Physical Therapy and Surgery

Rafael Valle-Onate, MD, Michael M. Ward, MD and Gail S. Kerr, MD

Guest Editors: *Atul Deodhar, MD and Daniel Clegg, MD*

Abstract: Physical therapy and orthopedic surgery are important components in the treatment of ankylosing spondylitis (AS). Supervised physical therapy is more effective than individual or unsupervised exercise in improving symptoms, but controlled trials suggest that combined inpatient and outpatient therapy provides the greatest improvement. Recommendations for exercise are universal, but the best types and sequence of therapies are not known. Total hip replacement is the surgery most commonly performed for AS, with good long-term implant survival. Heterotopic ossification may occur no more frequently after hip replacement in patients with AS than in patients with other diseases. Corrective spinal surgery is rarely performed and requires specialized centers and experienced surgeons.

Key Indexing Terms: Total hip arthroplasty; Physical therapy; Ankylosing spondylitis. **[Am J Med Sci 2012;343(5):353–356.]**

There are 2 main types of treatment of ankylosing spondylitis (AS): pharmacologic and nonpharmacologic. Although pharmacologic therapy has improved dramatically in recent years with the advent of anti-tumor necrosis factor (TNF) therapy, nonpharmacologic treatments remain an important component of comprehensive care throughout the course of AS.[1] Physical therapy and orthopedic surgery are the main nonpharmacologic treatments available for AS.

PHYSICAL THERAPY

A principal symptom of AS is loss of flexibility. This often causes abnormal body posture and affects spine biomechanics. Early limitation of spinal mobility has been identified as one of the most important prognostic factors in AS.[2] Physical therapy is directed mainly at patient education and regular exercise, with the goals of preserving spinal flexibility and fitness, preventing postural deformities and improving muscle strength, thereby reducing pain.[2] Rather than removing the motivation to exercise, patients treated with anti-TNF agents seem to exercise more than they did before using this medication and feel that physical therapy is even more helpful in improving their stiffness, function and motivation after starting treatment.[3]

Various types of exercise programs have been developed worldwide, which are as follows: individualized physical therapy, supervised group physical therapy and unsupervised self-administered exercise.[4] A meta-analysis of 11 clinical trials indicated that a home exercise program is better than no program at all; at the same time, supervised group physical therapy is better than home exercise, and finally combined inpatient spa-exercise therapy followed by supervised weekly group physical therapy is the most effective program available today.[5] Intensive inpatient courses have shown to be effective, but the results of outpatient programs have been more varied in therapeutic and educational effect.[6] Although inpatient treatment courses are common in Western Europe, they are not in other regions.

In practice, many patients often find it difficult to perform daily exercises on their own. Supervised group physical therapy is offered mainly to stimulate and motivate and provide social contact with fellow patients. Also, the supervising physiotherapist can closely monitor the intensity of the exercises to achieve improvement. Group physical therapy usually consists of 1 hour of physical exercise, 1 hour of sport and 1 hour of inpatient spa therapy.[4] Therapy in a spa provides complementary effects over self-exercise and group-exercise alone, and these effects may persist for several months. Furthermore, some evidence suggests that the cost-utility and cost-effectiveness of inpatient spa therapy are favorable compared with those of self-exercise and group-exercise alone.[6]

Although studies have tested several different physical therapy programs, the optimal exercise program for patients with AS is still not known, primarily because interventions are often poorly or incompletely described, different types of exercises and training doses are used and the expected physiologic responses to the exercises are not defined.[5]

When recommending sports, it is advisable for patients to engage in noncontact rather than contact sports. There are no uniform exercises for all patients, and therapists can serve an important role in examining each patient individually and developing a personalized protocol.[7] The therapist can teach the patient how to move, how to rest and which sports are appropriate (eg, badminton, volleyball, swimming and cross-country skiing,) and which are not (horseback riding and football).[4]

Individual variation in the course of AS is considerable, and an understanding of the pathophysiologic process and biomechanical principles is an important factor in planning individual programs; therefore, studies that include these aspects must be evaluated.[2] Additionally, controlled studies that compare different treatment programs would be of great value.[6] Research on physical therapy interventions in AS can be improved, including better measurement techniques, more detailed analysis of treatment programs and better understanding of the relationships between dose and effect.

