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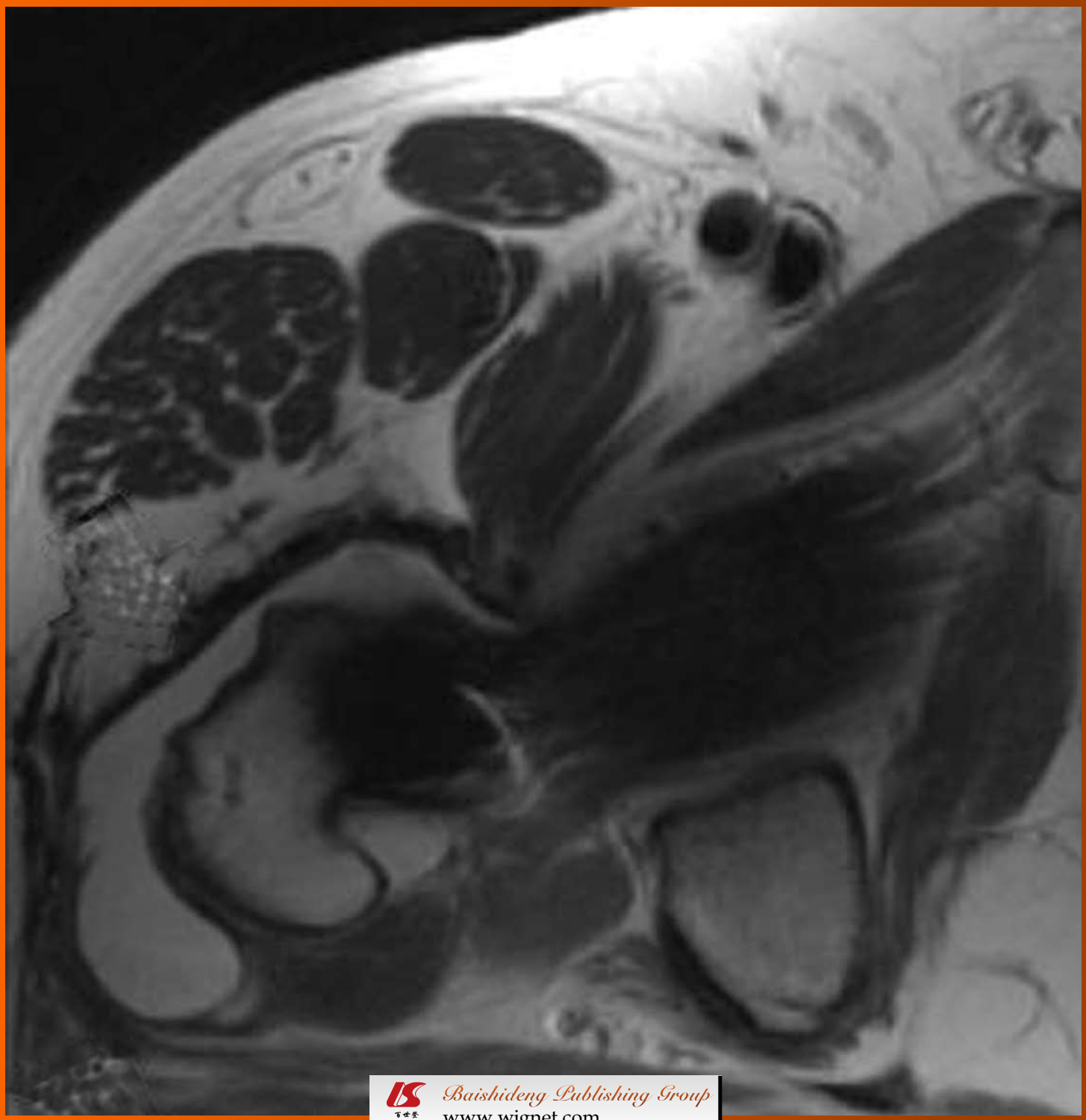

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Editorial Board of *World Journal of Orthopedics*
Room 903, Building D, Ocean International Center,
No. 62 Dongsihuan Zhonglu, Chaoyang District,
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Telephone: +86-10-85381892
Fax: +86-10-85381893
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EDITOR-IN-CHIEF
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World Journal of Orthopedics
Room 903, Building D, Ocean International Center,
No. 62 Dongsihuan Zhonglu, Chaoyang District,
Beijing 100025, China
Telephone: +86-10-85381892
Fax: +86-10-85381893
E-mail: wjo@wjgnet.com
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EDITORIAL

Tendon injuries of the hand

Volker Schöffl, Andreas Heid, Thomas Küpper

Volker Schöffl, Department of Sportorthopedics and Sportsmedicine, Klinikum Bamberg, 96049 Bamberg, Germany
Volker Schöffl, Department of Traumatology, Friedrich Alexander University Erlangen-Nuremberg, Germany
Andreas Heid, Department of Orthopedic and Trauma Surgery, Klinikum Bamberg, 96052 Bamberg, Germany
Thomas Küpper, Institute of Occupational and Social Medicine, Aachen Technical University, Aachen, Germany
Author contributions: Schöffl V made substantial contributions to the conception and design, acquisition, analysis and interpretation of data, drafting the article, revising it critically for important intellectual content and gave final approval of the version to be published; Heid A acquired, analyzed and interpreted the data, drafted the article in English and gave final approval of the version to be published; Küpper T made substantial contributions to the conception and design, acquisition of data, drafting the article, revising it critically for important intellectual content and gave final approval of the version to be published.
Correspondence to: Dr. Volker Schöffl, Associate Professor, Department of Sportorthopedics and Sportsmedicine, Klinikum Bamberg, Bugerstr.80, 96052 Bamberg, Germany. volker.schoeffl@me.com
Telephone: +49-951-50312241 Fax: +49-951-50312249
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Abstract

Tendon injuries are the second most common injuries of the hand and therefore an important topic in trauma and orthopedic patients. Most injuries are open injuries to the flexor or extensor tendons, but less frequent injuries, e.g., damage to the functional system tendon sheath and pulley or dull avulsions, also need to be considered. After clinical examination, ultrasound and magnetic resonance imaging have proved to be important diagnostic tools. Tendon injuries mostly require surgical repair, dull avulsions of the distal phalanges extensor tendon can receive conservative therapy. Injuries of the flexor tendon sheath or single pulley injuries are treated conservatively and multiple pulley injuries receive surgical repair. In the postoperative course of flexor tendon injuries, the principle of early passive movement is important to trigger an "intrinsic" tendon healing to guarantee a good outcome. Many substances were evaluated to see if they improved tendon healing; however, little evidence was found. Nevertheless, hyaluronic acid may improve intrinsic tendon healing.

Key words: Flexor tendon; Extensor tendon; Tendon sheath; Pulley injury; Tendon lesion

Peer reviewer: Stefan Grote, MD, Department of Orthopaedic and Trauma Surgery, University of Munich, Nußbaumstr. 20, 80336 Munich, Germany

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EPIDEMIOLOGY

The hand, as the human executing organ, is in the center of daily life activities in professions and sports. In this outstanding position, the hand is always exposed to injuries and overuse. With the change of our society from a industrial society to a service-based society, surprisingly, an assumed decrease in hand injuries has not been detected, probably due to an increase in private activities, such as sports and do-it-yourself work[1]. Based on 50 272 injured persons, Angermann and Lohmann[2] showed that 28.6% of treated patients in emergency care were caused by hand injuries, a risk of 3.7 injuries in 100 000 individuals of the Danish population. On average, hand injuries count for 14% to 30% of all treated patients in emergency care. Tendon lesions are in 2nd position (29%), whereas fractures are 1st (42%) and skin lesions 3rd of all patients treated for hand injuries. Even although only 2% of the patients are hospitalized, hand injuries, especially tendon lesions, play a key role in or-

thopedic and traumatic treatment. Degenerative lesions must be counted as well.

ANATOMY AND BIOMECHANICS

To begin with, flexor and extensor tendons must be looked at separately anatomically, even although the tendons of the mm. lumbricalis take an exceptional position.

EXTENSOR TENDONS

The long fingers hold 4 common extension tendons, namely extensores digitorum communes, and additionally the extensor indices for the 2nd and the extensor digiti minimi for the 5th finger. The tendon of the extensor digiti minimi runs through the 5th tendon compartment, all the others through the 4th compartment. On the level with the dorsum of the hand and the metacarpophalangeal joints, many cross-connections are found, the connexi intertendinei. On level with the proximal interphalangeal joint (PIP), the extensor tendon separates into two lateral reins and one central rein (tractus intermedius). Two different extensor tendons reach to the thumb, the extensor pollicis longus (3rd extensor compartment) and the extensor pollicis brevis (1st extensor compartment). All named tendons form the so called extrinsic system, tendons of muscles which originate proximal of the hand itself. The extrinsic system is supported by the intrinsic system, muscles originated within in the hand, to the mm. lumbricales, the mm. interossei and the thenar and hypothenar muscles.

FUNCTIONAL SYSTEM "FLEXOR TENDONS", TENDON SHEATH AND PULLEY

The system of flexor tendons is not looked at individually but as a functional unit of tendons, tendon sheath and pulleys. A differentiation between the thumb and the index finger is also necessary. Both flexor tendons of the long fingers, the flexor digitorum profundus (FDP) and m. flexor digitorum superficial (FDS) run commonly through the carpal tunnel and pulleys and subsequently intersect at the chiasm. The flexor tendon of the thumb (m. flexor pollicis longus) runs through the carpal tunnel on the radial side of the forearm and, strengthened by 2 pulleys, the muscle passes through an osteofibrous channel to the basis of the distal phalanx (Figure 1).

The annular ligaments and crucial ligaments are seen as a re-enforcement system of the flexor tendons along the osteofibrous channels of the fingers and hereby are fixed to the phalanges[3]. Four to 5 annular (A1-A5) and 3 weaker crucial ligaments (C1-C3) are distinguished (Figure 1)[3]. The pattern and arrangement of these ligaments vary. All pulleys have different functions in stabilising the flexor tendons at the palmar sides of the phalanges[4,5]. The main function of the flexor tendon digital pulley system is to maintain the flexor tendons close to the bone, thus converting linear translation and force developed in the flexor muscle-tendon unit into rotation and torque at the finger joints[6]. The A2 crucial ligament plays the most important role in guidance of the flexor tendons, whereas opinions concerning the A3 and A4 vary considerably (Figures 2 and 3). A smaller role in strength transmission and tendon deflection is performed by the A1 and A5[6]. The pulley system suppresses the tendon excursion and the strength of the flexor tendons is redirected efficiently in flexion and hyperextension to reach the full range of motion[6]. The mm. lumbricalis and mm. interossei are exceptions as they originate from the flexor tendons themselves and end in the tendinous hood of the extensor tendons. Their function is flexion in the metacarpophalangeal joint (MCP), extension in the proximal (PIP) and distal interphalangeal joint (DIP).

Blood supply of the flexor tendons is guaranteed through the "vincula tendinae" in the region of the osseous insertion of the tendon as well as in the osteofibrous channel. Venous drainage is performed through the same system. Verdan[7] subsequently divided the flexor and extensor tendons into different regions of interest regarding injuries, prognosis and nutrition[1].

TENDON HEALING

Histologically, tendons consist of extremely long collagenous fibers that are arranged into bundles. Similar to a rope ladder, elastic fibers and vessels are entangled in between those bundles[10]. To provide the necessary slippage, a peritendeum or a tendon sheath covers tendons. Pulleys strengthen these structures along the phalanges. Friction plays an important role in the origin of injuries and chronic inflammatory diseases of the tendons, their sheath and pulleys[4,5,11,12]. Following an injury, the healing process emerges from the peritendeum and the peritendinous tissue; therefore a distinction of an extrinsic to an intrinsic healing process has to be made[9,10,12,13]. Characteristic for an extrinsic healing process is a distinctive inflammatory response followed by proliferation and remodeling. Fibroblasts of the paratenon play an important role in migration which leads to adhesions. Immobilization supports the adhesion process[12-16]. The intrinsic healing process, supported by movement of the tendon, is characterized by immigration of "fibroblast-like tenocytes", which produce the collagenous tissue and carry out the remodeling process[10,12-16]. If the inflammatory response is minimal, the clinical outcome is better. This is the ratio of the widely recommended early passive movement therapy, which leads to better nutrition and strength of the tendon[10,12-16]. The following factors predict the tendon healing: age, overall health condition, scar formation disposition, motivation, injury risk based on Verdan's zones[7], injury type, synovial containment as well as the surgical technique[12]. Three phases of tendon healing are defined. Firstly, a migration of peripheral

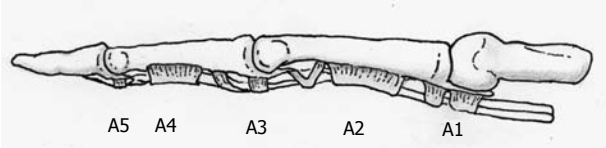

Figure 1 The pulley system of the long fingers (modified according to Schmidt and Lanz[8]).

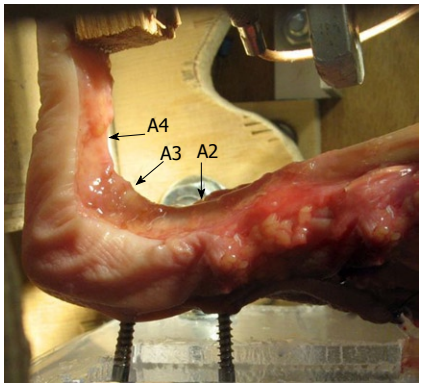

Figure 2 Complete pulley system during a stress test in the biomechanical laboratory. (Figure with permission of Schöffl V, MD, PhD, Institute of Anatomy, University of Erlangen-Nuremberg, Germany[9]).

cells and invasion of blood vessels occur and secondly, the tendon and surrounding tissues heals. Remodeling happens in the third phase of healing due to movement and function of the tendon[17].

The tendon gains its daily life loading capacity after 12 wk of healing and sporting activities are allowed 4 mo after injury at the earliest. The remodeling process can last up to 12 mo.

INJURY PATTERN

Injury patterns are differentiated into open or closed, sharp or blunt, traumatic or degenerative lesions, as well as injury to the dorsal or palmar part. Further subdivisions are osseous tendon lesions, complex lesions with concomitant trauma or injury of the tendon sheath and pulley system.

Closed tendon injuries are quite common[18] (Figure 4). Among others are the "mallet finger", the "boutonnière deformity", avulsions and injuries to the connexus intertendineus. The "mallet finger", also named baseball finger, is a blunt extensor tendon injury to the distal interphalangeal joint[13,18,19]. An osseous disruption of the extensor tendon is called mallet fracture[13]. This injury used to be named "Bush-fracture"; a term no longer used[1,9]. The rupture of the tractus intermedius, the "Boutonnière deformity" (French for button hole), is an interruption of the central reins of the extensor tendons at the level of the proximal interphalangeal joint and a subsequent slip of both lateral reins resulting in hyperflexion in the proximal and hyperextension in the distal interphalangeal joint. The full clinical signs of this injury

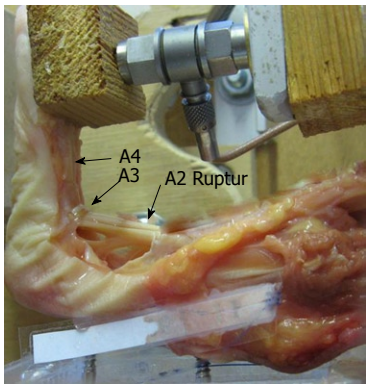

Figure 3 A2-pulley rupture during a stress test in the biomechanical laboratory. Note the increased distance between the flexor tendons and the bone. The A3-pulley is unharmed. (Figure with permission of Schöffl V, MD, PhD, Institute of Anatomy, University of Erlangen-Nuremberg, Germany[9]).

