Diagnosis and Treatment of the Hemiplegic Patient with Brachial Plexus Injury

(electromyograph, flaccidity, atrophy, brachial plexus, cerebrovascular accident)

Judy Meredith

Brachial plexus injury was observed as a complication in 5 of 12 hemiplegic patients admitted over a 3-week period to an inpatient unit of the Rehabilitation Institute of Chicago. These patients exhibited unusual patterns of muscle atrophy and return of function in the impaired upper extremity. Occupational therapists may play an important part in the diagnosis and treatment of this complication of hemiplegia by promptly recognizing its subtle clinical signs and instituting appropriate therapy. Electromyography may be recommended to confirm this diagnosis.

The treatment of choice is to maintain correct positioning of the limb both day and night, to use facilitation techniques for specific muscles in order to prevent atrophy, and to maintain passive range of motion as much as possible. Prevention of brachial plexus injury depends largely on the education of patient, family, and staff as to the potential hazards to a frail extremity that has no protective responses.

Ginny Taft

Reports of upper extremity complications of hemiplegia exist in the literature (1-3), but reports regarding the prevalence of upper trunk brachial plexus injury (BPI) in hemiplegic patients do not.

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The complication of BPI often remains undiagnosed because its symptoms are masked by the overlying hemiplegia. Health professionals need to become aware of the clinical symptoms and treatment of BPI because the condition may prolong or hinder the recovery of hemiplegic patients if unrecognized. This paper reports a study of five hemiplegic patients demonstrating clinical signs of BPI. It includes their initial electromyographic (EMG) evaluation, inpatient treatment, and physical and EMG re-evaluations 8 months later.

Criteria

Criteria for the diagnosis of BPI are derived from both physical examination and EMG data. Physical examination criteria are the following: flaccidity and atrophy of supraspinatus, infraspinatus, deltoïd and biceps muscles in the affected upper extremity (see Figures 1 and 2) with increased muscle tone or movement in the distal muscles of the same extremity. The electromyographic criterion for the diagnosis is the finding of fibrilla-
Figure 1 Hemiplegia without brachial plexus injury.

Figure 2 Hemiplegia and secondary brachial plexus injury. Note atrophy of supraspinatus and infraspinatus muscles on the affected side.

tion potentials in these muscles that are innervated by the upper trunk of the brachial plexus. Generalized or nonspecific fibrillations throughout the upper extremity in a hemiplegic patient are not diagnostic of BPI (4, 5).

Identification
Twelve hemiplegic patients who had had a cerebral vascular accident (CVA) 1 to 4 months earlier were admitted during a 5-week period to an inpatient division of the Rehabilitation Institute of Chicago for continuing therapy of their residual paralysis. They ranged in age from 53 to 73 years, with a mean of 63. Five of the 12 patients demonstrated clinical evidence of brachial plexus injury on physical examination (Figure 2), and were evaluated further. This is a noteworthy incidence in a relatively short period of time. All five patients shared a diagnosis of hemiplegia with middle cerebral artery thrombosis syndrome.

Initial Evaluation
Initial physical and neurological examination included detailed sensory evaluations, routine laboratory studies, X-rays, EMGs, and other diagnostic tests as indicated. All five patients met both physical examination and EMG criteria for the diagnosis of upper trunk brachial plexus injury (see Table 1).

The occupational therapist’s initial examination of the involved upper extremity included measurement of hand edema, subluxation
Table 1: Comparison chart of five hemiplegic patients with brachial plexus injuries.

<table>
<thead>
<tr>
<th>Patient</th>
<th>CE</th>
<th>RS</th>
<th>SC</th>
<th>EK</th>
<th>SZ</th>
</tr>
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<tbody>
<tr>
<td>Sex</td>
<td>M</td>
<td>M</td>
<td>F</td>
<td>F</td>
<td>M</td>
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<tr>
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<td>73</td>
<td>65</td>
<td>60</td>
<td>55</td>
<td>53</td>
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<td>R</td>
<td>R</td>
<td>R</td>
<td>R,L</td>
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<tr>
<td>Side of Hemiplegia</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
</tr>
<tr>
<td>Months after onset CVA (at admission)</td>
<td>4</td>
<td>1</td>
<td>1½</td>
<td>1</td>
<td>1 2</td>
</tr>
<tr>
<td>Months as Inpatient</td>
<td>1½</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>4½</td>
</tr>
<tr>
<td>Subluxation</td>
<td>Admission</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Discharge</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

Summary: Four out of five showed subluxed shoulders. Pt without subluxation was in most advanced stage of synergy development.

Atrophy of Supraspinatus, Infraspinatus, Deltoid, Biceps

Summary: Four out of five showed atrophy of involved muscles. Pt without atrophy was in most advanced stage of synergy development.

Hand Edema

Summary: All initially showed hand edema.

Brunnstrom Stage of Synergy

Summary: No consistent data; some showed conspicuous differences, others didn't.

Pain during P/R: Admission

Summary: Shoulder pain inconsistent at time of initial evaluation 1-4 mos. after CVA onset.

Sensation

Summary: No consistent data.

