ORIGINAL ARTICLE

Hemiplegic Shoulder Pain Syndrome: Frequency and Characteristics During Inpatient Stroke Rehabilitation

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ABSTRACT. Dromerick AW, Edwards DF, Kumar A. Hemiplegic shoulder pain syndrome: frequency and characteristics during inpatient stroke rehabilitation. Arch Phys Med Rehabil 2008;89:1589-93.

Objective: To clarify the pathophysiology of hemiplegic shoulder pain by determining the frequency of abnormal shoulder physical diagnosis signs and the accuracy of self-report.

Design: Prospective inception cohort.

Setting: Academic inpatient stroke rehabilitation service.

Participants: Consecutive admissions (N=46) to stroke rehabilitation service.

Interventions: Not applicable.

Main Outcome Measures: The Neer test, Speed test, acromioclavicular shear test, Rowe test, and palpation for point tenderness.

Results: Participants were enrolled at a mean time to evaluation of 18.9±14.1 days after stroke. Weakness of shoulder flexion, extension, or abduction was present in 94% of subjects, and neglect was found in 29%. Pain was present by self-report in 37%. The most common finding, which was found in nearly all persons with abnormalities in the study physical examination maneuvers, was bicipital tendon tenderness (54%), followed by supraspinatus tenderness (48%). The Neer sign was positive in 30%; 28% had the triad of bicipital tenderness, supraspinatus tenderness, and the Neer sign. Self-reported pain was a poor predictor of abnormalities elicited on the examination maneuvers, even in those without neglect.

Conclusions: Our data implicate 2 vertical stabilizers of the humerus in early onset hemiplegic shoulder pain, the long head of the biceps and the supraspinatus. Our results also suggest that simple questioning of stroke rehabilitation inpatients about shoulder pain may not be adequate for clinical care or research purposes, even in the absence of neglect.

Key Words: Cerebrovascular accident; Joint diseases; Physical examination; Rehabilitation; Shoulder; Shoulder pain; Stroke.

IF RECENT SCIENTIFIC FINDINGS regarding activity dependent neuroplasticity and recovery[1,2] are clinically relevant, then the hemiplegic shoulder pain syndrome takes on new importance in stroke rehabilitation. The presence of pain discourages movement and might hinder recovery. The effectiveness of any motor restoration technique will be diminished if patients cannot tolerate active or passive shoulder movements, and these people may be deprived of recovery they might otherwise have had. Thus, hemiplegic shoulder pain will need to be effectively prevented or treated so that it does not become a practical roadblock to implementation of newer upper-extremity rehabilitation strategies.

The reported frequency of hemiplegic shoulder pain syndrome varies from 5% to 84% of stroke patients.[3-5] Despite the high frequency of hemiplegic shoulder pain syndrome, the literature is full of conflicting reports about the epidemiology, risk factors, and etiology of hemiplegic shoulder pain syndrome. Symptoms can appear in the first weeks after the stroke, or develop much later.[6,7] Risk factors for hemiplegic shoulder pain syndrome identified in the literature include glenohumeral subluxation, hemineglect, spasticity, flaccidity, and prior shoulder pathology.[8-11] Several clinical diagnoses have been proposed as causes of hemiplegic shoulder pain syndrome, including rotator cuff tendonitis or tears, subacromial bursitis, bicipital tendonitis, diffuse shoulder pain or adhesive capsulitis, brachial neuralgias, sympathetically mediated pain, and referred pain.[12-17] The presence of hemiplegic shoulder pain syndrome is associated with poorer motor recovery, though a causal link has not been made.[5,8]

One approach to clarifying hemiplegic shoulder pain syndrome would be to determine which shoulder structures act as pain generators at any given time after stroke. Objectively classifying the symptoms experienced by persons with hemiplegic shoulder pain syndrome would be an important step toward understanding the pathophysiology of this condition. Ideally, such an assessment would be reliable, simple, and done at the bedside to minimize expense and time away from therapies.