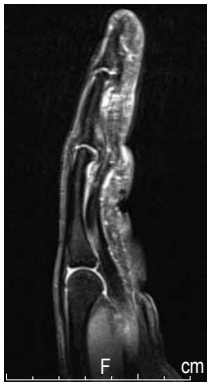

Figure 4 Closed flexor tendon rupture of the flexor digitorum profundus at the level of the middle phalanx (rock climber)[9].

often appear only secondary to a palmar dislocation of the PIP joint. Leddy and Cole[18,20] divide three different types of these injuries. Blunt disruptions, usually due to a direct trauma, of the radial sagittal ligament of the extensor tendon hood at level of the MCP joints lead to subluxation or luxation of the ligament, the majority to the ulnar side[21]. The "boxers knuckle", following repetitive blunt trauma to the MCP joints, should be taken into differential diagnostic considerations; however, this injury represents an injury of the extensor tendon hood as well but without luxation of the tendon. Avulsions, first mentioned by von Zander[22] in 1989, mostly affect the insertion of the FDP tendon at the distal phalanx. Usually this injury is seen on the 4th finger, as the FDP tendon is embedded in between the double-sided lumbrical tendons, as shown by Manske and Lesker[23] in cadaver dissection. We can verify this injury, especially in rock climbers, based on chronic degenerative damages to the tendon[9,24]. These injuries often result in poor postoperative outcomes[25]. Four factors determine the prognosis of avulsion injuries: the extent of the retraction of the tendon, the remaining blood supply, the time interval between trauma and surgery and the presence and size of an osseous fragment[18]. According to Leddy and Parker[18,20], three different injury types with varying prognosis and therapy exist. Characteristics for type 1 are retractions of the tendon into the palm, the Vinculae

are ruptured, the blood supply is interrupted and surgery is needed within 7 d to 10 d after trauma. In type 2 injuries, the tendon retracts to the PIP joint only and is held by an intact vinculum. Therefore, the blood supply is not compromised and surgery can be delayed without risking a poor outcome. Type 3 injuries are osseous ruptures of the FDP tendon at the distal phalanx. Thank to the intact vinculum, these injuries have a good prognosis. Blunt trauma, in terms of ruptures, also happen to the tendon sheath and the pulleys[3,11,13,26-28]. Rarely, blunt disruptions of the lumbricalis origins at the flexor tendon on the palm level do occur in high stress finger activity (e.g., rock climbing) (Figure 4).

Open tendon injuries are common findings in trauma and orthopedic patients and need primary surgical treatment. A differentiation between complete or partial rupture has to be made. A lesion of less than 60% of the tendon's diameter should be treated conservatively[13]. Multiple biomechanical *in vivo* and *ex vivo* studies proved that conservatively treated partial tendon ruptures showed a higher tear-resistance ($P < 0.05$) compared to the surgically treated ones[13]. Chronic degenerative tendon ruptures as seen in rheumatic patients must be distinguished from these traumatic ruptures. A combined entity we see in our patients, mostly in rock climbers, is a chronic tendinosis and degeneration and then finally an acute tear (Figures 5 and 6). High intensive stress combined with chronic tenosynovitis (peritendinosis) and tendinosis (endotendinitis) can lead to a rupture of the tendon, even although only minor stress was applied[25]. Additional to the poor blood supply of the tendon, microtrauma and microscopic structural damage can lead to a modest prognosis.

TENDON SHEATH AND PULLEY LESIONS

Injuries of the tendon sheath and the pulley system are often associated with flexor tendon injuries as these structures form a functional unit[1-3,5,6,10,13,29]. In open lesions, these structures must be repaired as exactly as possible to guarantee biomechanical efficiency and nutrition of the tendon[12,13]. Closed injuries, especially pulley and c-ligament lesions, are common findings in athletes (climbers)[3,6,25,30-32] and only casually in other patients groups[33].

DIAGNOSIS

Compared to closed injuries, open lesions are easy to diagnose. Closed injuries should especially be looked at carefully, not only to get a specific diagnosis but also to locate the topographical region of the injury. In clinical examinations of lacerations, one has to remember that even small cuts can cause severe damage underneath the surface, for example, a partial rupture of 90% of the tendon can appear to be functionally intact but after a couple of days this tendon will rupture secondary to relatively minor stress[13]. The function of the FDS and FDP tendons need to be examined separately. A pulley lesion may become apparent with a bowstring phenomenon (Figures 7 and 8). Concomitant injuries of the bundle of nerves and vessels or open joint capsule injuries combined with palmar plate injuries need to be excluded. In addition to the clinical examination, ultrasound and magnetic resonance imaging (MRI) are well established tools to detect closed tendon injuries, as well as to assess injuries to the pulleys and tendon sheaths. Ultrasound is performed in the supine position with longitudinal and transversal planes using a linear transducer (10-13 MHz). For signal enhancement, a gel standoff pad or examination in a warm water basin is used. Only in rare cases an additional MRI [or (computed tomography) CT] needs to be performed[3,30,34]. A considerably advantage of the ultrasound is the possibility of dynamic examination, which can demonstrate tendons excursions through "forced flexion" better than a static method[35-37]. Additionally, inflammatory processes can be easily demonstrated (effusion, increased blood flow) and cellulitis, ganglion cysts and phlegmonia can also be visualized and detected[38]. Diagnosis of a pulley lesion is performed in forced flexion of the finger, meaning active pressure of the finger towards the transducer[3,39]. Thereby quantifications of the enhanced distance between bone and flexor tendon, as seen in pulley ruptures, can be made[3,39]. If no specific diagnosis can be made using the ultrasound, CT and MRI are available as other diagnostic tools[30,40]. The ultrasound has the advantage that dynamic examinations[30] can be made[41]. Using the MRI, a more specific differentiation of inflammatory processes or post traumatic edema can be made[37].

THERAPY

In the following section, the essential therapeutic approach is presented. Nevertheless, focus is kept on considerably rare injuries, as the treatment of common injuries, e.g., flexor tendon ruptures, is standardized and well described elsewhere[1,10,13,14,17-20,42-50].

EXTENSOR TENDON INJURIES

Blunt disrupters of the extensor tendons of the DIP ("mallet finger") (Verdan Zone 1[7]) can be treated conservatively using a stack splint if the injury is just a partial rupture in which the collateral fibers of the "Landsmeer ligaments" (lig. retinaculare obliquum) are intact[19]. A surgical treatment analogous to open injuries is required if a extension deficit of more than 45° is present[18]. A solemnly percutaneous wire fixation in hyperextension is not recommended any more[19] and instead "mallet" fractures are treated by using osteosynthesis techniques, such as screws, tension band or K wiring.

In the extensor tendons, their specific shape has to be acknowledged in performing repairs. The further distal the muscular part, the flatter the diameter of the tendon gets. Therefore, suturing techniques used in

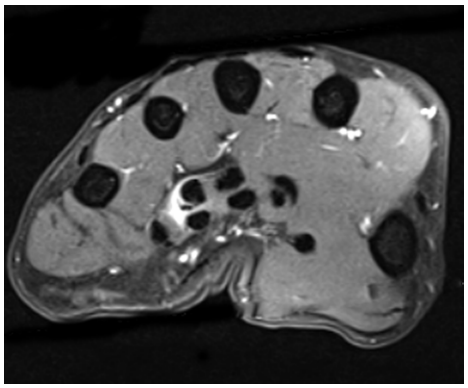

Figure 5 Lumbrical tendon rupture (note the edema and the dislocated lumbrical tendon from the flexor tendon).

Figure 6 Degenerative flexor tendon rupture in a long term rock climber with chronic tendinosis.

flexor tendon repairs, a central suture and circular fine adaptation of fibers, cannot be used[1]. In open injuries, the wound itself should be used as a surgical approach and extended Z-shaped. Due to the diameter of the tendon, several U-shaped sutures should be used, optionally combined with fine adaptation sutures using PDS (5-0, 6-0). In treatment of the tractus intermedius (PIP joint) and extensor tendon injuries at the level of the DIP joint, the suture should be secondarily stabilized with a temporary K-wire arthrodesis (diameter 0.8-1.0 mm) (6 weeks immobilization in a neutral position). The wire should be inserted diagonal to the joint space, not longitudinal through the fingertip, to avoid bacterial transmission[19]. The use of the "Lengemann wire suture" is no longer recommended[1,19]. Extensor tendon injuries of the hand require immobilization using the intrinsic plus position. Accompanying injuries of the finger joints need to be treated adequately and the articular capsule needs to be fixed. Extensor tendon subluxations and luxations on level with the MCP joint are mostly revealed subsequently and require a surgical repair or secondarily, a plastic reconstruction[18]. Osteophytic irritations of the extensor tendons ("extensor hood syndrome") can usually be treated conservatively[51].

FLEXOR TENDON INJURIES

Open flexor tendon injuries require a surgical procedure

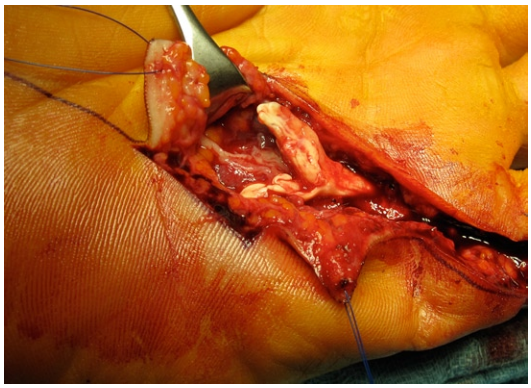

Figure 7 Degenerative flexor tendon rupture in a long term rock climber with chronic tendinosis. The histology showed a mucoid degeneration with numerous blood vessel proliferations and a siderosis as a sign of an older bleed[9].

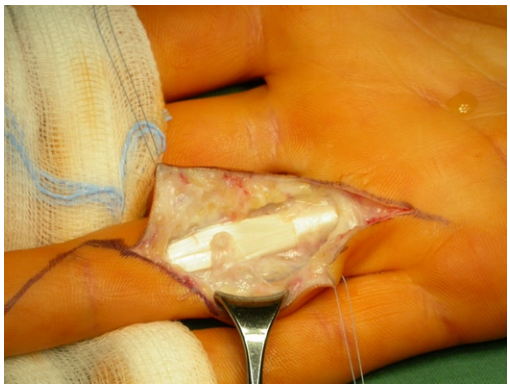

Figure 8 Ruptured A3 pulley (the tendon sheath with chronic inflammation is already removed).

using magnifying glasses[1]. Surgery should be performed under plexus or general anesthesia with the use of a tourniquet. Surgery should be performed in the operating room using perioperative antibiotic prophylaxis. As flexor tendon injuries are often combined with injuries to the vessels and nerves, these structures must be thoroughly displayed[1,9,10,12,13,17,42-45,52-59]. A palmar "Bruner" incision is followed by a modified (Zechner) Kirchmayr-Kessler suture with non absorbable plaited 4-0 yarn. For fine adaptation "running-sutures", a monofil 6-0 yarn is used, either absorbable or non absorbable (e.g., PDS)[1,9,10,12,13,17,42-45]. If annular pulleys need to be released, a double door wing shaped incision is performed and secondarily reconstructed as an extension plastic[42]. Extensive retracted proximal tendon stumps can be produced and refixed, using an additional incision to insert a flexible catheter through the tendon sheath-pulley system and fix the tendon stump to it, to pull the tendon further distally[18]. Blunt flexor tendon disruptions are sutured, osseous avulsions are refixed transosseus (using a periosteal flap if necessary[18]). A primary arthrodesis of the DIP should be considered in excessive degenerative tendon lesions. If tendon tissue is resected, the tendon must be lengthened using a Z-plastic or a forearm tendon recession[9].

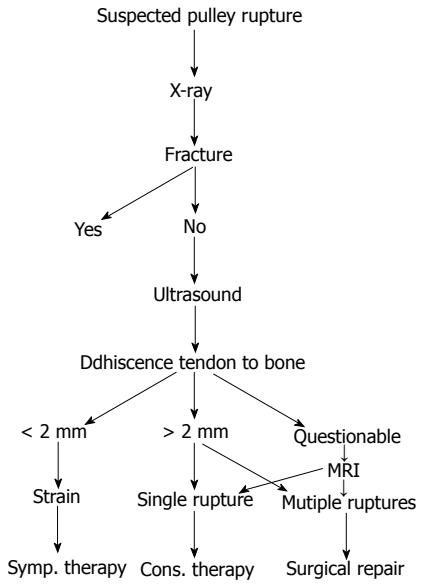

Figure 9 Algorithm for pulley injuries[3]. MRI: Magnetic resonance imaging.

INJURIES OF TENDON SHEATH AND PULLEY SYSTEM

Injuries of the tendon sheath and pulley system are often combined with flexor tendon lesions as these structures form a functional unit[1,2,6,9,10,13,34,60]. Open lacerations should be reconstructed, even with lengthening if necessary. Therapy of closed pulley lesions should be treated according to Figure 9. Singular pulley injuries should be treated conservatively, whereas multiple injuries should be treated surgically using pulley plastic surgery (Palmaris longus transplant as a 1.5 loop according to Widstroem[61,62] or Schöffl *et al*[63] or a Retinaculum Extensorum Flap, performed according to Lister[64], Moutet *et al*[65] or Gabl *et al*[60]).

POSTOPERATIVE CARE

Extensor tendon injuries require immobilization (transfixation or an "intrinsic plus" cast)[1,13,18,19]. A dynamic aftercare using a reversed "Kleinert" extension splint for extensor tendon injuries of the zones Ⅵ-Ⅷ was discussed recently[19]. Studies show a similar outcome for Verdan type Ⅱ-Ⅳ injuries[7], whereas type Ⅴ-Ⅶ seem to have a better outcome if treated with dynamic aftercare[19]. Aftercare of flexor tendon injuries should follow the scheme of dynamic early mobilisation, as proposed by Kleinert[66], which allows passive flexion, carried out by a rubber string and active extension[1,10,13,17,42,43,45]. The wrist flexion of the cast is reduced gradually and after 3 wk, a Kleinert-bandage with rubber band restraints can be applied. Free functional movement is allowed after 5 wk, full exposure after 3-4 mo. An "intrinsic tendon healing" is sought for, which requires intermittent tension stress[42], achieved by the "early passive movement" principle[10,43,45]. Recently, more progressive aftercare schemes postulate active flexion of the tendon immediately after surgery but are not yet commonly established[10]. Lohmeyer *et al*[44] evaluated standards in therapy of combined flexor tendon and nerve injuries in German centers of hand surgery. Isolated injuries of type Ⅱ-Ⅳ were treated with schemes of early dynamic movement in all hand surgery centers; combined injuries were treated dynamically immediately after surgery by 55% of the interviewed centers. Pulley plastic aftercare is treated with 2 wk of immobilization, followed by early functional treatment using an external pulley support, first as a thermoplastic ring and later through a protective tape (h-tape)[67].

PROGNOSIS AND COMPLICATIONS

The most concerning complications in flexor tendon surgery are adhesions and suture dehiscence, maybe due to prematurely active movement[12]. This is an expression of "extrinsic tendon healing", characterized by a distinctive inflammatory phase with migration of peritendinous fibroblasts[14,15]. For an intrinsic healing, an intact system of tendon sheath and pulleys is essential, which produces a synovial fluid-like environment that supports the healing process until the vascular system is regenerated. Scrubbing of the tendon along the tendon sheath pumps nutritive synovial fluid into the tenocytes and adhesions are prevented[12] (Figure 9). Many papers report excellent outcomes in 70% after flexor tendon sutures, similarly to Brug[42], who reports 71.8% excellent, 13% good and 8.8% poor results, according to the Buck-Gramcko-Score[68], in a study of 258 patients. According to Geldmacher, even with optimal premises, less than 90% excellent outcomes are to be expected and only 13% with poor premises[42]. Blunt and degenerative avulsion injuries show an even worse outcome[12].

In various studies, different agents to improve slippage after tendon sutures and prevent adhesions were evaluated. Alpha-Aminproprionitrile is said to prevent cross links of collagen but appeared to be toxic[12]. D-Penicillamine, cis-Hydroxyproline, Triamcinolone, Polyvinylpyrrolidone and others have not been proven to have a reliable effect or have been shown to be impracticable[12]. Hyaluronic acid may improve intrinsic tendon healing but studies have not shown reliable data[12]. Besides adhesions and following essential intense physiotherapy, Quengel treatment and tenolysis, dreaded complications are contractures, secondary ruptures, pulley malfunction, snapping finger and infection. Pulley malfunction either by rupture or insufficient initial reconstruction indicates secondary pulley plastic[3,12,28].