Notwithstanding the need for better knowledge of what constitutes the most effective exercise and physical therapy programs, a clinical prediction rule has been developed to identify patients with AS who are more likely to respond to an exercise program.[8] The study suggests that pain and function can be better indicators of the response to exercise than some traditionally used impairment measures such as spinal range of motion. Other clinical prediction rules have been used in the classification of patients with low-back pain, neck pain or tension headache and are useful in selecting a treatment protocol

From the Military Hospital School of Medicine (RV-O), Universidad la Sabana, Bogota, Colombia; National Institute of Arthritis and Musculoskeletal and Skin Diseases (MMW), National Institutes of Health, Bethesda, Maryland; and the Department of Medical Service (GSK), Veterans Affairs Medical Center, Washington, District of Columbia.

Presented at the annual research and education meeting of Spondyloarthritis Research and Treatment Network (SPARTAN), Portland, Oregon, July 29–30, 2011.

Correspondence: Michael M. Ward, MD, National Institute of Arthritis and Musculoskeletal and Skin Diseases, National Institutes of Health, 9000 Rockville Pike, Bethesda, MD 20893 (E-mail: wardm1@mail.nih.gov).

for each individual. Future studies are necessary to validate these prediction rules.

Although much is known regarding physical therapy and exercise, important advances are yet to be made in how best to apply these in the treatment of patients with AS. Such studies of physical therapy are urgently needed because physical therapy represents an important complement to pharmacologic treatments and helps to improve patients' physical function and emotional well-being.

SURGICAL CONSIDERATIONS

With regards to surgical intervention in AS, current Assessment of SpondyloArthritis International Society (ASAS) guidelines are based on level III evidence and expert opinion.[9] The low hierarchy of evidence for surgical intervention is attributed to the technical and ethical constraints of performing randomized, controlled trials for these procedures. Evidence-based cross-sectional and retrospective studies are of small patient numbers, short duration and have not included placebo arms nor stratified for disease-modifying antirheumatic or biologic therapy. Limitations of data, study design and level of evidence persist in studies published after these guidelines.

The first component of current ASAS surgical guidelines states "Total hip arthroplasty should be considered in patients with refractory pain or disability and radiographic evidence of structural damage, independent of age."[9] Recent data confirm that inflammatory hip disease occurs in 25% to 50% of patients with AS, and when present, is bilateral in as many as 47% to 90%.[10,11] Juvenile AS patients are more likely affected (~2:1), as are males and those with axial disease and enthesitis.[12,13] A cross-sectional analysis of 2718 AS patients from multinational registries found hip involvement to account for a differential in Bath Ankylosing Spondylitis Functional Index of 1.6 and was associated with cervical, shoulder and spine immobility.[10] Several studies continue to support improvement in function and pain relief after total hip replacement (THR).[11,14–18] Survival of prostheses remains favorable with 90% at 10 years and remains at 65% and 71% after 20 and 27 years, respectively. The failure rate of hip prostheses is approximately 1% per year, and revisions within 7 years result from aseptic loosening. Revised THR have a 20-year survival of 60%. Comparisons of cemented versus noncemented prosthesis report failure rates of 5% and 28%, respectively.[16] However, noncemented prostheses are preferred, as patients are usually young, and hence potential revisions are technically less problematic.[16,17]

Most studies report hip flexion contracture or complete ankylosis as indications for THR in AS. Details regarding an anti-inflammatory or degenerative etiology are lacking, and histological evaluations of specimens are not performed routinely. One retrospective study of 181 hips undergoing THR in 103 AS patients alluded to the etiology of hip disease by describing radiographic changes of protrusio acetabuli in 20.4%, bony ankylosis in 23.2% and either upper pole or concentric osteoarthritis in 42.5%.[18] The distinction is indeed important, as AS of the hip, unlike the osteoproliferative changes of the spine, involves erosive lesions with inflammation of subchondral bone marrow.[19] Further, unlike the reported, although short term, lack of efficacy of biologic therapy in disease modification of the spine in AS, there are reports of efficacy with anti-TNF agents, not only with symptoms and composite disease scores but also of joint space narrowing of the hip in AS patients.[20,21] Since the approval of anti-TNF agents in AS, preliminary cohort data indicate a decrease in frequency of THR for AS.[22] A more precise definition of hip disease and distinction of inflammatory versus noninflammatory hip arthritis by improved biomarkers, particularly in those with high Bath Ankylosing Spondylitis Functional Index scores, are needed to better assess the outcomes of THR in AS patients and to determine the appropriate use and efficacy of biologic agents.