We have recently shown interobserver reliability of an examination of many of the soft tissue structures of the shoulder joint commonly thought to participate in pain generation after stroke.[18] We now describe the use of this evaluation to catalog the clinical phenomena of hemiplegic shoulder pain syndrome involving these structures during inpatient stroke rehabilitation. We hypothesized that patients would accurately self-report

List of Abbreviations

ACJ	acromioclavicular joint
MAS	Modified Ashworth Scale
MRC	Medical Research Council
NIHSS	National Institutes of Health Stroke Scale

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when they had elicitable pain, and that we could sort them into different subgroups based on which soft tissue structures were found to be involved on physical examination. Understanding the different subgroups would lead to a deeper understanding of the pathophysiology of hemiplegic shoulder pain syndrome and thus toward improved treatment.

METHODS

This was a prospective cross-sectional study done in an academic stroke rehabilitation program, and was approved by the Washington University Human Studies Committee. All persons admitted for stroke rehabilitation during the 3-month study period were included in this study, except those who refused, were unable to follow 1-step commands, or could not provide informed consent.

Each consenting subject was interviewed and examined within 2 weeks of rehabilitation admission. This was a specialty academic service, and particular attention was paid to staff training and equipment to minimize inadvertent hemiplegic shoulder trauma. Upper-extremity care for hemiparesis was the routine provided on the service; this consisted primarily of arm boards, passive and active range of motion, and compression gloves in cases with substantial dependent edema. Slings were used only during mobility training sessions in the therapy gym. All staff was instructed in proper transfer techniques, and gait belts or lift devices were used for transfers.

Each assessment was performed by a single physiatrist. They interviewed subjects for self-report of handedness and for any history of stroke or shoulder pain. The interview also included a pain questionnaire noting the presence or absence of pain, location, intensity, radiation, and pain at rest versus end of range was completed. Each subject was interviewed by the investigator, who recorded pain intensity and location by using a visual analog scale for pain and a diagram of the shoulder.[19] To rate neglect, the NIHSS neglect item was used; a score of 0 indicates no neglect, a score of 1 indicates either visual or somatosensory neglect, and a score of 2 indicates both somatosensory and visual neglect.[20] Shoulder strength in flexion, abduction, and extension was measured by using the MRC scale.[21] Hypertonicity in shoulder flexion and abduction and elbow flexors were measured with the MAS.[22] Hypertonicity was present if the tested muscle group had a score of at least 1 on the MAS.

The result of the interobserver reliability study has been reported elsewhere.[18] Physical examination of the affected shoulder began with a structured musculoskeletal examination to identify sites of tenderness on palpation.[23] Specific sites were palpated. Anteriorly, the tendon of the long head of the biceps was palpated between the lesser and greater tuberosities of the humeral head. The supraspinatus tendon was palpated anteriorly over its insertion at the greater tuberosity of the humerus, with the arm at 30° of shoulder extension. The subacromial area was examined by palpating the gap between the acromial process and head of the humerus on the superolateral aspect of shoulder. Pain in the subacromial region is usually attributed to inflammation of the subacromial bursa. The ACJ, coracoid process, and surrounding soft tissues were also examined for any localized or diffuse tenderness. Diffuse tenderness was defined as generalized shoulder girdle tenderness without localizing features.