SECONDARY TENDON REPLACEMENT AND TRANSFER

If flexor tendon lesions were not treated adequately at first, secondary reconstruction is necessary. Decision to perform a one or two step reconstruction of the tendon is dependent, among other reasons, on the localization

of the injury, the extent of scar tissue and an intact pulley system[13,43]. In 1912, Lexer had already reported free tendon transplants[13]. In 1918, Bunnel called for an atraumatic approach to surgery: usage of the palmaris longus tendon as well as a tourniquet and conservation of the pulley system for secondary reconstruction[13,69]. Even although inconsistent, nowadays the tendon of the m. palmaris longus is usually used for reconstruction[13]. The tendon is found in 85% of patients[13]. As an alternative, the tendon of the m. plantaris can be used, which is found in 93% in patients[13]. Extensor tendons of fingers and toes can be other alternatives, as well as the FDP tendon of the fifth finger[12,13]. After reconstruction of the flexor tendon channel *via* insertion of a silastic bar and secondary transplantation, further reconstruction follows either in one or two steps. Other possibilities are tendon transfers and compensational replacement surgery, which are not discussed in this paper.

CONCLUSION

Open and blunt tendon injuries of the hand, especially injuries of flexor tendons, are serious injuries that should be treated by a skilled surgeon. Blunt injuries need to be detected and treated consequently as well as open lesions. If in doubt, ultrasound and MRI can be of great diagnostic benefit. Consistent initial therapy is as important for a good overall outcome as is proper aftercare. Close cooperation of the surgeon and the patient is essential, as well as physiotherapy and occupational therapy.

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TOPIC HIGHLIGHT

Quanjun Cui, MD, *Series Editor*

Management of failed metal-on-metal total hip arthroplasty

Justin W Griffin, Michele D'Apuzzo, James A Browne

Justin W Griffin, Michele D'Apuzzo, James A Browne, Department of Orthopaedic Surgery, University of Virginia Health System, 400 Ray C Hunt Dr. Charlottesville, VA 22903, United States

Author contributions: Griffin JW and D'Apuzzo M performed the literature search; Griffin JW, D'Apuzzo M and Browne JA wrote and edited the paper.

Correspondence to: James A Browne, MD, Department of Orthopaedic Surgery, University of Virginia Health System, 400 Ray C Hunt Dr. Charlottesville, VA 22903
United States. jab8hd@virginia.edu
Telephone: +1-434-2430236 Fax: +1-434-2430242
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Abstract

The theoretical advantages of metal-on-metal (MOM) bearing couples in total hip arthroplasty (THA) have been recently balanced by concerns regarding adverse local and systemic effects. Higher than anticipated early revision rates have been reported by several joint registries. Failed MOM hips present with a spectrum of symptoms and findings and traditional methods of failure must be considered in addition to the failure modes that appear to be unique to the MOM bearing couple. Metal hypersensitivity and soft tissue immune reactions remain incompletely understood and require careful ongoing study. The tools available to evaluate MOM THAs and the indications for revision surgery remain to be defined. Outcomes following revision of MOM hips appear to depend on appropriate evaluation, early identification, and appropriate surgical management.

Key words: Hip arthroplasty; Metal-on-metal; Revision joint arthroplasty; Pseudotumor; Aseptic lymphocytic-vasculitis-associated lesions; Metal hypersensitivity

Peer reviewer: NAS Kemal, Professor, Department of Physical Medicine and Rehabilitation, University of Dicle, Medical School, 21280 Diyarbakır, Turkey

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INTRODUCTION

Total hip replacement has been one of the most performed and predictable surgeries available in the United States for the past decade. In an effort to reduce wear debris and failure related to osteolysis, improved metal-on-metal (MOM) articulations were reintroduced in the 1990s and remain the only option for surface replacement arthroplasty[1]. Following this rebirth, a variety of soft-tissue reactions and periprosthetic lesions were described in patients with MOM arthroplasty[2,3]. This led to concerns regarding potential metal hypersensitivity or immunogenicity and its potential role in catastrophic failure of MOM implants. In addition, increased early rates of revision are being reported by national joint registries. Despite these concerns, outcomes of MOM hip replacements related to performance have been favorable in some studies[4,5].

At the present time, methods to evaluate the patient with a MOM bearing are poorly defined and indications for revision are unclear. Additionally, negative public press regarding MOM bearings has led patients to seek out diagnostic testing in the absence of symptoms[6]. The purpose of this paper is to describe current concepts related to the evaluation and management of MOM total hip arthroplasty (THA).

HISTORY

The first generation of total hip implant with a metal-

on-metal articulation is attributed to George McKee of Norwich, England in 1953. The design was primitive but many lasted for more than 7 years. However, a high number of early failures discredited this bearing couple. Concerns regarding "polyethylene disease" with the metal-on-polyethylene articulation advocated by Charnley led to the development of a second generation of MOM implant designs in the early 1980s. Intrigued by the good results of certain metal-on-metal configurations, many thought that this bearing could be an improvement on polyethylene and deserved further evaluation. From the beginning of this second generation of implants, however, serious concerns were raised regarding the risks associated with an increased level of circulating metal ions, and this concern slowed down further development of this bearing couple. The third generation of MOM design encompassed an uncemented version of the second generation implant, which provided a significant number of lessons as it was used in relatively high numbers in clinical practice. The failures associated with these implants appeared to be both mechanical and biological in nature.

The fourth and current generation of MOM design is the result of continued work on the tribology of metal-on-metal. Larger diameters of these implants appeared to improve the lubrication and wear properties of the bearing couple. In addition, the promise of better stability and increased range of motion led to widespread enthusiasm and early clinical adoption of this technology[7].

EVALUATION OF THE PAINFUL MOM THA

The diagnostic evaluation of MOM THAs has not been standardized and varies by surgeon experience and patient presentation. A detailed history and physical examination remains a critical first step in developing a differential diagnosis of the painful MOM hip. Failures seem to occur more in females. Initial clinical evaluation should focus on patient symptoms, which commonly include groin pain[8]. Other common symptoms include difficulty with stair climbing, clunking, catching and limited strength[9]. Many patients report never having recovered from the initial operation. Ilipsoas irritation may be present due to the large femoral head size. Symptoms that suggest infection and loosening should also be noted. Physical examination may reveal common problems such as trochanteric bursitis or radiating back pain and exclude the hip itself as the cause of symptoms.

Initial imaging should include high quality radiographs to evaluate for obvious causes of failure that are not unique to MOM THAs. Loosening and lysis may be observed. Iliopsoas impingement has been seen with excessively large femoral head implant size[10]. Component position and should be carefully assessed with anteroposterior and lateral radiographs, acknowledging that many of the modern acetabular components used in MOM THA measure less than a complete hemisphere. Increasing evidence suggests that high inclination angles and excessive anteversion of the acetabular component, small head sizes, and individual prosthesis designs are all contributing factors in the failure of MOM hips[11]. Desy *et al*[12] performed a retrospective review of 91 patients over 2 years evaluating radiographically the acetabular inclination, version and femoral component alignment of patents with MOM hip replacements and reported that a large acetabular inclination directly correlated with metal ion concentration. Hart *et al*[13] conducted a prospective trial analyzing blood metal ion levels, computed tomography (CT) scanning and clinical exam in one hundred patient with MOM hip replacements and found that cup inclination, version angles and gender influenced blood cobalt and chromium levels. Additionally, in this study the changes in inclination had the greatest effect on blood metal ion concentration[13]. While studies exist with conflicting results, the general consensus appears to be that poor component positioning appears to play a role in the failure of MOM implants.

There is no generally accepted test for metal hypersensitivity. Patch testing and lymphocyte tests have been used in an attempt to determine sensitivity[1]. Hallab *et al*[14] reported that the prevalence of dermal sensitivity in patients with failed MOM implants was 50%, which was higher than the general population at 25%. However, based on the current evidence, we are unable to suggest that general patch testing or commercial testing is recommended during assessment the painful MOM hip[15]. Lymphocyte transformation tests as well have shown limited clinical utility in the evaluation of MOM THA.

Routine laboratory data obtained in the evaluation of a painful MOM THA includes the erythrocyte sedimentation rate and c-reactive protein. Ruling out infection as a cause of the painful total hip remains paramount in MOM hip articulations. Infection remains a common cause of early with MOM implants and is likely more common than hypersensitivity responses[9]. Aspiration should be performed in the presence of abnormal inflammatory markers to further evaluation for infection.

Controversy exists regarding serum cobalt and chromium ion levels and the impact of elevated serum ion levels in not understood. Several methods of measuring metal ion levels in the blood are available and variability between different laboratories exists. Elevated metal-ion levels following MOM has been known to exist for some time, although the biological effects of these ions is uncertain. Several studies have suggested a higher rate of revision in the presence of elevated ion levels, although patients with asymptomatic MOM hips and elevated ion levels have been observed. It has been suggested that elevated metal-ion levels correlate to increased wear and pseudotumor formation[16]. Metal-ion levels measuring less than 7 parts per billion appear to be predictive of a well-functioning MOM THA after the initial run-in period[17].

Patients with a painful MOM THA are often evaluated with cross sectional imaging or ultrasound. Standard indications for obtaining further imaging do not exist, although this should be considered in patients with com-

ponent malpositioning, abductor dysfunction, or clinical concern for pseudotumor. Magnetic resonance imaging protocols to reduce the metal artifact (MARS) have been developed. Revision surgery should be considered in the event that this imaging reveals soft tissue reactions, fluid collections, or tissue masses.

UNIQUE FAILURE MODES IN MOM THA

Local adverse soft tissue reactions to MOM bearings have been given a variety of names: aseptic lymphocytic-vasculitis-associated lesions (ALVAL), pseudotumor, necrosis, adverse reaction to metal debris, and adverse local tissue response. The incidence of these reactions appears to be rare with an incidence likely below 1%. Various mechanisms have been proposed to explain these reactions, including patient, technique, and implant factors. The common pathway for this failure mode appears to be increased wear or corrosion with excessive release of metal ions and nanoparticles.

The relative surface area and biological activity usually increase as particle size diminishes. Metal particles are considerably smaller than debris from conventional metal-on-polyethylene bearings. This explains the increased total number of particles released from MOM hips, reported to be more than two orders of magnitude higher than that found with conventional bearings[18,19]. The wear debris is phagocytosed by macrophages and once in the acidic intramedullary environment of the lysosomes, the cobalt-chrome particles are subject to corrosion, producing high intracellular levels of ions, which can cause cell death. It has been shown that, following phagocytosis of metal particles, the osteoblastic activity of the cell is impaired which may contribute to the cellular events that occur during aseptic loosening and soft-tissue destruction[18,20]. Metal wear debris may also cause DNA damage and genomic instability.

A prominent histologic finding associated with MOM bearings is perivascular lymphocytic infiltration[21]. These findings have been termed ALVAL. A delayed-type hypersensitivity response to metal debris has been suggested as a cause of these symptoms, although the clinical significance and underlying cause of this local tissue response remains unclear.

SURVIVORSHIP OF MOM THA

Registries and long-term clinical studies have provided outcomes relating to the survivorship of metal bearing surfaces[22]. Several clinical series have reported good survivorship with certain implant designs. Neumann *et al*[5] recently provided 10 year follow up data from 100 MOM hip replacements with a 93% overall survivorship. Another 10 year radiographic survivorship study highlighted a 98.3% overall survivorship with pelvic osteolytic lesions in only 2.3% of patients. Engh[23] recently reported 5 year follow up results from a retrospective cohort of modular MOM with a 98% survivorship and a 0.3% rate of local reaction to the MOM bearing surface.

However, several recent studies and international total joint registries have documented increased early failure rates with certain MOM THA designs. Park *et al*[24] reported osteolysis in over 5% of patients at short term follow-up with MOM hips. Korovossis *et al*[25] also reported a significant rate of osteolysis with a high early revision rate. The DePuy ASR XL acebular system (Warsaw, Indiana, United States) was recalled in August 2010 after UK joint registry data indicated a 13 percent revision rate within 5 years.

Patient gender may influence MOM implant survival. Amstutz *et al*[4] conducted a large comparison study of clinical survivorship in men *vs* women and found that the revision rate was higher in the women's group; however, this effect did not hold up when component size and surgical technique were equal between groups. Further investigation into this topic is ongoing.

TECHNIQUES AND OUTCOMES OF REVISED MOM THA

Revision of the failed MOM hip must be individualized to each individual patient and the reason for failure. A common scenario involves revision of the modular femoral head and acetabular component in the presence of a well-fixed femoral stem (Figure 1). An isolated head and liner exchange may be possible if a well-fixed and well-positioned acetabular component will accept a modular polyethylene or ceramic insert. Resection of necrotic tissue and metallosis is recommended. Extensive soft tissue damage can be a concern in the presence of a pseudotumor and a constrained liner should be considered in the event that the abductors are insufficient.

Most published data on outcomes after revision of modern MOM bearings is limited and the results are from small cohorts with limited duration of follow-up. Common preoperative symptoms of groin pain and mechanical symptoms tend to resolve reliably following conversion to an alternative bearing surface[26]. However, revision of MOM bearings has a reported complication of 7%-9% including dislocation[26]. Metal ion levels tend to fall within the first 3 mo after revision surgery but may not fully normalize at short term follow-up (Figure 2).

CONCLUSION

Metal on metal bearing couples offer the theoretical benefit of low wear rates and increased stability. However, unique failure modes of these implants appear to exist with associated problems making them a poor choice for primary hip arthroplasty at this time. Clinical evaluation of painful MOM bearing surfaces should include a careful history and physical exam in addition to laboratory evaluation and radiographic studies. Outcomes following revision surgery vary and depend on the amount of tissue destruction and bone loss from osteolysis. Early revision when problems are identified seems to offer the

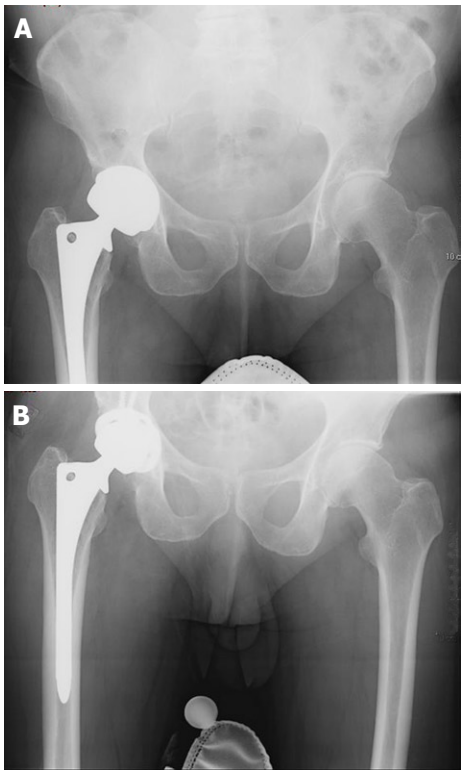

Figure 1 Pelvis radiograph of 56 year old male with a painful right metal-on-metal hip replacement (A). He complained of increasing groin pain and difficulty with ambulation. AP pelvis 6 mo after revision total hip replacement. The well-fixed femoral stem was left in place and the bearing revised to a metal-on-polyethylene bearing couple (B). The patient's preoperative symptoms resolved entirely.

best chance at a good outcome.

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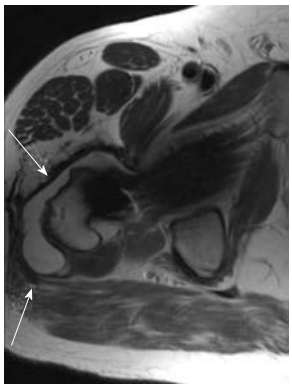

Figure 2 Complex pseudotumor-like fluid collection (arrows) around a painful metal-on-metal total hip arthroplasty.