A potential complication of THR in AS patients is heterotopic ossification (HO), but for which there are no ASAS guidelines. HO is reported to occur in as many as 40% of AS patients, often asymptomatic, and when moderate to severe, results in limited range of motion (Brooker class III-IV).[11,23] HO consists of both cancellous and cortical bone with areas of fibrocartilage, and remodeling may continue for as long as 3 years.[24] Contrary to older literature, AS patients seem not to be at greater risk of HO and have similar rates as patients with diffuse idiopathic skeletal hyperostosis, Paget's disease, unilateral hypertrophic osteoarthritis or who have reduced preoperative limitation in hip external rotation.[25,26] Patients with AS considered at risk are those who need repeat surgery, develop postoperative infection, undergo a transtrochanteric approach or have concurrent active disease.[11,15] One study of 20 AS patients found HO to occur in 30% and correlated with C-reactive protein levels.[27] Randomized, controlled studies support nonsteroidal anti-inflammatory agents (NSAIDs), most often indomethacin, and radiation as efficacious for prophylaxis but yield inconsistent results.[28,29] Both options are advised perioperatively, optimally 24 to 48 hours after surgery, and at least within 5 days.[25,29] Cyclo-oxygenase-2 inhibitors are reportedly as effective as NSAIDs, with fewer adverse effects, though more costly.[30,31] A cost comparison of NSAIDs and radiation found radiation to be about 45 times more expensive, and a meta-analysis reported an incremental cost-effectiveness ratio of approximately $6000 per additional case of HO prevented.[30,32] Although no difference in short-term side effects was reported, the rate of NSAID complications requiring treatment and long-term effects still needs to be considered. Despite the evidence, it is debatable if prophylaxis of HO is cost-effective and without long-term sequelae, and whether it is indicated for all AS patients.

The second component of the 2005 ASAS surgical guidelines states "spinal surgery—for example, corrective osteotomy and stabilization—may be of value in selected patients."[9] Indications for corrective osteotomy include functional (the inability to eat or swallow), clinical (loss of horizontal gaze and abnormal chin-brow angles) or radiographic (rigid deformities). Three surgical options have been reported as follows: open, closed and polysegmental osteotomy. A large series of 856 patients that utilized all 3 techniques concluded that the closed method had the fewest complications although mortality was 4%.[33] Specialized support is often needed, including fiberoptic intubation, an intraoperative "wake up test" and, when performed in the cervical region, continuous neurological monitoring, which often requires the patient to remain awake. A retrospective study of 148 patients who underwent corrective spinal surgery and survived found 88% satisfied and 60% able to return to work.[34]

Despite improved therapies and sensitivity to the need to treat osteoporosis associated with inflammatory rheumatic disease, vertebral fractures still occur more commonly in patients with AS than in patients without AS and are often misdiagnosed. In a case-control study of 53,108 patients with inflammatory rheumatic disease, vertebral fractures were 7.1 times more likely among those with AS.[35] In a 7-year retrospective study of hypertrophic spine disease inclusive of diffuse idiopathic skeletal hyperostosis, vertebral fractures were most common in the cervical spine (C6-C7), neurological complications occurred in 58% and mortality in 32%.[36] In the AS group, there was a delay

in diagnosis of vertebral fractures, and patients were younger. In another retrospective study of 119 patients, more than a quarter of vertebral fractures were assessed incorrectly, one-half were preceded by trivial trauma and spinal cord injury ensued in 60%.[37] Mortality was 32% and correlated with age, number of comorbidities and low-energy mechanism of injury. Controversy persists regarding the sequence of intervention when more than one kyphotic lesion exists, or where there is coexistent hip disease.[11] Careful positioning, appropriate imaging, interpretation and referral are required for early detection and optimum outcomes of vertebral fractures.