Four physical diagnosis maneuvers were tested, described in detail in a previous article.[18] In the Neer impingement test,[24] the subject's arm was elevated through forward flexion by the examiner passively bringing greater tuberosity against the anteroinferior border of the acromion. An abnormal finding is the provocation of pain; such provocation is said to be indicative of impingement of the rotator cuff or injury to the supraspinatus muscle. In the Speed test,[25] the examiner actively resisted the shoulder elevated in forward flexion at the plane of the scapula in a completely extended elbow with the forearm medially rotated by the patient. Pain in the bicipital groove is said to be indicative of bicipital tendon involvement. The acromioclavicular shear test[26] is said to indicate pathology at the ACJ. This maneuver is performed with the subject sitting; the examiner cups his/her hands anteriorly on the clavicle and posteriorly on the spine of scapula. Squeezing the heel of the hands together elicits pain in the presence of ACJ inflammation. Finally, we used the Rowe test to show multidirectional instability in the shoulder.[25] In this test, the patient was seated in bed with the waist flexed at a 45° angle while the examiner held the head of the humerus by placing 1 hand over the shoulder so that the index and the middle fingers sat over the anterior aspect of the humeral head and the thumb on the posterior aspect of the humeral head. The examiner then exerted anterior and posterior force to elicit instability in either direction. For inferior instability, traction was applied vertically to elicit a sulcus sign.

Data were entered into SPSS[a] for Windows. Descriptive statistics were computed for all variables. Chi-square analyses were used to determine significant differences between groups. An a priori P value of less than or equal to .05 was used as the criterion for significance.

Table 1: Participant Characteristics (N=46)

Characteristics	Values
Age (y)	57.30±25.20 (33−78)
Male subjects	24 (52)
Prior nondisabling shoulder pain	7 (15)
Prior stroke	13 (28)
Stroke location	
Left	24 (52)
Right	22 (48)
Dominant side affected	23 (50)
Shoulder weakness present	
Flexion	39 (85)
External rotation	40 (87)
Abduction	43 (94)
Hypertonicity present	
Shoulder flexion	30 (66)
Shoulder abduction	30 (66)
Elbow flexion	32 (70)
Self-reported pain	17 (37)
Neglect	13 (28)
Sensory loss	4 (9)

NOTE. Values are mean ± SD (range) or n (%).

RESULTS

Evaluations were completed in all 46 subjects who met entry criteria. Five other patients admitted for stroke rehabilitation during the study period were not included: 3 were unable to participate due to severe aphasia, 1 patient refused, and one was transferred back to the acute medical floor for medical complications. The mean time from stroke onset to the first evaluation was 18.9±14.1 days.

Table 1 presents the demographic characteristics of the sample. Fifty percent of all strokes involved the dominant hand; all but 2 subjects reported right hand dominance prior to stroke. Thirteen (28%) subjects reported a history of a prior stroke, and 7 (15%) self-reported preexisting shoulder pain on the affected side. Using the NIHSS neglect item, 71% of subjects were

Table 2: Frequency of Physical Examination Findings

Examination Finding	Frequency
Tenderness on palpation	
Bicipital	25 (54)
Supraspinatus	22 (48)
Subacromial	12 (26)
ACJ	3 (7)
Diffuse	1 (2)
Positive physical examination maneuver	
Neer test	22 (48)
Rowe test	6 (13)
Acromioclavicular shear test	5 (11)
Speed test*	4 (9)

NOTE. Values are n (%).
*Thirty-three percent of subjects could not cooperate due to weakness.

rated as having no neglect, 21% with either visual or somatosensory neglect, and the remainder (8%) were found to have both visual and somatosensory neglect. Weakness of shoulder flexion, extension, or abduction was found in 94% of subjects using the MRC muscle grading scale; the frequencies for each muscle group can be found in table 1. Hypertonicity on passive shoulder flexion or abduction was found in 70% of subjects, measured by the MAS.

Pain was reported on interview by 17 (37%) of 46 subjects; 7 of these had pre-existing pain. On the interview, the most frequently reported location of pain was anterior, found in 14 (30%) of the 46. Two subjects reported pain at rest, and the remaining 15 reported pain only with passive movement. Pain radiated down the arm in 12 subjects and to the chest in 1 subject. No subjects reported radiation of pain to the neck or back.