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REVIEW

Adaptability of anticipatory postural adjustments associated with voluntary movement

Eric Yiou, Teddy Caderby, Tarek Hussein

Eric Yiou, Tarek Hussein, Laboratory Complexité, Innovation, Activités Motrices et Sportives, Team Risque, Intervention, Mouvement, Equilibre, UFR STAPS, University of Paris-sud, 91440 Orsay Cedex, France
Teddy Caderby, UFR of Human and Environmental Sciences, University of La Réunion, 97430 Le Tampon, Reunion Island, France
Author contributions: Yiou E, Caderby T and Hussein T were all involved in the conception and writing of the manuscript.
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Correspondence to: Eric Yiou, PhD, Laboratory Complexité, Innovation, Activités Motrices et Sportives, Team Risque, Intervention, Mouvement, Equilibre, UFR STAPS, University of Paris-sud, 91440 Orsay Cedex,
France. e.yiou@libertysurf.fr
Telephone: +33-1-69153158 Fax: +33-1-69156222
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Abstract

The control of balance is crucial for efficiently performing most of our daily motor tasks, such as those involving goal-directed arm movements or whole body displacement. The purpose of this article is twofold. Firstly, it is to recall how balance can be maintained despite the different sources of postural perturbation arising during voluntary movement. The importance of the so-called "anticipatory postural adjustments" (APA), taken as a "line of defence" against the destabilizing effect induced by a predicted perturbation, is emphasized. Secondly, it is to report the results of recent studies that questioned the adaptability of APA to various constraints imposed on the postural system. The postural constraints envisaged here are classified into biomechanical (postural stability, superimposition of motor tasks), (neuro) physiological (fatigue), temporal (time pressure) and psychological (fear of falling, emotion). Overall, the results of these studies point out the capacity of the central nervous system (CNS) to adapt the spatio-temporal features of APA to each of these constraints. However, it seems that, depending on the constraint, the "priority" of the CNS was focused on postural stability maintenance, on body protection and/or on maintenance of focal movement performance.

Key words: Anticipatory postural adjustments; Adaptability; Voluntary movement; Balance; Biomechanics; Motor control

Peer reviewer: Vassilis Paschalis, PhD, Deparment of Physical Education and Sport Science, University of Thessaly, Karies, 42100 Trikala, Greece

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INTRODUCTION

The control of balance is crucial for efficiently performing most of our daily motor tasks, such as those involving goal-directed arm movements or whole body displacement. Humans, as in all terrestrial species, indeed evolve in a gravity field that permanently tends to induce postural destabilization by its attracting effect towards the center of the earth. The control of these gravitational forces is necessary to maintain balance in a given posture, which constitutes a prerequisite in efficiently performing goal-directed movements and also to displace our body in the intended direction. In addition, movements themselves perturb our balance and can be considered as self-inflicted perturbation. This perturbation originates from the internal forces and torques elicited by the movements which are transmitted through the whole body to the support surface, where ground reaction forces are produced (the latter are external forc-

es). According to the laws of mechanics, these external forces act to destabilize the whole body in the direction opposite to that of the movement. For example, raising the arm forwards to the horizontal induces internal forces and torques at the shoulder level that initially tend to destabilize the rest of the body backwards and downwards. In addition, raising the arm displaces the center of gravity which disrupts the initial conditions of balance. A biomechanical model of the human body showed that the perturbation elicited by a rapid arm raising movement from the erect posture may potentially leave the subject "to fall flat on their face"[1].

The purpose of this article is twofold. Firstly, it is to recall how balance can be maintained during voluntary movement despite these different sources of perturbation. The importance of the so-called "anticipatory postural adjustments" (APA), taken as a "line of defence" against the destabilizing effect induced by a predicted perturbation, will be emphasized. In daily situations, this line of defence might be challenged by various constraints imposed on the postural system (or "postural constraints"). Examples of postural constraints are fatigue of the postural musculature, instability, time pressure (i.e., having to perform a motor task in a limited time), "fear of falling" (e.g., when one is to move on an elevated surface) and so on. When facing such constraints, the central nervous system has to develop adaptive postural strategies to efficiently perform the intended motor task and maintain balance. The second purpose of this review is to report the results of recent studies that questioned the adaptability of APA to various postural constraints in healthy young adults.

BALANCE MAINTENANCE DURING VOLUNTARY MOVEMENT

The way that balance can be maintained during voluntary movement has inspired many authors, at least since Leonardo da Vinci, and it is still a matter of intensive research. One common statement stressed in most of these researches is that voluntary movements are subjected to two antagonistic constraints. One is to move the "focal" segment(s) (i.e., the body segments directly involved in the voluntary action) towards a goal; the other is to stabilize the "postural" segment(s) (i.e., the body segments not directly involved in the voluntary action) in order to maintain balance. This process of postural stabilization necessarily involves that dynamical phenomena take place in the postural segments that act to minimize the self-inflicted perturbation. These dynamical phenomena correspond to the "postural adjustments". The following paragraph traces back a brief history of this notion of postural adjustment (for more details on this aspect, the reader may refer to the reviews by[2-4]), with an emphasis on those postural adjustments that precede the onset of the voluntary movement, i.e., on APA.

Brief history of the "postural adjustment" concept

de Vinci[5] wrote in around 1500: "I maintain that, when a man stands motionless upon his feet, if he extends his arm in front of his chest, he must move backwards a natural weight equal to that, both natural and accidental, which he moves towards the front" (cited from Gahery[3]). This backward body movement corresponds to what we now call "postural adjustments". The existence of postural phenomena related to voluntary movement was described more recently by Babinski[6] at the end of the nineteenth century. Babinski observed that when healthy subjects were asked to voluntarily bend their head and trunk backwards, their knees systematically flexed. Thank to this postural adjustment, the center of gravity remains over the base of support and balance is maintained. Babinski also noted that this compensatory leg movement was absent in cerebellar patients, which caused them to fall backward. Although important, this observation was limited because it did not provide information regarding the timing of these postural adjustments in relationship to the onset and termination of the voluntary movement. The necessity to develop postural adjustments during voluntary movement was also emphasized by Hess[7] in the middle of the twentieth century. Hess distinguished three components in his model of voluntary movement. Each component was symbolized by one character: the first one, the leaper was to jump from the shoulders of the second character, the postural frameworker. This second character also requires the intervention of the third character, the supporter, to maintain balance during the jump. The jump is efficient when the three characters properly coordinate their activity. The jump is unsuccessful when there is a lack of coordination between the three characters. In this model, the activity of the leaper, qualified as "teleokinetic", was distinguished from the postural activity of the frameworker and of the supporter, qualified as "ereismatic".

The modern view of postural adjustments associated with a voluntary movement integrates these different historical sources. The "Posturo-Kinetic Capacity" (PKC) has been defined as the capacity of an individual to generate efficient postural adjustments in response to a perturbation (internal or external) (see[2,8,9] for reviews). The PKC theory emphasizes the necessity to develop postural adjustments in anticipation of the forthcoming perturbation in order to optimize the control of balance. These postural adjustments correspond to the so-called APA and are related to the "action equilibration" mechanisms of André-Thomas[10]. Because of their precedence relative to the onset of the voluntary movement, APA has been thought to reflect the existence of an internal forward model within the central nervous system that takes into account the dynamic consequence of an expected perturbation and that generates responses to counteract these consequences[11]. The PKC theory also emphasized the importance of postural joint mobility to ensure an efficient postural counter perturbation. Specifically, this theory predicts that any factor constraining postural joint mobility (e.g., with aging or pathology) would alter the focal movement performance (e.g., the maximal ve-

locity of a pointing task) and the postural stability. The performance of a voluntary movement and postural stability would thus tightly depend on the PKC, which is in agreement with the Hess's model of posture and movement coordination[7]. Recent experimental data are in agreement with this theory (see paragraph Ⅲ.1).

Anticipatory postural adjustments

To date, it is to the merit of the Russian school to report the existence of APA in human[12] and animals[13]. Specifically, authors reported an activation of the ipsilateral biceps femoris muscle (hip extensor/knee flexor) prior to the activation of the focal muscle (deltoideus anterior) during rapid arm raising from the erect posture in humans[12]. The existence of APA was later confirmed by several authors during a more or less similar paradigm involving the upper limb and it was completed with an exhaustive description of the complex patterns of anticipatory activation/inhibition of the postural muscle within the legs and trunk segments[14-17].

The function of APA during an upper limb task performed from a quiet standing posture was argued based on experimental data obtained with a force plate[18,19]. Specifically, authors showed that the onset of voluntary arm raising was systematically preceded by a dynamical phenomena detected by the force plate, which included forward and upward acceleration of the center of gravity. As these phenomena cannot be due to the transmission of forces associated with the focal movement to the support surface (because they precede it), they are necessarily due to postural movements. The latter correspond to APA. It is noteworthy that the biomechanical effects of these APA are opposite in sign to the biomechanical effects of the perturbation associated with the forthcoming arm movement, which is initially directed backwards and downwards as described above. For this reason, it was proposed that the function of the APA is to counter the perturbation associated with the forthcoming voluntary movement in advance[18,19]. The result is that the APA duration and/or amplitude increases with the velocity of the arm and when an inertia is added to the wrist[18-21], i.e., when the postural perturbation is increased in line with this function of postural counter perturbation. As proposed in a recent study by Yiou *et al*[22], the need to counter this perturbation in advance might stem from the existence of electromechanical delay between the onset of postural muscle activation and the onset of the counter perturbing inertial forces generated by the postural segments. This delay ranged from 80 ms to 100 ms, depending on the muscle[23]. Thanks to this anticipatory activation of postural muscles, the postural counter perturbation could be effective at the beginning of the voluntary movement, i.e., at the beginning of the perturbation. It is now generally admitted that APA do not totally compensate for the postural perturbation. Postural adjustments occurring during and after the voluntary movement were also described in the literature (albeit to a much less extent[19,24-26]). The latter are termed "consecutive postural adjustments" (CPA) and deal with the actual postural perturbation.

Besides the fact that APA are associated with motor tasks performed from a fixed base of support (e.g., arm raising from the erect posture), APA are also described in various motor tasks involving a change in the size of the base of support, such as during leg flexion, rising on the toes, gait initiation, *etc*. Gait initiation has classically been defined as the transient period between a quiet standing posture and steady walking[27]. It can be decomposed into an APA period, which is the period between the onset of the mechanical or electromyographical (EMG) changes from the background level and the time of swing heel off (which corresponds to the onset of the voluntary movement), and an execution phase that ends at the time of swing foot contact with the support surface. Balance in the initial standing posture is disrupted during APA by backward center of pressure displacement, which promotes the propulsive forces necessary to displace the center of gravity forwards during the execution phase[27,28]. This anticipatory center of pressure displacement is induced by ankle synergy (bilateral soleus inhibition followed by strong tibialis anterior activation). APA along the anteroposterior direction thus serve to create dynamical conditions necessary for gait progression. Authors have shown that the amplitude of these APA (in terms of maximal backward center of pressure displacement) is predictive of the progression velocity of the center of gravity, i.e., gait initiation performance[27]. Along the axis orthogonal to the progression axis (mediolateral axis), APA are also generated and act to stabilize the whole body during the execution phase[28-32]. Indeed, it is noteworthy that the act of lifting the swing foot induces a reduction in the base of support size, which is then limited to the single stance foot's contact with the ground. It follows that if the center of gravity is not repositioned above the base of support before the time of swing foot off, the whole body will become unstable during the execution phase and will tend to fall laterally towards the swing leg side under the effect of gravity. During voluntary gait initiation, this natural tendency toward instability is invariably countered in advance by mediolateral APA. These mediolateral APA are manifested as a center of pressure displacement towards the swing leg side which serves to shift the center of gravity in the opposite direction and to provide an initial velocity at the start of the execution phase. During rapid gait initiation, it is known that the center of gravity is not propelled directly above the base of support[29,30]. Nevertheless, in moving the center of gravity closer to the point of support, i.e., by reducing the mediolateral "gap" between the center of pressure and the center of gravity at the time of swing foot off, the disequilibrium torque at the onset of the execution phase is reduced so the subsequent mediolateral shift of the center of gravity is attenuated. Thus, although mediolateral APA could also serve other functions (e.g., unloading of the swing leg), they appear to be crucial in minimizing postural instability during the execution phase of gait initiation.

ADAPTABILITY OF APA ASSOCIATED WITH VOLUNTARY MOVEMENT

In many daily situations, factors of a different nature may constrain the development of APA and thus affect balance and/or the focal movement performance. This paragraph reports the results of recent studies that questioned the adaptability of APA to various constraints imposed on the postural system. Those postural constraints envisaged here can be classified into biomechanical (postural stability, superimposition of tasks), (neuro) physiological (fatigue), temporal (time pressure) and psychological (fear of falling, emotion).

APA and postural stability

The question of the focal movement performance and the associated APA scale with changes in postural stability has been thoroughly investigated in the literature. As stressed by authors, postural stability depends on both strictly mechanical factors (e.g., the initial height of the center of gravity, the size of the base of support, the presence of an additive support) and on strictly neurophysiological factors, i.e., on the capacity of the postural muscle system to efficiently counter an external or internal perturbation[2,33]. When the posture is mechanically highly stable, e.g., when standing upright with an additive thoracic support[14], when leaning against a wall[15] or when sitting or lying[34], APA have been reported to be attenuated, very likely because they are then not crucial to maintain posture and balance during task execution. When the posture is mechanically highly unstable, e.g., when the base of support size is drastically reduced in the direction of the perturbation applied to posture, attenuation of APA has also been reported. This attenuation has been thought to reflect a protective strategy directed to minimize the potential destabilizing effect by APA themselves[35-37].

Results of recent studies illustrate the relationship between base of support size, APA and focal movement performance. For example, in the study of Yiou *et al*[37], subjects performed a series of forward arm pointing tasks at maximal velocity, from five postures that differed by the anteroposterior distance between the heels (Figure 1). This distance was decreased stepwise from 40 cm (P40 condition) to 0 cm (P0 condition with feet in contact). Kinetics data were collected with a large force plate and kinematics data of the pointing was collected with a bi-axial accelerometer fixed at the wrist (Figure 1). Results showed that the amplitude (maximal backward center of pressure displacement) and the efficiency of the APA (anticipatory forward center of gravity velocity), as well as the pointing performance (maximal hand velocity towards the target), all statistically decreased from P40 to P0. In contrast, the duration of APA did not change with the base of support size. These results provided support to the PKC theory, according to which the performance of the focal component of a motor task tightly depends on the capacity of the postural component to develop efficient anticipatory dynamics[2,8,9]. This capacity is hindered when the base of support size is reduced in the direction of the perturbation.

In the continuity of these results, Yiou *et al*[33] questioned how young healthy subjects control their balance in situation of instability specifically elicited by a reduced capacity of force production in the postural muscle system. Subjects displaced a horizontal bar forwards with both hands at a maximal velocity towards a target while standing on one or both legs. It is noteworthy that, in terms of mechanics, the postural stability along the anteroposterior direction is roughly equivalent in these two conditions since the base of support length remained unchanged. In contrast, the postural stability along the mediolateral direction was lower in the unipedal compared to the bipedal stance since the base of support width was reduced. The focal movement was expected to induce very low mediolateral perturbation as it was symmetrical with respect to the sagittal plane. Thus, it was assumed that any change in APA parameters or in focal movement performance in the unipedal stance predominantly reflected adaptation to the reduced postural muscle system efficiency, rather than adaptation to the reduced mediolateral base of support size. The main results showed that, along the anteroposterior axis, APA were twice as long in the unipedal stance than in the bipedal stance, while the anticipatory inertia forces directed to offset the forthcoming perturbation remained equivalent. The focal movement performance was maintained without any additive postural perturbation during task execution. These results showed that young healthy subjects do not use a "protective strategy of APA attenuation"[35,36] when exposed to a situation of instability specifically elicited by reduced postural muscle system efficiency. Instead, when confronted with such a situation herein experimentally elicited by having subjects standing on one leg, they lengthen APA duration in order to reach an as efficient anticipatory postural counter perturbation as under the more stable bipedal posture (in terms of whole body anticipatory inertia forces). As a consequence, they could maintain an equivalent focal movement performance (maximal velocity) without any additive postural perturbation. This postural adaptation was possible here because the possibility of center of pressure shift in the direction of the perturbation remained unchanged across conditions.

Overall, these studies suggest that when APA cannot be adapted to the perturbation induced by the voluntary movement, the perturbation, and hence the focal movement performance, is reduced. When APA can adapt to the perturbation, the focal movement performance remains optimal.