Case series and meta-analyses confirm cauda equina to be a rare complication of AS, in which dural sac enlargement and arachnoid diverticulae lead to progressive neurological impairment.[38,39] Lumboperitoneal decompression and infliximab have both been reported to be effective treatments.[40,41]

REFERENCES

1. **Elyan M, Khan MA.** Does physical therapy still have a place in the treatment of ankylosing spondylitis? Curr Opin Rheumatol 2008;20: 282–6.
2. **Nghiem FT, Donohue JP.** Rehabilitation in ankylosing spondylitis. Curr Opin Rheumatol 2008;20:203–7.
3. **Masiero S, Bonaldo L, Pigatto M, et al.** Rehabilitation treatment in patients with ankylosing spondylitis stabilized with tumor necrosis factor inhibitor therapy. A randomized controlled trial. J Rheumatol 2011; 38:1335–42.
4. **van der Linden S, van Tubergen A, Hidding A.** Physiotherapy interventions for ankylosing spondylitis: what is the evidence? Clin Exp Rheumatol 2002;20(suppl 28):S60–4.
5. **Dagfinrud H, Hagen KB, Kvien TK.** Physiotherapy interventions for ankylosing spondylitis. Cochrane Database Syst Rev 2008;23:CD002822.
6. **van Tubergen A, Hidding A.** Spa and exercise treatment in ankylosing spondylitis: fact or fancy? Best Pract Res Clin Rheumatol 2002;16: 653–66.
7. **Viitanen JV, Suni J.** Management principles of physiotherapy in ankylosing spondylitis—which treatments are effective? Physiotherapy 1995;81:322–9.
8. **Alonso-Blanco C, Fernandez-de-las-Peñas C, Cleland JA.** Preliminary clinical prediction rule for identifying patients with ankylosing spondylitis who are likely to respond to an exercise program: a pilot study. Am J Phys Med Rehabil 2009;88:445–54.
9. **Zochling J, van der Heijde D, Burgos-Vargas R, et al.** ASAS/EULAR recommendations for the management of ankylosing spondylitis. Ann Rheum Dis 2006;65:442–52.
10. **Vander Cruyssen B, Muñoz-Gomariz E, Font P, et al**; **ASPECT-REGISPONSER-RESPONDIA working group.** Hip involvement in ankylosing spondylitis: epidemiology and risk factors associated with hip replacement surgery. Rheumatology (Oxford) 2010;49:73–81.
11. **Kubiak E, Moskovich R, Errico T, et al.** Orthopaedic management of ankylosing spondylitis. J Am Acad Orthop Surg 2005;13:267–78.
12. **Ibn YY, Amine B, Laatiris A, et al.** Gender and disease features in Moroccan patients with ankylosing spondylitis. Clin Rheumatol 2012; 31:293–7.
13. **Jang JH, Ward MM, Rucker AN, et al.** Ankylosing spondylitis: patterns of radiographic involvement—a re-examination of accepted principles in a cohort of 769 patients. Radiology 2011;258:192–8.
14. **Sweeney S, Gupta R, Taylor G, et al.** Total hip arthroplasty in ankylosing spondylitis: outcome in 340 patients. J Rheumatol 2001;28: 1862–6.
15. **Brinker MR, Rosenberg AG, Kull L, et al.** Primary noncemented total hip arthroplasty in patients with ankylosing spondylitis: clinical and radiographic results at an average follow-up period of 6 years. J Arthroplasty 1996;11:802–12.
16. **Tang WM, Chiu KY.** Primary total hip arthroplasty in patients with ankylosing spondylitis. J Arthroplasty 2000;15:52–8.
17. **Sochart DH, Porter ML.** Long-term results of total hip replacement in young patients who had ankylosing spondylitis: eighteen to thirty-year results with survivorship analysis. J Bone Joint Surg Am 1997;79: 1181–9.
18. **Joshi AB, Markovic I, Hardinge K, et al.** Total hip arthroplasty in ankylosing spondylitis: an analysis of 1.81 hips. J Arthroplasty 2002;17: 427–33.
19. **Appel H, Kuhne M, Spiekermann S, et al.** Immunohistochemical analysis of hip arthritis in ankylosing spondylitis: evaluation of the bone-cartilage interface and subchondral bone marrow. Arthritis Rheum 2006;54:1805–13.
20. **Verbruggen G.** Chondroprotective drugs in degenerative joint diseases. Rheumatology (Oxford) 2006;45:129–38.
21. **Lian F, Yang X, Liang L, et al.** Treatment efficacy of etanercept and MTX combination therapy for ankylosing spondylitis hip joint lesion in Chinese population [published online ahead of print March 9, 2011]. Rheumatol Int doi: 10.1007/s00296-011-1844-8.
22. **Baraliakos X, Braun J.** Hip involvement in ankylosing spondylitis: what is the verdict? Rheumatology (Oxford) 2010;49:3–4.
23. **Brooker Al, Bowerman JW, Robinson RA, et al.** Ectopic ossification following total hip replacement: incidence and a method of classification. J Bone Joint Surg Am 1973;55:1629–32.
24. **Isaacson BM, Brown AA, Brunker LB, et al.** Clarifying the structure and bone mineral content of heterotopic ossification. J Surg Res 2011; 167:e163–70.
25. **Iorio R, Healy W.** Heterotopic ossification after hip and knee arthroplasty: risk factors, prevention, and treatment. J Am Acad Orthop Surg 2002;10:409–16.
26. **Schwarzkopf R, Cohn RM, Skoda EC, et al.** The predictive power of preoperative hip range of motion for the development of heterotopic ossification. Orthopedics. 2011;34:169.
27. **Tani Y, Nishioka J, Inoue K, et al.** Relation between ectopic ossification after total hip arthroplasty and activity of general inflammation in patients with ankylosing spondylitis. Ann Rheum Dis 1998;57:634.
28. **Pakos E, Ioannidis J.** Radiotherapy vs. nonsteroidal anti-inflammatory drugs for the prevention of heterotopic ossification after major hip procedures: a meta-analysis of randomized trials. Int J Radiat Oncol Biol Phys 2004;60:888–95.
29. **Hashem R, Tanzer M, Rene N, et al.** Postoperative radiation therapy after hip replacement in high-risk patients for development of heterotopic bone formation. Cancer Radiother 2011;15:261–4.
30. **Vavken P, Castellani L, Sculco T.** Prophylaxis of heterotopic ossification of the hip: systematic review and meta-analysis. Clin Orthop Relat Res 2009;467:3283–9.
31. **Vasileiadis GI, Sioutis IC, Mavrogenis AF, et al.** COX-2 inhibitors for the prevention of heterotopic ossification after THA. Orthopedics 2011;34:467–72.
32. **Strauss JB, Chen S, Shah AP, et al.** Cost of radiotherapy versus NSAID administration for prevention of heterotopic classification after total hip arthroplasty. Int J Radiat Oncol Biol Phys 2008;71:1460–4.
33. **Van Royen BJ, De Gast A.** Lumbar osteotomy for correction of thoracolumbar kyphotic deformity in ankylosing spondylitis: a structured review of three methods of treatment. Ann Rheum Dis 1999;58: 399–406.