Table 2 shows the frequency of tenderness elicited by palpation. The most common finding, bicipital tendon tenderness on the affected side (54%), was found in all persons with pain on palpation, suggesting that the long head of the biceps was involved in all subjects with pain elicited by palpation.

Table 2 also shows that 25 subjects were positive on 1 or more of the physical diagnosis maneuvers; the majority had only 1 of the 4 signs present. The largest group (n=22) had the Neer sign without any of the other 3 physical diagnosis maneuvers being positive. There were no other obvious clusters of signs. The Speed test for bicipital tendonitis was present in 9%, but the maneuver could not be used in 33% of subjects because of weakness.

Interestingly, inspection of individual data showed that the single largest constellation of findings was the triad of bicipital tenderness, supraspinatus tenderness, and shoulder impingement as indicated by a positive Neer sign. This triad was found in 13 of 46 participants, representing 50% of subjects with elicited pain, and 28% of all study participants.

We compared the frequency of self-reported pain to bicipital tenderness elicited by palpation to determine whether subjects might underreport pain (table 3). The majority of patients had hypertonicity at the elbow and shoulder, as well as weakness in the major shoulder muscle groups. The frequency of self-reported pain was significantly lower than the frequency of bicipital tenderness elicited by palpation. As an example of the difficulty or relying solely on self-report, we found that 38% of the patients who did not report pain on the interview had bicipital tenderness (χ^2=8.5, P<.004). Similar results were found for rotator cuff tenderness (χ^2=8.9, P<.003) and Neer sign (χ^2=12.6, P<.004). Using bicipital tenderness elicited on palpation as the criterion standard, the sensitivity of self-reported pain was .56 and the specificity was .86. Negative and positive predictive values were .62 and .82, respectively. The presence of neglect was not significantly associated with underreported pain (χ^2=2.11, P=.15). Patients with bicipital tenderness were also significantly more likely to have rotator cuff (χ^2=12.82, P=.001) and subacromial joint tenderness (χ^2=9.11, P=.003) as well.

DISCUSSION

The most scientifically promising motor restoration strategies require active or passive movement to improve upper-extremity motor function. The applicability of treatments currently under study such as constraint-induced movement

Table 3: Characteristics of Patients With Pain on Palpation (tenderness) Elicited on Physical Examination

Variable	Bicipital Tenderness (n=25)	No Bicipital Tenderness (n=21)	χ^2 (P)
Prior stroke	5 (20)	8 (38)	1.84 (.18)
Dominant side affected	15 (60)	8 (38)	2.19 (.14)
Self-reported pain	14 (56)	3 (14)	8.52 (.004)
Prior shoulder pain	7 (28)	0 (0)	6.93 (.008)
Neglect	19 (76)	14 (67)	0.53 (.77)
Hypertonicity present			
Shoulder flexion	17 (68)	13 (62)	.254 (.88)
Shoulder abduction	17 (68)	13 (62	.254 (.88)
Elbow flexion	17 (68)	6 (29)	.093 (.95)
Shoulder weakness present			
Abduction	18 (72)	16 (76)	4.90 (.08)
Flexion	18 (72)	16 (76)	4.29 (.12)
External rotation	15 (64)	7 (33)	3.48 (.17)
Rotator cuff tenderness	18 (72)	4 (19)	12.83 (<.001)
Subacromial tenderness	11 (44)	1 (5)	9.11 (.003)
ACJ tenderness	3 (12)	0 (0)	2.67 (.10)
Positive acromioclavicular shear test	4 (16)	1 (5)	1.49 (.36)
Positive Neer test	16 (64)	6 (29)	5.74 (.02)
Positive Rowe test	4 (16)	2 (10)	0.42 (.67)

therapy,[27,28] robotics,[29,30] and functional electric stimulation[31] will be limited if the hemiplegic shoulder pain syndrome cannot be better managed. Pain on movement will discourage upper-extremity use, and may cause patients to use their arm in productive activity less than their motor capacity or future assistive technologies would otherwise allow.