APA and fatigue

The question of how the central nervous system adapts APA in order to take into account the internal pertur-

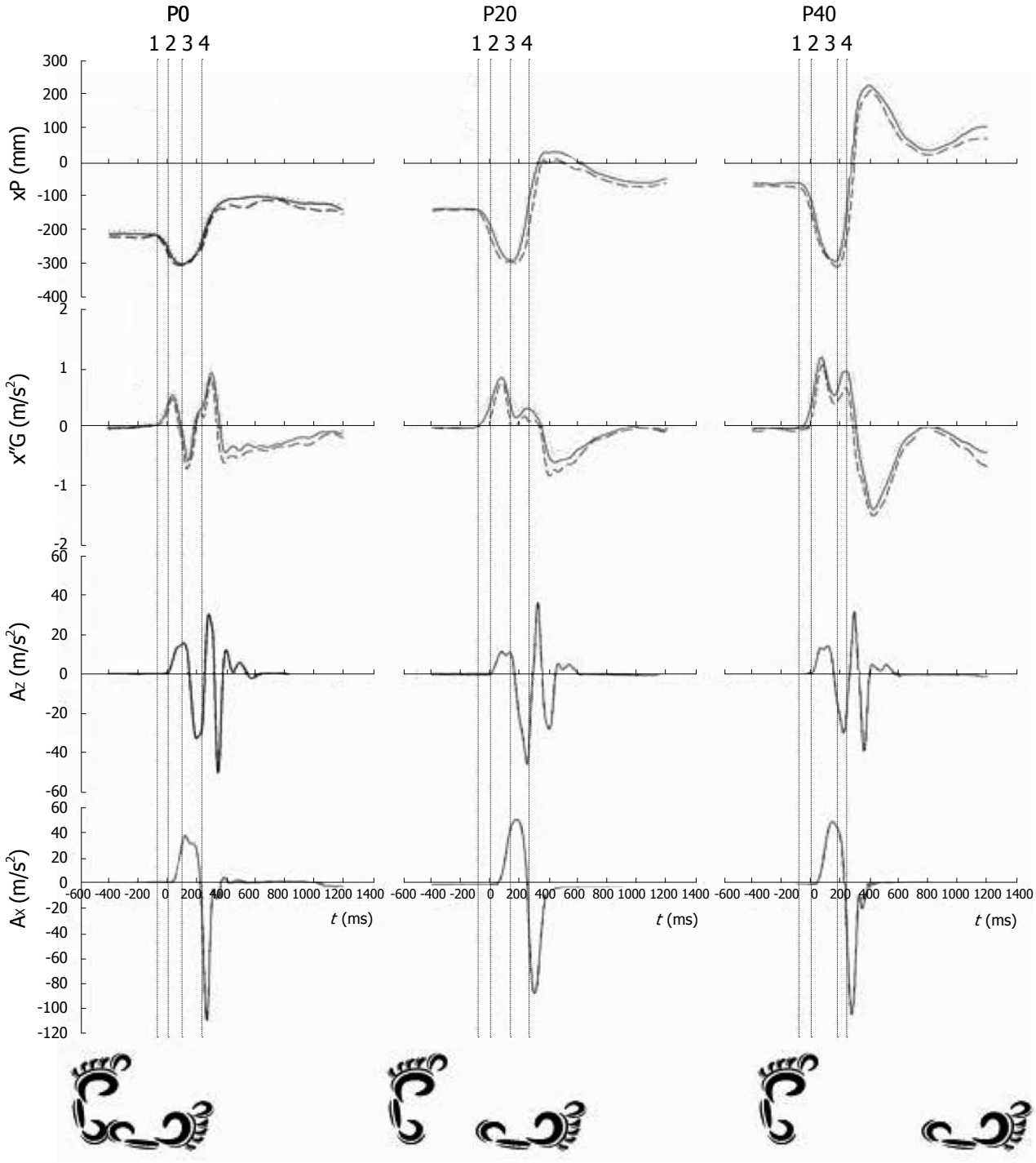

Figure 1 Example of biomechanical profiles associated with forward arm pointing in the P0 (heels in contact), P20 (20 cm between the heels) and P40 conditions (40 cm between the heels), and feet position (below panels). Mean traces (± 1 STD) are presented in one representative subject (n = 7 trials) pointing the arm to the right of the figure. xP, x''G, Az, Ax: Anteroposterior center of pressure displacement, anteroposterior center of gravity acceleration, vertical and anteroposterior hand acceleration, respectively. Line 1: Onset of biomechanical traces/onset of anticipatory postural adjustments (APA); Line 2: Onset of voluntary hand acceleration; Line 3: Peak of maximal center of pressure displacement/end of APA; Line 4: Target hit. An upward variation indicates a forward or an upward oriented displacement or acceleration. The time scales reported at the bottom of the figure are the same for all panels. The y-scales reported on the left side are the same for the panels in the P0, P20 and P40 conditions. (From Yiou *et al*[37]) .

bation induced by postural muscle fatigue (hereafter referred to as "fatigue") has been addressed in several recent studies[38-42]. In these studies, subjects performed a series of rapid discrete motor tasks involving one or both arms from a bipedal stance, before and after a fatiguing procedure designed to obtain fatigue in the postural musculature [generally in the upper leg (hamstrings) and/or the trunk muscles]. The fatiguing procedure typically consisted of a series of low to moderate level isometric contractions i.e., isometric contractions ranging from 7% of maximal voluntary isometric contraction (MIC) to 50% MIC, that were performed until

exhaustion. As an alternative procedure, exhausting aerobic exercise was used by Strang *et al*[43] to fatigue the whole postural musculature. These studies repeatedly reported that, under such experimental conditions, the level of muscle excitation was drastically decreased in the fatigued state compared to the normal state and that APA onset occurred earlier, which was responsible for a longer APA duration. Because the amplitude of the motor outcome (as measured with the anticipatory displacement of the center of pressure) remained unchanged with fatigue[38,40], this longer APA duration was thought to reflect adaptive change to the reduced capability of force production in the postural muscle system[38-43].

In contrast with these latter studies, Yiou *et al*[26] reported that, following fatigue of the dorsal part of leg muscles induced by series of high level isometric contractions (60% MIC), neither the level of excitation of the fatigued muscles (hamstrings), APA onset, postural joint kinematics nor anticipatory center of pressure displacement changed. In this study, the motor task was to displace forwards a 2 kg bar (grasp bar) with both arms at a maximal velocity towards a target from a bipedal stance [bilateral forward reach (BFR)]. To explain this discrepancy with the literature, it was proposed that the effects of fatigue on APA might be dependent on the adequacy between the motor units fatigued during the fatiguing procedure and those motor units recruited during the APA. This interpretation was based on the well-known "size principle"[44], according to which slow and fatigue-resistant motor units [i.e., motor units of type I (MU Ⅰ)] are recruited before fast, more fatigable motor units [i.e., motor units of type Ⅱ (MU Ⅱ)]. In this hypothesis, those motor units that were fatigued by the high level isometric contractions (MU Ⅱ) were not those predominantly recruited during the APA (MU Ⅰ) because the force level required in the postural musculature to counterbalance the destabilizing effect of the BFR was too weak. Consequently, no adaptive EMG change of APA was required following the fatiguing procedure to ensure an equivalent motor outcome.

The results of this latter study further raised the question whether and how the central nervous system adapts the anticipatory EMG activity in the fatigued postural muscles if the force level required in these muscles to counterbalance the destabilizing effect of the BFR was elevated[45]. In such a condition, the fatigued muscular fibers might then indeed partly match with those used during the APA. Consequently, adaptive EMG changes in the fatigued postural muscles would then be required unless biomechanical features of APA (peaks of anticipatory center of pressure displacement/anticipatory center of gravity acceleration) would be altered. In order to increase the force level required in the postural musculature (in particular in the hamstrings) to counterbalance the destabilizing effect of the BFR, the subjects stood upright on one single leg (unipedal stance) rather than on both legs. The mass of the grasp bar to be displaced forward was also purposely doubled compared to the study described above (4 kg *vs* 2 kg in Yiou *et al*[26]). Subjects performed a series of bilateral forward reach tasks (BFR) under a unipedal stance (dominant and non dominant) before ["no fatigue" condition (NF)] and after ["fatigue" condition (F)] a procedure designed to obtain major fatigue in hamstrings. Center of gravity acceleration, center of pressure displacement and electrical activity of trunk and leg muscles were recorded and quantified within a time window typical of APA. The main results showed that there was no significant effect of fatigue on the level of muscle excitation and APA onset in any of the postural muscles recorded. Similarly, no change in APA onset could be detected from the biomechanical traces. In contrast, the results showed that the peaks of anticipatory center of pressure displacement and anticipatory center of gravity acceleration were much lower in F than in NF. These results suggest that, following the fatiguing series of high level isometric contractions, the capacity of the central nervous system to adapt APA to the reduced muscular efficiency might be altered. Subjects might then possibly more strongly rely on postural adjustments occurring during and/or following the voluntary movement to maintain postural stability. These results markedly contrast with the previous studies reporting adaptive anticipatory changes in the electrical activity of the fatigued postural (and in particular an earlier APA onset), along with an equivalent motor outcome[38,40]. This discrepancy with the literature might possibly be ascribed to the different intensity of isometric contractions used to elicit fatigue in the postural muscle system, with low to medium level isometric contractions generating lower perturbation of the proprioceptive information sources used to control posture and balance than high level isometric contractions. This effect might be exacerbated when subjects stood on one single leg.

The generality of these latter results obtained with upper limb tasks from a quiet standing posture was recently tested with another experimental model of posture and movement coordination, stepping initiation[22]. Specifically, the question was how the central nervous system organizes the APA associated with stepping initiation under acute fatigue of ankle dorsiflexors (tibialis anterior, primum movens of anticipatory backward center of pressure shift during APA; see Figure 2 for examples of biomechanical and electromyographical traces). The subjects performed a series of stepping initiation at spontaneous velocity before NF and after F, a protocol designed to induce tibialis anterior fatigue on both sides. The main results showed that the level of tibialis anterior activation during the APA, the amplitude of anticipatory dynamical phenomena and the peak of center of gravity velocity (motor performance) decreased with fatigue, while the duration of the APA and the execution phase increased. Subjects were, however, able to increase the level of tibialis anterior activation and thus reach a greater center of gravity velocity when the instruction was to step faster. It

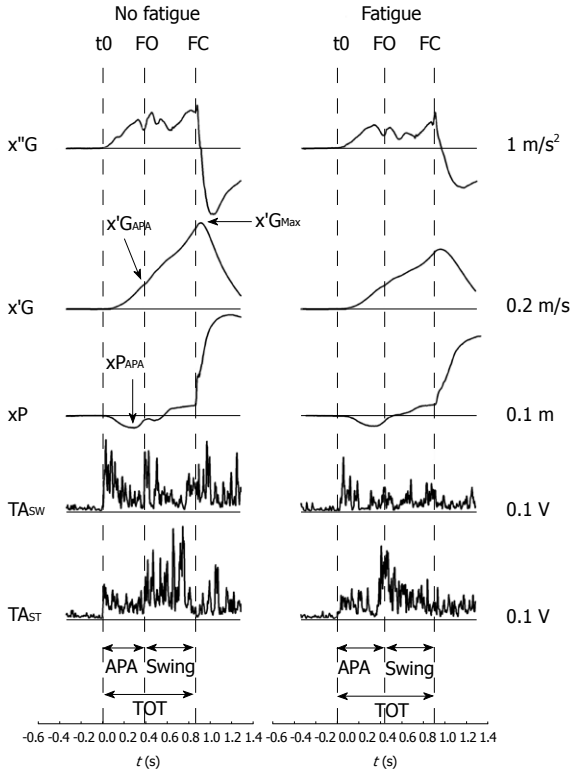

Figure 2 Example of biomechanical and electromyographical profiles of stepping initiation in the fatigue and no fatigue condition (one representative participant at spontaneous speed). x"G, x'G, xP: Anteroposterior center of gravity acceleration, center of gravity velocity and center of pressure displacement, respectively; t0, FO, FC: Onset rise of x"G trace, swing foot off, swing foot contact, respectively; TA: Rectified electrical activity of tibialis anterior; SW, ST: Swing and stance leg, respectively; APA, SW, TOT: Anticipatory postural adjustments, swing phase and total stepping initiation time windows, respectively; X'GMax, X'GAPA, xPAPA: Peak of center of gravity velocity, center of gravity velocity at foot off, peak of backward center of pressure displacement, respectively. Positive variation of the biomechanical traces indicates forward displacement, velocity or acceleration. Negative variation of these traces indicates backward displacement, velocity or acceleration (from Yiou *et al*[22]).

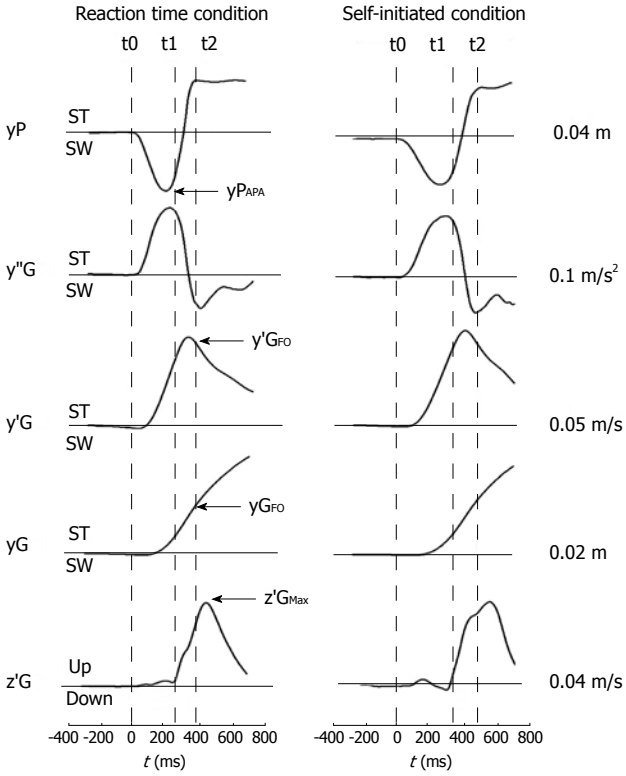

Figure 3 Example of the biomechanical traces of leg flexion in the reaction time and self-initiated conditions (one trial in one representative participant). The figure presents the main experimental variables: yP, y"G, y'G, yG, z'G, medio-lateral (ML) center of pressure (CoP) displacement, ML center of gravity (CoG) acceleration, ML CoG velocity, ML CoG displacement and vertical CoG velocity. The markers t0, t1, t2 represent respectively the onset variation of the y"G trace from the baseline, swing heel off and swing foot off. SW and ST indicate the swing and stance leg side, respectively. Finally, yPAPA, y'GFO, yGFO, z'GMax are the peak of ML CoP displacement during APA, ML CoG velocity/displacement at the foot off time, peak of vertical CoG velocity (motor performance), respectively (from Yiou *et al*[52]).

was proposed that the changes in stepping initiation parameters with fatigue might reflect the existence of protective strategy directed to preserve the fatigued muscles, rather than muscle weakness associated with fatigue. In other words, priority was given to body protection rather than to motor performance maintenance.

Overall, these studies suggest that the adaptability of APA to fatigue depends upon the motor task to be performed and upon the way in which fatigue is induced.

APA and temporal constraints

Voluntary movements can be performed in at least two conditions of temporal constraint: a reaction time condition in response to an external signal and a self-triggered condition in which movement initiation is self-initiated[46,47]. A literature review showed that the duration of APA in various motor tasks, such as upper limb task[48,49] and gait initiation[50,51], was shortened in a reaction time condition compared to a self-triggered condition. A recent study by Yiou *et al*[52] tested the hypothesis that, during rapid leg flexion, this shortening of APA could be compensated by a larger APA amplitude so that the dynamic stability reached at the foot off time (i.e., at the end of APA) remains the same as in the self-triggered condition. Young healthy participants performed series of leg flexions (1) as soon as possible in response to an acoustic signal (reaction time condition; condition with temporal pressure); and (2) in a self-triggered condition (no temporal pressure). In this study, the focus was on the anticipatory postural control of mediolateral stability, i.e., mediolateral dynamics was mainly considered (Figure 3). Results showed that the swing foot off was triggered sooner in the reaction time condition compared to the self-triggered condition. As a consequence, the APA duration was shorter in the former condition; this shortening was compensated by an increase in the medio-lateral anticipatory center of pressure displacement so that the dynamic stability reached at the foot off time remained unchanged. Overall, these results showed that when a complex task is performed under temporal pressure, the central nervous system is able to adapt the spatio-temporal features of APA in a way to both hasten the initiation of the voluntary movement and maintain optimal conditions of dynamic

stability. It therefore seems that the central nervous system does not "trade off optimal stability for speed of movement initiation under reaction time condition", as it had been proposed in the literature[53]. Now, these results must be taken with caution since it remains to be verified whether a more challenging task (e.g., with higher precision requirement on the leg movement, as in Bertucco *et al*[54]), would affect motor performance and/or dynamic stability in the condition with temporal pressure.

