patients.

### Surgical treatment

The reported experience showed us that these postoperative sacral fractures responded well to conservative treatments, which included activity modification, external immobilization, and medical treatment of osteoporosis[3-5]. However, sacral insufficiency fractures with significant displacement, sagittal imbalance, neurologic symptoms, or painful nonunion may necessitate surgical stabilization. The most commonly performed procedure is to extend the fusion construct to the iliac wings using iliac screws. Fracture union and pain relief were achieved in all the surgically treated cases reported in the literature[1-8].

In conclusion, the incidence of postoperative sacral fractures could have been rather underestimated, because most of these fractures are not visible on a
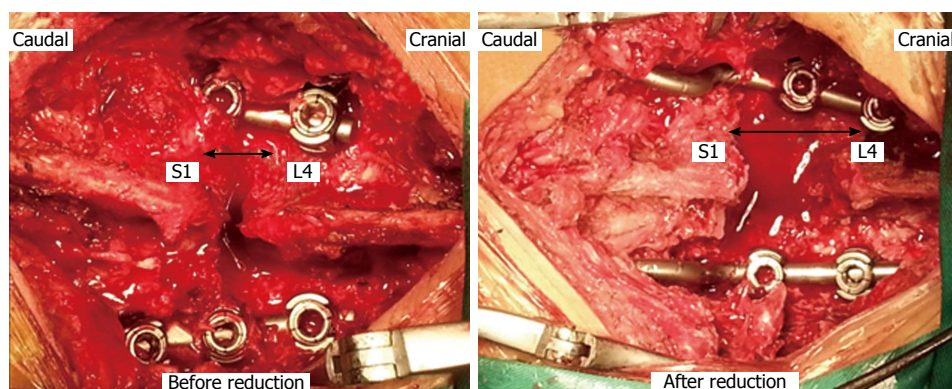
**Figure 3 The interspinous process of the sacrum was totally displaced.** It moved backward and cranially onto the back of the instruments. To reduce the fracture, we used a spreader to distract the spinal processes of S1 and L4, which was effective. After the distraction, the distance between the spinal processes of the S1 and L4 increased significantly.
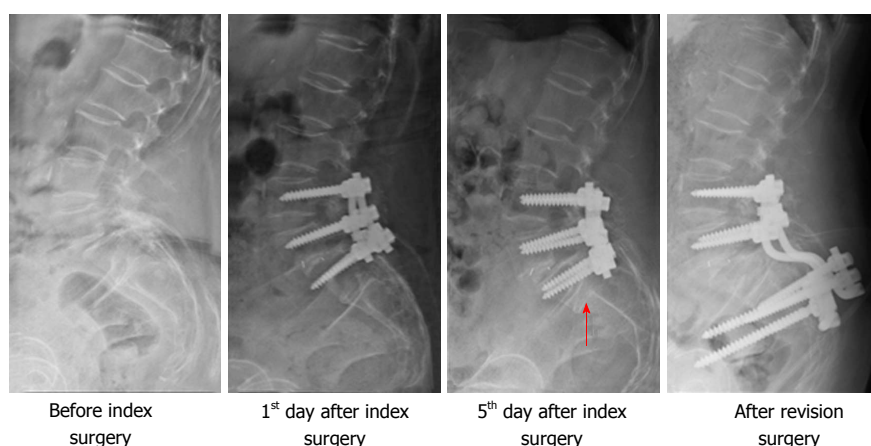


Before index surgery     1st day after index surgery     5th day after index surgery     After revision surgery

**Figure 4 Sacral fracture found on the 5th day after surgery.** Two weeks after the index operation, the fusion construct was extended to the iliac wings using iliac screws.

plain radiograph. CT has been proved to be able to detect most of such fractures and should probably be performed routinely when patients complain of renewed buttock pain within 3 mo after lumbosacral fusion. The majority of the patients responded well to conservative treatments, and extending the fusion construct to the iliac wings using iliac screws may be needed when there is concurrent fracture displacement, sagittal imbalance, neurologic symptoms, or painful nonunion.

## COMMENTS

### Case characteristics
A 65-year-old patient with a 15-year history of corticosteroid use reported a sudden exacerbation of bilateral buttock pain, left-leg radicular pain and sphincter disturbances without precedent trauma on the 5th day after posterior L4-S1 fusion.

### Clinical diagnosis
Physical examination revealed a 3/5 weakness of left lower extremity.

### Differential diagnosis
Osteoporotic vertebral compressive fracture, epidural hematoma, malposition of pedicle screws, and migration of pedicle screws.

### Imaging diagnosis
A computed tomography (CT) scan revealed a horizontal fracture at the S1/S2 level with S2 being totally displaced.

### Treatment
The authors performed posterior neural decompression and hardware revision with deformity reduction for the patient at 2 wk after the index operation. The fusion construct was extended to the iliac wings using iliac screws.

### Related reports
Sacral fractures following posterior lumbosacral fusion are rarely seen, there has been a paucity of data on the association of this condition in the published literature. Before 2013, there were only 34 cases that had been reported. No cohort with more than 5 cases had been published until recently.

### Term explanation
Sacral fractures following posterior lumbosacral fusion are an uncommon complication. Risk factors include old age, female sex, obesity, smoking, postmenopausal osteoporosis, chronic corticosteroid use, prior radiation therapy, graft harvesting, multisegmental lumbosacral fusion, and abnormal spinopelvic alignment.

### Experiences and lessons
The incidence of postoperative sacral fractures could have been rather underestimated, because most of these fractures are not visible on plain

radiograph. CT has been proved to be able to detect most of such fractures and should probably be performed routinely when patients complain of renewed buttock pain within 3 mo after lumbosacral fusion. The majority of the patients responded well to conservative treatments, and extending the fusion construct to the iliac wings using iliac screws may be needed when there is concurrent fracture displacement, sagittal imbalance, neurologic symptoms, or painful nonunion.

### *Peer-review*

It's a well-organised study.

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