The syndrome of hemiplegic shoulder pain syndrome remains poorly understood. In many published studies, the syndrome is often treated as a unitary condition, as if the pathophysiology and sources of pain within the shoulder were no different from patient to patient. The validity of this assumption is unknown. Many studies have indirectly addressed pathophysiology by evaluating possible risk factors for shoulder pain[6,9,11,32,33]; we now extend their findings by using standard and reliable physical diagnosis techniques to specifically identify pain generators in the hemiplegic shoulder. A newly published study with different goals and a different design showed similar rates of self-reported pain and abnormal physical findings.[34]

The data in tables 2 and 3 implicate the long head of the biceps and the rotator cuff (particularly the supraspinatus insertion) as common pain generators in early-onset hemiplegic shoulder pain syndrome. Biceps tenderness, supraspinatus tenderness, and Neer sign were found far more frequently than other signs, and the triad occurred together in 50% of subjects with elicited pain and 28% of all participants. The function of the long head of the biceps tendon is poorly understood (reviews[35,36]) but the most widely accepted roles for this structure are to center the humeral head in the glenoid fossa and to reduce vertical translations.[37] The biceps plays an increasingly important role in glenohumeral stabilization as the shoulder becomes more unstable,[38] such as in the case of hemiplegia, and this may explain why the most frequent sign was bicipital tenderness. Like the long head of the biceps, the supraspinatus is a vertical stabilizer of the glenohumeral joint. That 2 vertical stabilizers are so frequently involved supports Cailliet's contention[7] that downward stretching of the joint capsule and surrounding musculotendinous structures causes injury and thus pain. Shoulder impingement, common in the elderly, is known to injure the supraspinatus tendon,[39] and may also contribute to the high prevalence of supraspinatus findings in our sample. Pathologic findings in other portions of the shoulder complex were far less frequent than those involving the long head of the biceps and the supraspinatus tendon. Because this study used a single time point early after stroke, it does not address the temporal sequence of shoulder pathology after stroke, but suggests that these other structures become involved, if at all, only later in the process. Our results are consistent with 2 other studies that attempt to assign a specific etiology for hemiplegic shoulder pain syndrome: a report that 50% of those with late hemiplegic shoulder pain syndrome experience improvement in shoulder pain after an injection of local anesthetic in the subacromial space[16] and a shoulder arthrography study in which 33% to 40% of persons with late hemiplegic shoulder pain syndrome had rotator cuff tears.[17]

Our hypothesis that self-report would correspond to physical findings was not supported. We found that patient self-reports of pain significantly underestimated the extent of pain found on physical examination, even in participants without visuospatial neglect. Thus, simply questioning patients about shoulder pain in the early stages may not be adequate for clinical care or for future studies of the pathophysiology or treatment of hemiplegic shoulder pain. Based on the data from our sample, if one were to rely only on patient reports of pain, almost 40% of those who denied pain would subsequently show pain on physical examination. We conclude that in the clinical setting, simple questioning of patients may not be sufficient, and that in the research setting, clinical examinations performed by skilled raters will be important.

Most studies first evaluate patients many weeks after initiation of stroke rehabilitation[33,40]; by then, the primary causes of the pain may be obscured by common secondary complications such as contracture or trauma. One goal of this study was to gain a better understanding of the beginnings of hemiplegic shoulder pain syndrome, at a time when the underlying pathophysiology would be least obscured by secondary complications. Our data confirm that in many people, hemiplegic shoulder pain syndrome is present soon after stroke. More than a third of our subjects reported pain on evaluation, and half had pain elicited on physical examination. Our results are consistent with those reported by Wanklyn et al,[40] who found that nearly 20% of their subjects reported that pain began immediately after stroke. Thus, a more definitive study of the pathophysiology of hemiplegic shoulder pain syndrome will need to begin earlier than the mean of 19 days used in this study, using a larger sample combined with imaging and electrophysiological data. Clinically, our results suggest that for many people, interventions should be implemented early after stroke after careful clinical examination of the affected shoulder.