APA and superimposition of motor tasks

In many daily situations, humans have to perform goal-directed arm movements in multiple possible directions while voluntarily displacing the whole body, i.e., they have to superimpose "elementary" motor tasks. As stated above, each of these elementary motor tasks necessitates the development of specific APA, in terms of duration, amplitude and direction. In a series of recent papers[55-59], the question was asked how the central nervous system organizes the APA associated with each of these elementary tasks when they are superimposed in a motor sequence. The experimental model used in these studies was typically composed of two elementary tasks that both required APA. These tasks were either performed in isolation or superimposed in a sequence. For example, in Yiou *et al*[60], the three following experimental conditions were carried out: (1) "Isolated stepping" (the stepping initiation was performed alone); (2) "Isolated pointing" (the pointing was performed alone); and (3) "Experimental sequence" (the pointing and stepping were combined). In this study, the question was whether the APA associated with the coordination of arm pointing and stepping initiation resulted from the sole juxtaposition of APA associated with each isolated elementary tasks ("juxtaposition hypothesis"). The postural dynamics of a "theoretical sequence" was purposely calculated based on the linear summation of the acceleration of the center of gravity and the displacement of the center of pressure traces recorded in conditions (1) and (2) (elementary tasks performed in isolation). The "juxtaposition hypothesis" was tested by between condition comparisons of postural dynamics computed during APA for stepping (stepping APA). The main results showed that: (1) The amplitude of the stepping APA was higher in the "experimental sequence" than in the "theoretical sequence"; (2) The stepping APA amplitude was higher in the "isolated stepping" than in the "theoretical sequence" while, in contrast, the model of the "juxtaposition" predicted that it should be lower; and (3) The stepping APA amplitude was higher in the "experimental sequence" than in the "isolated stepping" condition. These results showed that the arm movement facilitated the stepping initiation rather than hindering it, as predicted from the "juxtaposition hypothesis". It was therefore proposed that the APA generated during the experimental sequence did not result from a simple juxtaposition of isolated elementary tasks. Rather, it was proposed that the central nervous system was able to adapt the APA associated with the arm movement to fit with the biomechanical constraints associated with the stepping initiation.

Conversely, the question was asked in Yiou and Do' s study whether the central nervous system was able to adapt the APA associated with stepping initiation to take into account the postural perturbation induced by voluntary arm movement. In this study, the focus was on the anticipatory postural control of mediolateral stability during the stepping initiation. Subjects purposely initiate stepping in isolation ("isolated stepping") or in combination with a lateral arm raising ("motor sequence"). Stepping initiation was carried out with the leg ipsilateral or controlateral to raising arm (ipsilateral and controlateral sequence, respectively) (Figure 4). The arm movement was expected to accentuate the lateral fall of the center of gravity towards the swing leg in the ipsilateral sequence (destabilizing effect) while it is expected to attenuate its fall in the controlateral sequence (stabilizing effect).

The main results showed that the amplitude of APA along the mediolateral direction increased from "ipsilateral isolated stepping" to "ipsilateral sequence", but did not change in conditions involving the controlateral leg. In addition, the mediolateral instability increased from "ipsilateral isolated stepping" to "ipsilateral sequence", but decreased from "controlateral isolated stepping" to "controlateral sequence". These changes were exacerbated when inertia was added at the hand during raising (Figure 5). The results of this study showed that markedly different strategies are used to control mediolateral stability in the "ipsilateral sequence" and the "controlateral sequence". In the "ipsilateral sequence", the central nervous system increased the amplitude of APA for stepping to counter the destabilizing effect induced by the forthcoming arm raising. This strategy of up-regulation was, however, not sufficient to completely offset this additive destabilizing effect. Part of this additive destabilizing effect was therefore probably taken charge of during the CPA. In the "controlateral sequence", the APA amplitude did not change and even tended to decrease as the inertia became higher and instability was lower. In this case, the central nervous system may then simply take advantage of the postural dynamics induced by arm raising to facilitate the process of postural stabilization during CPA. Altogether, these results support the hypothesis that, in young healthy subjects, the central nervous system scales the amplitude of APA for stepping as a function of the biomechanical consequences of forthcoming arm raising on postural stability.

Overall, these studies suggest that during sequential tasks involving whole body displacement, the central nervous system is able to adapt the APA associated with each elementary task to facilitate both whole body progression and postural stability.

APA and psychological factors

Fear of falling: In addition to biomechanical factors such as those reported above, recent studies have shown that psychological factors, such as fear of falling (FoF)

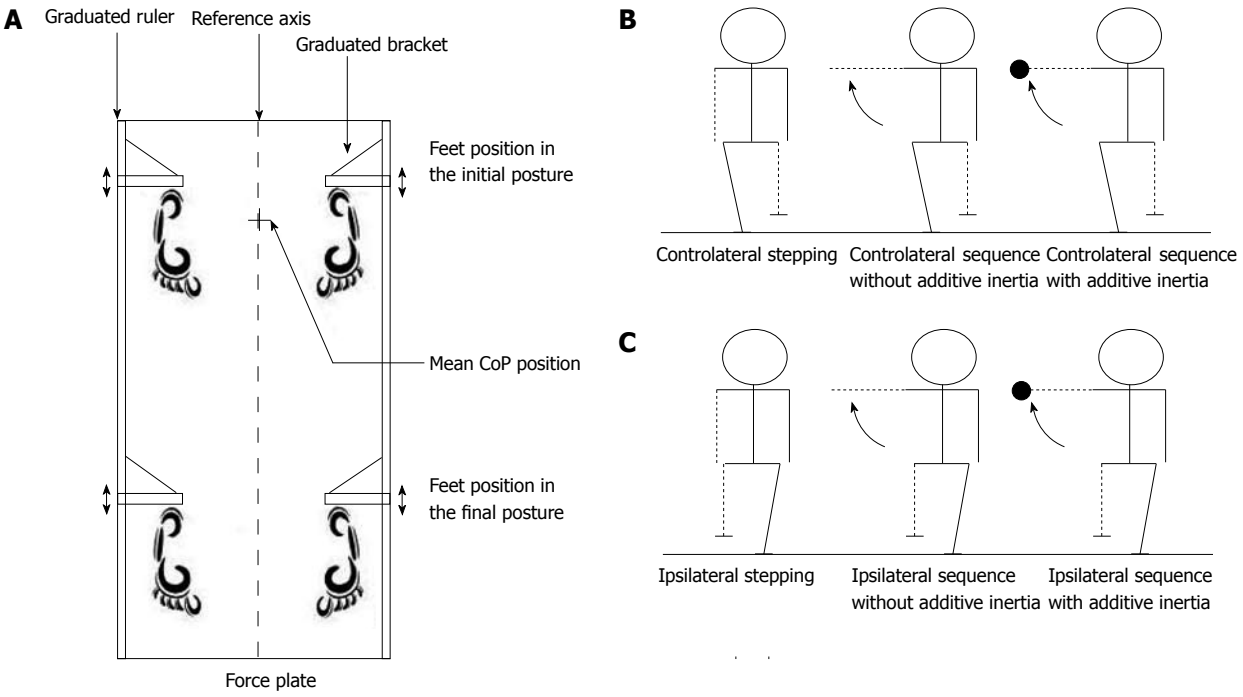

Figure 4 Example of experimental tasks and setup to investigate the organization of anticipatory postural adjustments in a motor sequence. A: Experimental setup. The center of pressure and center of gravity displacement are determined relative to the reference line passing through the center of pressure position in the initial posture; B: Experimental tasks involving the controlateral leg for stepping; C: Experimental tasks involving the ipsilateral leg for stepping. Dotted lines represent the body segments involved in raising and stepping (from Yiou *et al*[32]). CoP: Center of pressure.

and related concepts such as low balance confidence, may induce adaptive changes in the way postural equilibrium is controlled[61-63]. For example, we examined in a recent study[64] how FoF may influence the anticipatory postural control of mediolateral stability during rapid leg flexion. Young healthy participants performed a series of leg flexions at maximal velocity from low and high surface heights (6 cm and 66 cm above ground, respectively). In the latter condition with increased FoF, the stance foot was placed at the lateral edge of the support surface to induce a maximal postural threat. Results showed that the peak of mediolateral center of gravity velocity reached during APA decreased with FoF; this decrease was compensated by an increase in APA duration (which resulted in a longer reaction time) so that the center of gravity position at the time of swing foot off was located further towards the stance leg side. With these changes in APA, the center of gravity was propelled in the same final (unipodal) position above the stance foot as in the condition with low FoF, i.e., it reached an as stable position. The focal movement performance also remained unchanged. Based on these results, it was suggested that, in the condition with increased FoF, the central nervous system was able to compensate for the lower initial center of gravity velocity by an increase in the APA duration so that the center of gravity could be propelled further towards the stance leg side at the foot off time. With this strategy, the center of gravity was brought nearer to its position of equilibrium at the time when the voluntary leg raising was initiated. It thus seems that the central nervous system more carefully ensured that the conditions of stability could effectively be reached in the final posture before triggering the voluntary movement. It was thus proposed that the changes in APA with FoF reflected an adaptive strategy directed to simultaneously avoid lateral fall towards the edge of the support surface and to maintain stability in the final unipodal posture.

Emotion: Darwin already proposed a relationship between emotional states and postural changes in man and animals[65]. Nowadays, there is growing evidence from psychology and neurophysiology that human motor control centers and emotion centers are largely intertwined and reciprocally interrelated[66]. The so-called "motivational direction hypothesis"[67] is founded on Darwin's theory of adaptation of species to their environment. This hypothesis proposes that unpleasant emotions activate defensive circuitry and prime avoidance behaviors, whereas pleasant emotions activate appetitive circuitry that prime approach behaviors. This theory has initially been supported by experiments that manipulate emotional states prior to or during the execution of upper extremity movements that are made towards or away from the body, e.g., wrist flexion or extension[68]. Only very recently, studies investigated the influence of the emotional state on APA associated with complex task involving whole body displacement such as gait initiation[69-71]. In these studies, emotional state was elicited by the presentation of unpleasant (e.g., mutilation) or

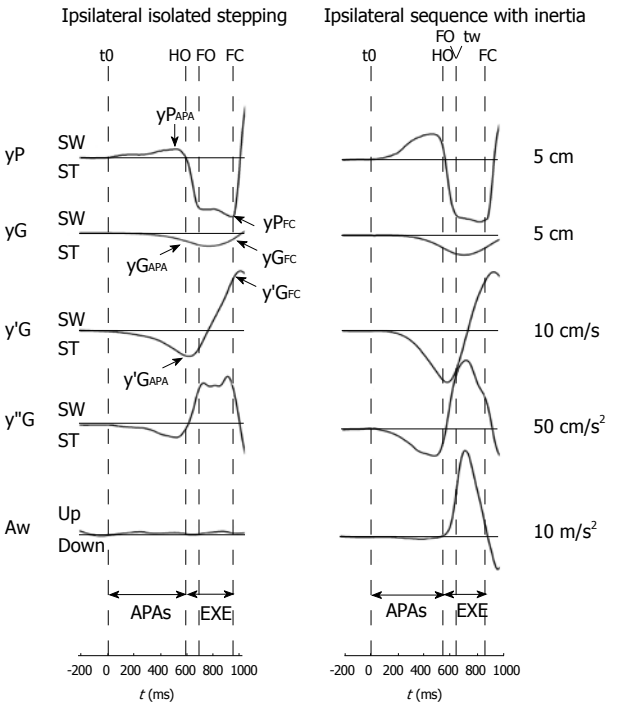

Figure 5 Example of biomechanical traces in "ipsilateral isolated stepping" and "ipsilateral stepping + arm raising sequence" with additional inertia (one trial in one representative subject). yP, yG, y''G, y'G, Aw: Mediolateral (ML) center of pressure and center of gravity displacement, ML center of gravity acceleration, ML center of gravity velocity and tangential wrist acceleration, respectively; t0, HO, FO, FC, tw: Onset variation of the yP trace from the reference line, swing heel off, swing foot off, swing foot contact and onset of raising, respectively; SW, ST: Swing and stance leg side, respectively; APAs, EXE: Anticipatory postural adjustments and stepping execution phase, respectively. Main postural variables were reported only in the traces of the "ipsilateral isolated stepping"; yPAPA, yGAPA, y'GAPA: Maximal center of pressure displacement, center of gravity displacement and velocity during APAs; yPFC, yGFC, y'GFC: Center of pressure displacement, center of gravity displacement and velocity at time of swing foot contact. Positive variation of the traces indicates displacement, velocity or acceleration towards the swing leg side. Negative variation of the traces indicates displacement, velocity or acceleration towards the stance leg side (from Yiou *et al*[32]).

pleasant (e.g., erotic, baby faces) pictures. In brief, these results showed that pleasant images induced an increase in APA amplitude and therefore facilitated forward gait initiation, whereas unpleasant images induced a decrease in APA amplitude and therefore hindered gait initiation. This latter result was interpreted as reflecting a defensive response, probably associated with freezing like behavior. The results of these studies are thought to be in agreement with the "motivational direction hypothesis" and would reflect adaptive changes of APA to environmental stimuli.

Overall, these studies suggest that changes of APA with FoF or emotion might reflect adaptive behavior to the subject's environment.

CONCLUSION

This paper first addressed the question of how balance is controlled during voluntary movement, with a focus on APA, considered as a major "line of defence" against self-inflicted postural perturbations. Results of recent studies that focused on the adaptability of APA to various sources of constraints imposed to the postural system (biomechanical, temporal and psychological) were then reviewed. Overall, these results point out the capacity of the central nervous system to adapt the APA parameters to each of these constraints, but in specific way. Hence, depending on the constraints, it seems that the "priority" of the central nervous system was focused on postural stability maintenance, on body protection and/or on maintenance of focal movement performance. Knowing how balance is controlled during voluntary movement and how this control adapts to postural constraints may be beneficial for the clinicians to better understand the etiology of falls in populations with postural impairments (e.g., the elderly or persons with Parkinson disease) and to better individualize rehabilitation programs.

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ORIGINAL ARTICLE

Assessment of scapulohumeral rhythm for scapular plane shoulder elevation using a modified digital inclinometer

Jason S Scibek, Christopher R Carcia

Jason S Scibek, Department of Athletic Training, School of Health Sciences, Duquesne University, John G Rangos, Sr., Pittsburgh, PA 15282, United States
Christopher R Carcia, Department of Physical Therapy, School of Health Sciences, Duquesne University, John G Rangos, Sr., Pittsburgh, PA 15282, United States
Author contributions: Scibek JS and Carcia CR were responsible for conceptualizing the study, developing and designing the methodology, collecting and analyzing the data, and contributing to the development, writing and editing of the manuscript.
Supported by Pennsylvania Athletic Trainers' Society Research Grant, No. G0900028
Correspondence to: Jason S Scibek, PhD, ATC, Associate Professor, Duquesne University, 600 Forbes Avenue, 118 HLSB, Pittsburgh, PA 15282, United States. scibekj@duq.edu
Telephone: +1-412-3965960 Fax: +1-412-3964160
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Abstract

AIM: To develop a better understanding of scapulohumeral rhythm during scapular plane shoulder elevation.