34. **Halm H, Metz-Stavenhagen P, Zielke K.** Results of surgical correction of kyphotic deformities of the spine in ankylosing spondylitis on the basis of the modified arthritis impact measurement scales. Spine (Phila Pa 1976) 1995;20:1612–9.

35. **Weiss RJ, Wick MS, Ackermann PW, et al.** Increased fracture risk in patients with rheumatic disorders and other inflammatory diseases—a case-control study with 53,108 patients with fracture. J Rheumatol 2010;37:2247–50.

36. **Caron T, Bransford R, Nguyen Q, et al.** Spine fractures in patients with ankylosing spinal disorders. Spine (Phila Pa 1976) 2010;35:e458–64.

37. **Backhaus M, Citak M, Kälicke T, et al.** Spine fractures in patients with ankylosing spondylitis: an analysis of 129 fractures after surgical treatment. Orthopade 2011;49:917–20, 922-4.

38. **Ahn NA, Ahn MU, Nallamshetty L, et al.** Cauda equina syndrome in ankylosing spondylitis (the CES-AS Syndrome): meta-analysis of outcomes after medical and surgical treatments. J Spinal Disord 2001;14:427–43.

39. **Liu CC, Lin YC, Lo CP, et al.** Cauda equina syndrome and dural ectasia: rare manifestations in chronic ankylosing spondylitis. Br J Radiol 2011;84:e123–5.

40. **Ea HK, Lioté F, Lot G, et al.** Cauda equina syndrome in ankylosing spondylitis: successful treatment with lumboperitoneal shunting. Spine (Phila Pa 1976) 2010;35:e1423–9.

41. **Cornec D, Devauchelle Pensec V, Joulin SJ, et al.** Dramatic efficacy of infliximab in cauda equina syndrome complicating ankylosing spondylitis. Arthritis Rheum 2009;60:1657–60.