Study Limitations

This was a cross-sectional study design, and only a single time point can be evaluated. The sample was obtained at a single center; although it is reflective of this center, it may differ from the stroke rehabilitation populations at other centers. A larger sample would have permitted more statistical analysis.

CONCLUSIONS

Our results imply a role for the vertical stabilizers of the shoulder in early-onset hemiplegic shoulder pain. We found that simple questioning of patients was inadequate to reliably identify those with abnormalities on physical examination, even when hemispatial neglect was not present.

References

1. Nudo RJ. Mechanisms for recovery of motor function following cortical damage. Curr Opin Neurobiol 2006;16:638-44.
2. Good DC. Stroke: promising neurorehabilitation interventions and steps toward testing them. Am J Phys Med Rehabil 2003;82(10 Suppl):S50-7.
3. Dromerick AW, Reding MJ. Medical and neurological complications during stroke rehabilitation. Stroke 1994;25:358-61.
4. Roy CW. Shoulder pain in hemiplegia: a literature review. Clin Rehabil 1988;2:35-44.
5. Roy CW, Sands MR, Hill LD. Shoulder pain in acutely admitted hemiplegics. Clin Rehabil 1994;8:334-40.
6. Poulin de Courval L, Barsauskas A, Berenbaum B, et al. Painful shoulder in the hemiplegic and unilateral neglect. Arch Phys Med Rehabil 1990;71:673-6.
7. Cailliet R. The shoulder in hemiplegia. Philadelphia: FA Davis; 1980.
8. Gamble GE, Barberan E, Laasch H, Bowsher D, Tyrrell PJ, Jones AK. Poststroke shoulder pain: a prospective study of the association and risk factors in 152 patients from a consecutive cohort of 205 patients presenting with stroke. Eur J Pain 2002;6:467-74.
9. Zorowitz RD, Hughes MB, Idank D, Ikai T, Johnston MV. Shoulder pain and subluxation after stroke: correlation or coincidence? Am J Occup Ther 1996;50:194-201.
10. Bohannon RW, Andrews AW. Shoulder subluxation and pain in stroke patients. Am J Occup Ther 1990;44:507-9.