METHODS: Thirteen healthy, college-aged subjects participated in this study. Subjects were free from any upper extremity, neck or back pathology. A modified digital inclinometer was utilized to measure scapular upward rotation of the subject's dominant shoulder. Upward rotation was measured statically as subjects performed clinically relevant amounts of shoulder elevation in the scapular plane. Testing order was randomized by arm position. Scapular upward rotation was assessed over the entire arc of motion and over a series of increments. The percent contributions to shoulder elevation for the scapula and glenohumeral joint were calculated. Scapulohumeral rhythm was assessed and represented the ratio of glenohumeral motion to scapulothoracic motion (glenohumeral elevation: scapular upward rotation). A one-way ANOVA was used to compare scapular upward rotation between elevation increments.

RESULTS: Scapulohumeral rhythm for the entire arc of shoulder elevation was equal to a ratio of 2.34 :1 and ranged from 40.01:1 to 0.90:1 when assessed across the different increments of humeral elevation. Total scapular motion increased over the arc of shoulder elevation. The scapula contributed 2.53% of total motion for the first 30 degrees of shoulder elevation, between 20.87% and 37.53% for 30°-90° of shoulder elevation, and 52.73% for 90°-120° of shoulder elevation. Statistically significant differences in scapular upward rotation were identified across the shoulder elevation increments [$F_{(3,48)} = 12.63$, $P = 0.0001$].

CONCLUSION: Clinically, we must recognize the usefulness of the inclinometer in documenting the variable nature of scapulohumeral rhythm in healthy and injured shoulders.

Key words: Biomechanics; Kinematics; Scapula; Shoulder

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INTRODUCTION

Optimal function of the shoulder is reliant on the co-

ordinated movement of the scapula and the humerus[1]. Alterations in scapular position and control afforded by the scapula stabilizing muscles are believed to disrupt stability and function of the glenohumeral joint[1-3], thereby contributing to shoulder impingement, rotator cuff pathology and shoulder instability[4]. Given the role of the scapula in shoulder function, the ability to monitor the coordinated motion of the scapula and humerus, or scapulohumeral rhythm[5,6], may have clinical implications when dealing with overhead athletes and patients with shoulder pathologies.

Inman, Saunders and Abbott[7] were the first to measure scapulohumeral rhythm using radiography and suggested what became the widely accepted 2:1 ratio between glenohumeral elevation and scapulothoracic upward rotation (SUR). Since then imaging modalities (X-ray and magnetic resonance imaging)[8], cinematography[9], goniometry[10-12], and more recently 3-dimensional tracking systems[13-16] have been used to gain a better appreciation of shoulder kinematics. This evolution in kinematic assessment has resulted in new understandings of scapulohumeral rhythm in both healthy and injured populations. Some of the literature suggests the 2:1 ratio is not consistent across an entire arc of shoulder elevation[8-10,17,18] and that variability in this ratio may increase when considering the scapulohumeral rhythm exhibited by shoulder injured subjects[19,20].

Instrumentation accessibility has often precluded clinicians from being able to quantify glenohumeral and scapulothoracic joint contributions to scapulohumeral rhythm. Whether we consider exposure to electromagnetic radiation associated with radiography or the expense and time intensive nature of electromagnetic tracking systems, there are some limitations associated with quantifying the relative contributions of the glenohumeral and scapulothoracic joints to shoulder elevation in clinical settings. To enhance accessibility, Johnson *et al*[12] validated use of a digital inclinometer to quantify SUR, which has since been incorporated into a variety of clinically oriented research studies[21-27]. While many studies involving digital inclinometers have been successful in quantifying SUR, no studies involving digital inclinometers have attempted to examine scapulohumeral rhythm specifically. Although the aforementioned studies provide valuable information relative to scapulothoracic joint motion, return to activity following shoulder injury is dependent on function of both the scapulothoracic and the glenohumeral joint, and how both joints function together. The ability to utilize the inclinometer for quantifying scapulohumeral rhythm will provide clinicians with a means to monitor the often variable nature of scapulohumeral rhythm while also comparing the functional capacity of the shoulder to the established 2:1 scapulohumeral movement ratio. Therefore, the purpose of this study was to develop a better understanding of the coordinated movement of the scapula and humerus in order to enhance the evaluation and rehabilitation efforts of clinicians. We hypothesized that the coordinated movement of the scapula and humerus as measured by a digital inclinometer, would be different than the widely accepted 2:1 scapulohumeral rhythm ratio (glenohumeral: scapulothoracic motion) initially described by Inman[7]. Furthermore, we hypothesized that scapular contributions to incremental increases in glenohumeral elevation would not be consistent.

MATERIALS AND METHODS

Subjects

Thirteen healthy subjects (21.46 ± 1.13 years, 1.76 ± 0.11 meters, 76.18 ± 12.57 kg; 8 male, 5 female; 11 right, 2 left) were recruited to participate in this study. All subjects completed an informed consent document approved by the University's Institutional Review Board. Participants provided demographic data and completed a health history questionnaire. Subjects also underwent a brief physical examination, which involved measures of shoulder range of motion, strength normalized to body weight, and a subset of evaluative special tests to ensure a healthy dominant shoulder free of any current or previous upper extremity, neck or back injuries or conditions that would result in muscle weakness or reduced shoulder range of motion. The dominant shoulder was selected based upon which hand subjects chose to throw a ball with and sign their name.

Instrumentation

A digital inclinometer (Pro 360, Baseline®, Fabrication Enterprises, White Plains, NY) was used to assess scapular upward rotation during static humeral elevation trials. Using an electromagnetic tracking system, Johnson *et al*[12] validated use of the digital inclinometer to quantify SUR associated with varying amounts of humeral elevation (r = 0.66 to 0.89). A series of modifications were made to the inclinometer, consistent with the work of Johnson et al, to make it suitable for measuring SUR. Specially designed wooden locator rods were used with the inclinometer in order to appropriately align the inclinometer with the contours of the scapular spine. A custom made plate (Lexan, SABIC Innovative Plastics, Pittsfield, MA) was attached to the inclinometer, allowing for adjustable spacing of the rods (Figure 1A and B). Each locator rod was attached to the Lexan plate using hardware. A bubble level was also affixed to the inclinometer to maintain its position perpendicular orientation relative to the horizontal plane of the inclinometer[12]. With the modifications the inclinometer was aligned with the root of the spine of the scapula and the posterolateral corner of the acromion process[12].

A hand held dynamometer (ergoFET 300, Hogan Health Industries©, West Jordon, UT) was used to assess maximal strength during the initial screening process. Maximal strength tests for shoulder flexion, abduction, and internal and external rotation were performed according to the manual muscle testing guidelines presented by Hislop and Montgomery[28]. When applying the

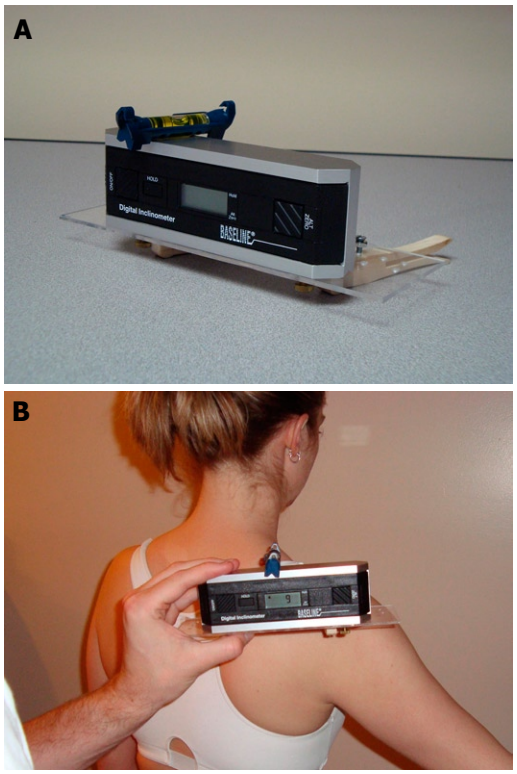

Figure 1 Modified digital inclinometer (A) and scapular upward rotation measurement (B).

manual resistance for each test the hand held dynamometer was used in order to obtain force measures. Two trials were performed for each shoulder motion; means were calculated and were normalized to body weight.

Intervention

Subjects were seated on a plastic stool one arm's length from a stationary screen. Subjects were seated to minimize compensatory changes of the lower extremity and trunk, which could impact shoulder biomechanics. Subjects were asked to move their hands to selected points along the screen that corresponded to specific shoulder ranges of motion (rest, 30°, 45°, 60°, 75°, 90°, 120°) in the scapular plane. Shoulder ranges of motion were monitored with a digital inclinometer and hand placement was marked on the screen for each of the respective shoulder ranges of motion. The reliability and validity of our humeral elevation protocol was established previously in our laboratory using an electromagnetic tracking system. When performing humeral elevation in the scapular plane, our intra-session reliability ranged from $ICC_{(3,1)}$ = 0.935 to $ICC_{(3,1)}$ = 0.947, while scapular kinematic reliability ranged from $ICC_{(3,1)}$ = 0.964 to $ICC_{(3,1)}$ = 0.999. Similarly, we were able to report significant correlations between the electromagnetic tracking system and the digital inclinometer for shoulder elevation in the scapular plane with Pearson correlation coefficients ranging from 0.846 to 0.986. In addition to having used the inclinometer as part of a previous study to measure scapular upward rotation, intra-rater reliability for the primary investigator has been determined to be excellent when used to quantify scapular rotation [$ICC_{(3,1)}$ = 0.951 to $ICC_{(3,1)}$ = 0.996] with the inclinometer.

Testing order was randomized by arm position for each subject. Subjects began with their dominant hand at his/her side and were then asked to move their hand to a selected position and to hold that position while SUR was measured. Each trial lasted 10-15 s, and subjects were provided with 5-10 s rest in between each trial. For all inclinometer measures, the primary investigator relied on palpation to identify the position of the medial border and spine of the scapula and the posterolateral corner of the acromion. The primary investigator was responsible for all measures taken with the inclinometer. The inclinometer was repositioned each trial, and inclinometer values were confirmed and recorded by a laboratory assistant for each trial to eliminate investigator bias.

Table 1 Humeral elevation increments, mean scapular upward rotation measures, and scapulohumeral rhythm ratios

Shoulder elevation increments	Mean scapular upward rotation (± SD)	Scapulohumeral rhythm
0°-120°	35.95 (6.50)	2.34:1
0°-30°	0.73 (4.91)	40.05:1
30°-60°	10.99 (7.18)	1.73:1
60°-90°	8.45 (6.17)	2.55:1
90°-120°	15.78 (7.01)	0.90:1
0°-30°	0.73 (4.91)	40.05:1
30°-45°	5.69 (2.33)	1.64:1
45°-60°	5.29 (6.15)	1.83:1
60°-75°	3.15 (3.67)	3.76:1
75°-90°	5.29 (5.83)	1.83:1
90°-120°	15.78 (7.01)	0.09:1

For each shoulder elevation increment mean ± SD were calculated for scapular upward rotation. Scapular rotation was subtracted from each 15, 30, or 120 degree increment to determine glenohumeral contribution to shoulder elevation. Using the glenohumeral and scapular rotation values, scapulohumeral rhythm (glenohumeral:scapular) was calculated for each increment. SD represents standard deviation.

Statistical analysis

Means and standard deviations were calculated for the demographic data. Mean scapular rotation was calculated at 15 and 30 degree increments. Scapular motion was examined over several increments (Table 1).

Scapulohumeral rhythm was calculated using both the 15 degree and 30 degree increments by subtracting scapular rotation from shoulder elevation to determine glenohumeral joint contribution to shoulder elevation (Equation 1). The ratio of glenohumeral motion to scapular motion was then calculated to derive scapulohumeral rhythm (Equation 2).

(Eq. 1) Glenohumeral motion = (Total shoulder motion) - (scapular upward rotation)

(Eq. 2) Scapulohumeral rhythm = (Glenohumeral elevation)/(Scapular upward rotation)

Scapular contributions to shoulder elevation were calculated and percent contributions were calculated according to the aforementioned increment (Equation 3).

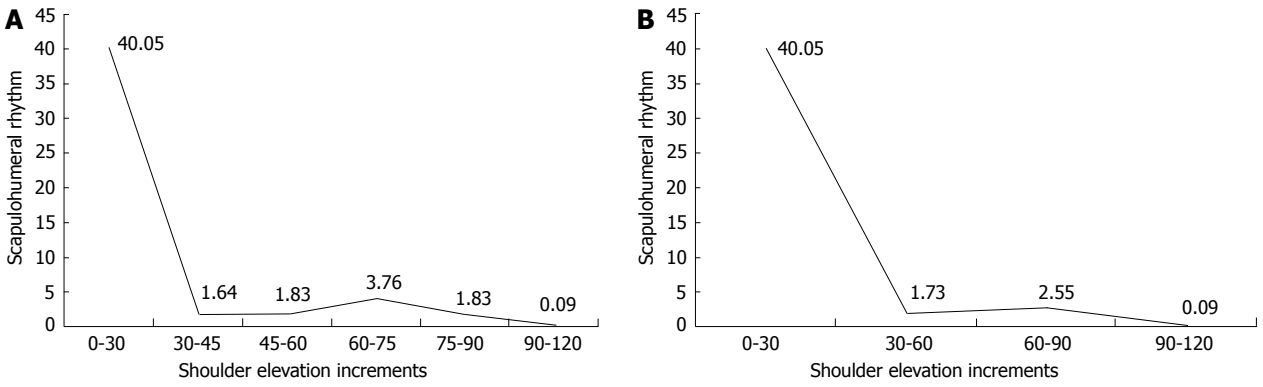

Figure 2 Scapulohumeral rhythm for 15 shoulder elevation increments (A) and 30 degree shoulder elevation Increments (B). Shoulder elevation increments represent degrees of shoulder elevation in the scapular plane. Values listed in the figure represents degrees of glenohumeral motion for every 1° of scapular upward rotation.

Table 2 Dominant shoulder range of motion and normalized strength

	Active range of motion (± SD)	Passive range of motion (± SD)	Normalized strength (%) (± SD)
Flexion	175.86° (7.04°)	179.86° (0.53°)	19.29 (4.80)
Abduction	179.07° (1.86°)	180.00° (0.00°)	18.53 (3.90)
Internal rotation	52.43° (12.11°)	60.21° (13.72°)	17.25 (4.43)
External rotation	107.79° (7.78°)	129.14° (11.95°)	12.54 (3.37)

(Eq. 3)% Contribution $_{Scapula}$ = (Scapular rotation)/(Total shoulder elevation).

Excel (Microsoft, Redmond, WA) and SPSS version 17.0 (IBM Corporation, Armonk, NY) were used for all statistical analyses. A one-way ANOVA was performed to compare mean SUR between 30 degree elevation increments. A Bonferroni *t*-test was used to address multiple comparisons between the elevation increments. An $\alpha = 0.05$ was set a priori.

RESULTS

Means and standard deviations for shoulder ranges of motion and strength are provided in Table 2. Ranges of shoulder motion and strength were within clinically acceptable normal limits and were representative of subjects reporting healthy shoulders.

The assessment of scapulohumeral rhythm across the entire arc of humeral elevation was very consistent with the 2:1 ratio defined by Inman. Scapulohumeral rhythm for the observed range represented a ratio of 2.34:1. However, when assessing scapulohumeral rhythm across the different increments of humeral elevation ratios ranged from 40.01:1 to 0.90:1. Following minimal contributions from the scapula during the first 30 degrees of humeral elevation mean scapulohumeral rhythm ratios when assessed over 15 and 30 degree increments ranged from 0.90:1 to 3.76:1 (Figure 2 A and B).

Total scapular motion increased over the arc of shoulder elevation. Contributions of the scapula to overall shoulder elevation were initially minimal when considering the first 30 degrees of shoulder elevation. These contributions continued to increase by varying degrees over the course of shoulder elevation. However, these increases did not appear to occur in a linear fashion. Assessing the scapula's percent contributions to shoulder elevation across all subjects provided confirmation of this non-linear trend. Percent contributions of the scapula to shoulder elevation were substantially lower for the first 30 degrees of elevation, representing only 2.53% of the first 30 degrees of shoulder elevation. When examining 15° increments of shoulder elevation ranging from 30° to 90°, scapular rotation contributed between 20.87% and 37.53% to total shoulder motion. These contributions were not consistent across increments, and this trend continued when examining the increased scapular contributions percentage from 90° to 120° of elevation which reached 52.73% of total shoulder motion (Figure 3).