KEY — L — Left
| R — Right |
| Temp — Temperature |
| shldr — Shoulder |
| wr — Wrist |
| elb — Elbow |
| mos. — Months |
| + — Present |
| - — Absent |

P/R — Passive Range of Motion pt — Patient

(limbs, passive range of motion (noting pain), and muscle tone. Brunnstrom synergy development was evaluated by standard methods (6). Visual fields and and cognitive functions (including body schema, error recognition, orientation, motor planning, awareness of impaired side) were evaluated. Resulting deficits in self-care skills were also noted.

Treatment

Treatment involved passive and active range of motion exercises and limb positioning during ambulation, while in wheelchair, and during sleep. The affected upper extremity was given passive range of motion as tolerated and active assisted exercise in which gravity was monitored to prevent further traction injury to the upper trunk of the brachial plexus.

Positioning involved supporting the subluxed shoulder and providing a position for optimal healing. Ideally, the affected upper extremity was in a position of 45° external rotation, 90° elbow flexion, and 0° forearm supination. In the wheelchair, mobile armrests and elevated armrests were constructed to maintain this position. Kagle "hemi" slings were used by patients with subluxed shoulders when ambulating in order to support the shoulder and to maintain limb position.

Since sleeping on the affected side risks compression and traction injuries to the upper trunk of the brachial plexus, patients were required to learn to sleep supine and roll to the unimpaired side. A wedge-shaped pillow (Figure 3)
that fits under the impaired arm was very helpful for maintaining a good sleeping position. The pillow was strapped to the trunk and maintained 45° shoulder abduction, 45° external rotation, 90° elbow flexion, and forearm in neutral position. The pillow kept the hand elevated and this helped to diminish edema. It also increased the patient’s ease in rolling to the unimpaired side. Other positioning devices, such as a modified airplane splint and an overhead sling attached to the bed, were tried but discontinued because traction could not be avoided as the patient turned in bed. Regular pillows were also tried but were not as successful in maintaining the ideal position during movements of sleep. All treatment was explained to staff, to nursing personnel involved in implementing the night positioning, to patients, and to their families in order to ensure maximum carryover of positioning and to avoid traction.

Re-evaluation
Two patients received a second EMG and physical re-examination 8 months after initial evaluation. On examination, one patient showed no muscle atrophy, was in stage 5 Brunnstrom synergy development, and had shoulder pain during passive range of motion. His second EMG documented a return of voluntary neuromuscular function and the absence of fibrillations in the involved supraspinatus, infraspinatus, deltoid and biceps muscles. Apraxia interfered with a functional evaluation of his affected upper extremity, although his wife stated that he used this extremity more than he had previously. It is noteworthy that this patient was the only one without a subluxed shoulder at the time of initial evaluation and was further advanced in

Figure 3 Position pillow used for night positioning of the hemiplegic patient with brachial plexus injury, which maintains the extremity in 45° shoulder abduction, 45° external rotation, 90° elbow flexion, and forearm in neutral position.
synergy development at initial evaluation than the remaining patients.

The other patient on physical re-examination continued to show shoulder subluxation and stage 2 Brunnstrom synergy development with movements or shoulder protraction, retraction, elevation, elbow flexion, and wrist extension. Atrophy was apparent in the supraspinatus, infraspinatus, and deltoid muscles. Pain was present during all shoulder movement and supination. Spasticity was noted in the right elbow flexors, extensors, and metacarpophalangeal extensors. The second EMG showed a return of voluntary neuromuscular function. This was consistent with the finding made by Rodriguez and Oester that “EMG evidence of recovery may precede clinical signs of recovery by eight weeks.” (7, p 331)

Discussion

These five patients had atypical patterns of recovery of muscle tone and function following hemiplegia because of middle cerebral artery thrombosis syndrome that led to the diagnosis of complicating upper trunc BPI. The hemiplegic patient without complications most commonly shows a course in which flaccidity is followed by spasticity (8), and in which return of function and muscle tone proceeds from proximal to distal muscle groups (9, 10). An atypical pattern, demonstrated by these five hemiplegic patients, includes flaccidity and atrophy of the supraspinatus, infraspinatus, deltoid, and biceps muscles in the impaired upper extremity (see Figures 1 and 2) with increased muscle tone or movement in the distal muscles of this extremity. This atypical pattern of muscle tone should alert the occupational therapy and nursing staffs to the possibility of complicating brachial plexus injury; this possible diagnosis should be decided by EMG.

Treatment was designed to prevent atrophy and further compression or traction injury to the paralyzed upper extremity. The most important aspects of treatment, therefore, consisted of education of nursing staff, occupational therapists, patients, and their families regarding proper care and positioning of the upper extremity. EMG re-evaluations performed on two of these patients appear promising: return of voluntary neuromuscular function was documented in muscles that had been denervated on initial examination.

A prospective study of randomly taken groups of patients with flaccid hemiplegia under controlled conditions would be necessary to evaluate the prevalence of this complication. In addition, such a study could evaluate critically the impact of this complication on the ultimate degree and rate of recovery from hemiplegia resulting from cerebrovascular accidents.

The present study, however, allows the following tentative, but practical conclusions:

1. BPI may be a more common occurrence following flaccid hemiplegia than is generally appreciated.

2. Similarly, although it is not known to what extent this complication impedes overall recovery of function in the hemiplegic upper extremity, such excess morbidity may be significant.

3. The hemiplegic patient with a complicating BPI follows an atypical progression of recovery. Such a progression is clinically recognizable.

REFERENCES


RELATED READINGS

Codman EA: The Shoulder, Boston, Thomas Todd Co., 1934