11. Bohannon RW, Larkin PA, Smith MB, Horton MG. Shoulder pain in hemiplegia: statistical relationship with five variables. Arch Phys Med Rehabil 1986;67:514-6.
12. Braus DF, Krauss JK, Strobel J. The shoulder-hand syndrome after stroke: a prospective clinical trial. Ann Neurol 1994;36:728-33.
13. Davies SW, Petrillo CR, Eichberg RD, Chu DS. Shoulder-hand syndrome in a hemiplegic population: a 5-year prospective study. Arch Phys Med Rehabil 1977;58:356.
14. Lo SF, Chen SY, Lin HC, Jim YF, Meng NH, Kao MJ. Arthrographic and clinical findings in patients with hemiplegic shoulder pain. Arch Phys Med Rehabil 2003;84:1786-91.
15. Kaplan PE, Merideth JT, Betts HB. Stroke and brachial plexus injury: a difficult problem. Arch Phys Med Rehabil 1977;58:415-8.
16. Joynt RI. The source of shoulder pain in hemiplegia. Arch Phys Med Rehabil 1992;73:409-13.
17. Najenson T, Yacbocih E, Pikielni S. Rotator cuff injury in shoulder joints of hemiplegic patients. Scand J Rehabil Med 1971;3:131-7.
18. Dromerick AW, Kumar A, Volshteyn O, Edwards DF. Hemiplegic shoulder pain syndrome: interrater reliability of physical diagnosis signs. Arch Phys Med Rehabil 2006;87:294-5.
19. Melzack R. The McGill pain questionnaire: major properties and scoring methods. Pain 1975;1:277-99.
20. Brott T, Adams HP, Olinger CP, et al. Measurement of acute cerebral infarction: a clinical examination scale. Stroke 1989;20:864-70.
21. DeMyer WE. Technique of the neurologic examination. New York: McGraw-Hill; 2004.
22. Brashear A, Zafonte R, Corcoran M, et al. Inter- and intrarater reliability of the Ashworth Scale and the Disability Assessment Scale in patients with upper-limb poststroke spasticity. Arch Phys Med Rehabil 2002;83:1349-54.
23. Hoppenfeld S. Physical examination of the shoulder: physical examination of the spine and extremities. East Norwalk: Appleton-Century-Crofts; 1976. p 1-34.
24. Neer CS, Welsh RP. The shoulder in sports. Orthop Clin N Am 1997;8:583-91.
25. Rowe CR. The shoulder. Edinburgh: Churchill Livingstone; 1988.
26. Davies GI, Gould IA, Larson RL. Functional examination of the shoulder girdle. Phys Sports Med 1981;9:82-104.
27. Wolf SL, Winstein CJ, Miller JP, et al; EXCITE investigators. Effect of constraint-induced movement therapy on upper extremity function 3 to 9 months after stroke: the EXCITE randomized clinical trial. JAMA 2006;296:2095-104.
28. Dromerick AW, Lang CE, Powers WJ, et al. Very Early Constraint Induced Movement Therapy (VECTORS): phase II trial results [abstract]. Stroke 2007;38:465.
29. Lum PS, Burgar CG, Shor PC. Evidence for improved muscle activation patterns after retraining of reaching movements with the MIME robotic system in subjects with post-stroke hemiparesis. IEEE Trans Neural Syst Rehabil Eng 2004;12:186-94.
30. Volpe BT, Ferraro M, Lynch D, et al. Robotics and other devices in the treatment of patients recovering from stroke. Curr Atheroscler Rep 2004;6:314-9.
31. Chae J, Fang ZP, Walker M, Pourmehdi D. Intramuscular electromyographically controlled neuromuscular electrical stimulation for upper limb recovery in chronic hemiplegia. Am J Phys Med Rehabil 2001;80:935-41.
32. Barreca S, Wolf SL, Fasoli S, Bohannon RW. Treatment interventions for the paretic upper limb of stroke survivors: a critical review. Neurorehabil Neural Repair 2003;17:220-6.
33. Ikai T, Tei K, Yoshida K, Miyano S, Yonemoto K. Evaluation and treatment of shoulder subluxation in hemiplegia. Am J Phys Med Rehabil 1998;77:421-6.
34. Rajaratnam BS, Venketasubramanian N, Kumar PV, Goh JC, Chan YH. Predictability of simple clinical tests to identify shoulder pain after stroke. Arch Phys Med Rehabil 2007;88:1016-21.
35. Paynter KS. Disorders of the long head of the biceps tendon. Phys Med Rehabil Clin N Am 2004;15:511-28.
36. Levangie PK, Norkin CC. The shoulder complex: joint structure and function. Philadelphia: FA Davis; 2001. p 196-225.
37. Pagnani M, Deng XH, Warren RF, Torzilli PA, O'Brien SJ. Role of the long head of the biceps brachii in glenohumeral stability: a biomechanical study in cadavera. J Shoulder Elbow Surg 1996;5:255-62.
38. Itoi E, Kuechle DK, Newman SR, Morrey BF, An K. Stabilising function of the biceps in stable and unstable shoulders. J Bone Joint Surg Br 1993;75:546-50.
39. Chang WK. Shoulder impingement syndrome. Phys Med Rehabil Clin N Am 2004;15:493-510.
40. Wanklyn P, Forster A, Young J. Hemiplegic shoulder pain (HSP): natural history and investigation of associated features. Disabil Rehabil 1996;18:497-501.

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