Statistically significant differences in scapular upward rotation were identified across the shoulder elevation increments [$F_{(3,48)} = 12.63$, $P = 0.0001$]. The Boneferoni assessment revealed multiple significant differences between the 30 degree increments (Table 3). Differences in scapular rotation were not assessed for the 15 degree humeral elevation increments as this data was only obtained for a portion of the humeral elevation arc (30°-90°).

DISCUSSION

We were able to quantify the contributions of scapular upward rotation to shoulder elevation occurring in the scapular plane and we were able to provide evidence, using clinically available instrumentation, that scapulohumeral rhythm, as it relates to scapular upward rotation, does not conform entirely to the often accepted 2:1 ratio described by Inman[7]. While the overall ratio of 2.34:1 is in close agreement with the accepted norm, incremental observations of scapulohumeral rhythm were more consistent with recent observations, which are not entirely consistent with the 2:1 ratio[17,29,30]. We also observed increasingly more scapular upward rotation as the shoulder achieved greater amounts of elevation in the scapular plane, which is consistent with the scapula's role as it relates to optimizing function of the glenohumeral

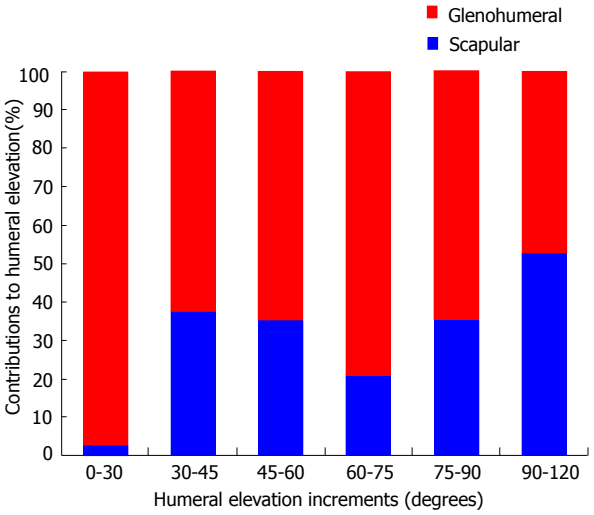

Figure 3 Glenohumeral and scapular upward rotation contributions to shoulder elevation.

Table 3 Multiple comparisons analyzing scapular upward rotation per 30 degree increments of shoulder elevation

Increment$_1$	Increment$_2$	Mean difference	Significance	95% confidence interval
0°-30°	30°-60° [1]	-10.26	0.001	-17.14; -3.37
	60°-90° [1]	-7.72	0.02	-14.60; -0.83
	90°-120° [1]	-15.05	≤ 0.001	-21.94; -8.17
30°-60°	0°-30° [1]	10.26	0.001	3.37; 17.14
	60°-90°	2.54	1	-4.35; 9.42
	90°-120°	-4.8	0.367	-11.68; 2.09
60°-90°	0°-30° [1]	7.72	0.02	0.83; 14.60
	30°-60°	-2.53	1	-9.42; 4.35
	90°-120° [1]	-7.33	0.031	-14.22; -0.45
90°-120°	0°-30° [1]	15.05	≤ 0.001	8.17; 21.94
	30°-60°	4.8	0.367	-2.09; 11.68
	60°-90° [1]	7.33	0.031	0.45; 14.22

One-way ANOVA revealed a statistically significant difference between 30 degree shoulder elevation increments for scapular rotation [$F_{(3,48)}$= 12.63, P ≤ 0.0001]. Boneferoni t-tests revealed statistically significant differences. [1]for scapular rotation between 30 degree elevation increments.

joint during overhead activity. Much like scapulohumeral rhythm, upward rotation contributions of the scapula to shoulder elevation varied across the arc of shoulder elevation, which is both consistent with previous reports[23,26,31] and provides support for use of the digital inclinometer in clinical settings to effectively monitor scapular motion as part of both injury evaluation and rehabilitation.

The scapulohumeral rhythm ratios we observed ranged from 40:1 to 0.90:1. Although our results relative to scapulohumeral rhythm across the entire range of motion are in fairly close agreement with the 2:1 ratio suggested by Inman *et al*[7], the 2:1 ratio is an average that does not represent the substantial variability observed throughout the range. The exceptionally high ratio obtained during the first 30 degrees of humeral elevation is consistent with the scapular setting phase that is often described when discussing shoulder kinematics and confirmed the observations of some investigators[8,10,17]. The observed ratio not only suggests that the scapula engages in a minimal amount of upward rotation, but rather that during the initial stages of shoulder elevation a period of downward rotation may be encountered. Borsa *et al*[26] reported a similar decline in upward rotation during the first 30° of shoulder elevation in the scapular plane. It is unclear what may have caused this downward rotation and while it is beyond the scope of our study it is plausible that weakness of the scapula stabilizing muscles may have contributed to the observed pattern. Following the setting phase, scapulohumeral rhythm approached the norms of scapulohumeral rhythm in a fashion consistent with what others have presented[17,29,30,32-34]. Scapulohumeral rhythm ratios between 30° and 90° ranged from 1.64:1 to 3.76:1; but none were equal to the clinically accepted 2:1 ratio. We then observed a decline in scapulohumeral rhythm (0.90:1) as subjects elevated their shoulders from 90° to 120° of elevation. This decline in scapulohumeral rhythm adds to the degree of heterogeneity in the literature as it relates to scapulohumeral rhythm at higher ranges of shoulder elevation[8-10,17,26,29-31]. However, our results are more consistent with those investigators that have indicated that scapulohumeral rhythm ranges between a 1:1 and 2:1 ratio[8-10,17,18,29,30,32-35]. Factors that could explain this observed shift include increases in inferior glenohumeral ligament tension[34], required positioning of the scapula to maintain function and stability of the glenohumeral joint, and maintenance of the subacromial space to avoid impingement[4].

We observed increases in total scapular upward rotation as the level of shoulder elevation increased; however, these increases varied considerably between observed increments. Over the arc of shoulder elevation the scapula encountered 35.95° of upward rotation, resulting in a scapulohumeral rhythm of 2.34:1. Consistent function of the shoulder complex relies on reliable patterns of scapular and glenohumeral motion during shoulder elevation and lowering to ensure optimal stability and mobility[29,31]. The relative increase in scapular upward rotation is similar to both what has been observed with electromagnetic tracking systems[17,29,31-34] and modified digital inclinometers[12,21-27]. Although we noted a relative overall increase in scapular upward rotation, we identified significant differences in scapular rotation between 30 degree shoulder elevation increments, which served to support our secondary hypothesis. The results of Witwer and Sauer[23] and Borsa *et al*[23,26,31] relative to scapular upward rotation and Borstad and Ludewig[33] specific to scapular tilting foreshadowed our findings with each noting differences or variability in scapular motion when comparing increments of shoulder elevation. The differences we observed between the 0°-30° increment and all other increments are logical given the limited degree to which the scapula moved during the setting phase. Given the often reported stability in scapulohumeral motion between 30° and 90° it is not surprising that differences were not noted between the 30°-60° and 60°-90°

increments. It is, however, interesting that the 90°-120° increment was not different when compared to all other increments in light of the scapulohumeral rhythm shift often observed when shoulder elevation occurs above 90°. One factor that may have contributed is the slightly higher relative variability in scapular rotation measures for the 30°-60° increment. This pattern in variability is not unlike what has been reported in other studies[23,26,31] and may have contributed to the lack of significant differences between the 30°-60° and 90°-120° increments. Ultimately, the pattern of scapular rotation we observed during shoulder elevation is similar to what clinicians might observe including (1) a scapular setting phase with little scapular rotation; (2) a relative but inconsistent increase in scapular contributions to shoulder elevation; followed finally by (3) increasingly more upward rotation of the scapula, which mirrors that of the humerus.

Contributions of the scapula to shoulder elevation reflected both inconsistencies in scapulohumeral rhythm and variable scapular upward rotation contributions to shoulder elevation. Again, the contributions of the scapula to shoulder elevation increments 0°-30° and 90°-120° were comparable to what has been reported in the literature, with the scapula contributing 2.53% and 52.73% of the total shoulder elevation, respectively. Examining the shoulder elevation arc incrementally from 30° to 90°, we noted fluctuations in percent scapular contributions to shoulder elevation, which mirrored the inconsistent patterns of scapulohumeral rhythm we observed. In order to be consistent with the 2:1 ratio scapular contributions needed to account for 33.33% of each increment while the glenohumeral joint accounted for the remaining 66.67%. The 15 degree increments 30°-45°, 45°-60° and the 75°-90° and the 30 degree increments (30°-60° and 60°-90°) approached but did not achieve these percent contributions. Reflecting on the scapular contributions across all increments of shoulder elevation, variability in scapular contributions are apparent, which suggests that while the 2:1 ratio may be easy to recall for clinical use, the 2:1 ratio does not accurately portray both scapular contributions and scapulohumeral rhythm observed during shoulder elevation in the scapular plane.

As healthcare and the focus on patient outcomes continue to evolve and our understanding of shoulder kinematics and shoulder pathologies changes as clinicians we must be prepared to add new measures to our clinical repertoires. A number of investigators have examined the contributions of the scapula to shoulder motion in pathologic shoulders using a variety of instruments[19,20,25,35-41], ultimately providing investigators and clinicians with a greater appreciation of scapular kinematics exhibited by various shoulder patient populations. While some have focused on scapulohumeral rhythm and changes in scapular upward rotation, many have expanded their efforts to consider the roles that the secondary scapular rotations (anterior-posterior tilt and medial-lateral tilt)[4] also have in shoulder function and shoulder pathologies. Much of our more recent understandings have come through the use of electromagnetic tracking systems, which are not always clinically viable options. Though visual inspection systems[42,43] may afford us the opportunity to globally identify scapular dysfunction, without the ability to quantify and fully appreciate these patterns in our patients the ability to deliver optimal care may be impacted. Our work and the recent work of others continues to support the clinical usefulness and applicability of the digital inclinometer for quantifying scapular kinematics. As our understanding of scapular kinematics continues to evolve and the needs for patient outcomes relative to these areas changes, the digital inclinometer will continue to provide an affordable and accessible means for quantifying shoulder and scapular kinematics.

Limitations

Even though we were able to demonstrate the clinical usefulness of the digital inclinometer for assessing scapulohumeral rhythm there were some areas of our study that may be viewed as weaknesses or that should be considered in future investigations. In our attempts to be more discriminate in our analysis of scapular kinematics we utilized 15 degree increments throughout the mid-range of the shoulder elevation arc. Unfortunately, we did not utilize this approach across the entire arc of elevation, focusing primarily on clinically relevant angles of shoulder elevation, thus limiting our ability to perform this detailed analysis throughout the observed motion. Future efforts should consider adding additional assessment increments and potentially extending the arc of shoulder elevation beyond 120°. Furthermore, it may have been useful to expand the study and include gender comparisons and bilateral comparison to ensure that the observed pattern occurred similarly in genders and in both the dominant and non-dominant shoulders. Future studies should also consider assessing scapula stabilizing strength at various points in the arc of shoulder elevation to determine if any relationships exist between scapular kinematics and stabilizing muscle strength. The roles of both generalized joint laxity and specifically shoulder ligament laxity should also be considered in future work in order to determine their respective impacts on scapulohumeral rhythm. Additionally, while we were able to call into question the widely accepted 2:1 ratio for scapulohumeral rhythm, greater attention should be directed at determining the clinical meaningfulness of this difference and the extent to which it may contribute to or the role it may have in perpetuating various shoulder pathologies.

In our efforts to gain a better appreciation of the coordinated movements of the scapula and humerus, while using a digital inclinometer we observed scapulohumeral rhythms that were not in total agreement with the widely accepted 2:1 ratio for shoulder elevation. Our observations were confirmed based upon percent contributions of the scapula to shoulder elevation and the significant differences in scapular contribution we noted when comparing shoulder elevation increments. Our findings

confirm the presence of a scapular setting phase early in the arc of shoulder elevation, followed by a period of relatively stable scapulohumeral rhythms which then continue to decline as the shoulder moves above 90° of elevation. While our results do not fully support the accepted 2:1 ratio, as investigators and clinicians we must recognize the usefulness of the inclinometer in documenting not only the variable nature of scapulohumeral rhythm in healthy shoulders but the likelihood of increasingly more variant scapulohumeral rhythm patterns exhibited in shoulder injured patients.

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COMMENTS

Background

The introduction of electromagnetic tracking systems has resulted in an evolution in kinematic assessment as it relates to the understanding of scapulohumeral rhythm in both healthy and injured populations. Electromagnetic tracking systems have improved our understanding of shoulder kinematics; however, instrumentation accessibility has often precluded clinicians from being able to quantify glenohumeral and scapulothoracic joint contributions to shoulder motion. Although, the validation of the digital inclinometer for assessing scapular upward rotation has made scapular kinematic assessment more accessible, no studies involving digital inclinometers have attempted to examine scapulohumeral rhythm specifically.

Research frontiers

The ability to utilize the inclinometer for quantifying scapulohumeral rhythm will provide clinicians with a means to monitor the often variable nature of scapulohumeral rhythm while also comparing the functional capacity of the shoulder to the established 2:1 scapulohumeral movement ratio.

Innovations and breakthroughs

Scapulohumeral rhythm for the entire arc of shoulder elevation was equal to a ratio of 2.34:1 and ranged from 40.01:1 to 0.90:1, which was not entirely consistent with the accepted scapulohumeral rhythm ratio. Although the scapula experienced variable upward rotation throughout the arc of shoulder elevation, our findings confirmed the presence of a scapular setting phase early in the arc of shoulder elevation, followed by a period of relatively stable scapulohumeral rhythms, which proceeded to decline as the shoulder moved above 90° of elevation.

Applications

The significance of the study is that investigators and clinicians should consider the usefulness of the inclinometer in documenting the variable nature of scapulohumeral rhythm in healthy shoulders and shoulder injured patients.

Terminology

Scapulohumeral rhythm: the coordinated motion of the scapula and humerus experienced during shoulder movement and motion that has been traditionally viewed as occurring at a ratio of 2:1 (2 degrees of humeral flexion/abduction to 1 degree of scapular upward rotation).

Peer review

A modified digital inclinometer was used to measure scapular upward rotation of the subject's dominant shoulder. The authors concluded that the scapulohumeral rhythms measured by a modified digital inclinometer were not consistent with the widely accepted 2:1 ratio for shoulder elevation. Future research should consider the role of hand dominance, gender, and ligamentous laxity as it relates to scapulohumeral rhythm.

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Sorin Siegler, PhD, Professor of Mechanical Engineering and Biomechanics, Department of Mechanical Engineering, Drexel University, Philadelphia, Pennsylvania 19104, United States

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January 23-25, 2012
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American Academy of Orthopaedic Surgeons
San Diego, CA, United States

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Orthopaedic MRI and Small Parts
Scottsdale, AZ, United States

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The 30th Annual Emergencies in Medicine Conference
Utah, UT, United States

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Editor-in-chief

Bao-Gan Peng, MD, PhD, Professor, Department of Spinal Surgery, General Hospital of Armed Police Force, 69 Yongding Road, Beijing 100039, China

Editorial Office

World Journal of Orthopedics
Editorial Department: Room 903, Building D,
Ocean International Center,

No. 62 Dongsihuan Zhonglu,
Chaoyang District, Beijing 100025, China
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6 21st century heart solution may have a sting in the tail. *BMJ* 2002; **325**: 184 [PMID: 12142303 DOI:10.1136/bmj.325.7357.184]

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10 **Sherlock S**, Dooley J. Diseases of the liver and billiary system. 9th ed. Oxford: Blackwell Sci Pub, 1993: 258-296

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15 Morse SS. Factors in the emergence of infectious diseases. Emerg Infect Dis serial online, 1995-01-03, cited 1996-06-05; 1(1): 24 screens. Available from: URL: http://www.cdc.gov/ncidod/eid/index.htm

Patent (list all authors)

16 **Pagedas AC**, inventor; Ancel Surgical R&D Inc., assignee. Flexible endoscopic grasping and cutting device and positioning tool assembly. United States patent US 20020103498. 2002 Aug 1

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