Inappropriate intrinsic muscle action in the rheumatoid hand

R. L. SWEZEY⁽¹⁾ AND D. S. FIEGENBERG⁽²⁾

From the ⁽¹⁾Department of Physical Medicine and Rehabilitation and the ⁽¹⁾ ⁽²⁾Section of Rheumatic Diseases and Clinical Immunology, Department of Medicine, University of Southern California School of Medicine

The classic appearance of the rheumatoid hand with swollen and often dislocated metacarpophalangeal (MCP) joints, atrophic intrinsic muscle compartments, ulnar deviating fingers, and variously hyperextended or flexed and swollen interphalangeal joints is easily recognized. The explanation of the mechanisms involved in the pathogenesis of the intrinsic muscle atrophy (exclusive of the atrophy of the intrinsic muscles of the median nerve innervated thenar group in the carpal tunnel syndrome) has not been generally accepted. This, no doubt, has been in part attributable to the paradox of the apparent increased intrinsic muscle activity (intrinsic plus of Bunnell) seen in the rheumatoid hand associated with obvious gross interosseous atrophy and weakness of hand function (Bunnell, Doherty, and Curtis, 1948; Brewerton, 1957; Flatt, 1959).

Boyes (1964) called attention to overactivity or 'spasm' of the intrinsic muscles. Bunnell showed that, as a consequence of this overactivity or intrinsic 'plus' activity occurring in spastic patients (pill-roller hand) and as a result of electrical stimulation, the finger when maximally extended at the MCP joint would be unable to fully flex at the proximal interphalangeal (PIP) joint. This restriction of motion was attributable to traction of the 'spastic' or tight intrinsics on their attachments to the extensor hood (Bunnell, 1953).

In the presence of abnormally mobile or diseased finger joints, this intrinsic 'plus' action, resulting from prolonged contraction or ischaemic or post-traumatic contracture, could lead not only to subluxation of the MCP joint but to hyperextension of the PIP joint as well (Boyes, 1964; Harris and Riordan, 1954; Flatt, 1968).

Thus the stage is set for the well-known swan-neck deformity (Fig. 1). That this deformity is not exclusively found in rheumatoid arthritis is well illustrated in one of Bunnell's cases of an ischaemia-induced intrinsic 'plus' deformity resulting from a Volkmann's contracture (Flatt, 1959, 1968). Since Bunnell's identification of the intrinsic 'plus' deformity there has arisen some difficulty in the

FIG. 1 'Swan-neck' deformity. Elongated spring represents active interosseous muscle contraction. Note hyperextension at proximal interphalangeal joint.

electromyographic documentation of spasm or active contraction of the intrinsic muscles.* Although various observers have noted electromyographic abnormalities in rheumatoid muscles, particularly in the interossei, these changes have not been characterized by evidence of persistent spontaneous

* (Backhouse, 1968; Steinberg and Wynn Parry, 1961; Morrison, Short, Ludwig, and Schwab, 1947; Mueller and Mead, 1952; Wozny and Long, 1966; Moritz, 1963; Wramner, 1950.)

involuntary motor unit discharge in the resting state or spasms. Vainio (1967) further observed that the intrinsic 'plus' deformity did not subside with general anaesthesia, and Tiselius (1969) made the same observation on patients receiving axillary blocks, thereby further refuting the concept of intrinsic muscle spasms.

The possibility that the intrinsic muscles became taut because of fibrosis and shortening secondary to rheumatoid myositis has stimulated considerable discussion (Brewerton, 1957; Flatt, 1959; Boyes, 1964; Harris and Riordan, 1954; Wozny and Long, 1966; Wramner, 1950; Vainio, 1967). Pathological changes in rheumatoid muscle characterized by small focal lymphocytic infiltrates are commonly seen, but are not specific and are not unusual in control muscle samples (Clawson, Noble, and Lufkin, 1947; Bunim, Sokoloff, Wilens, and McEwen, 1948; Horwitz, 1949; Kestler, 1949; Wegelius, Pasternack, and Kuhlback, 1969). Vainio (1967) was unable consistently to correlate intrinsic myositis with intrinsic atrophy and intrinsic plus deformity at surgery.

Electromyographic studies have been equally unsuccessful in consistently documenting myopathic changes in rheumatoid muscle, although evidence for myopathy (myositis) was commonly observed (Steinberg and Wynn Parry, 1961; Morrison and others, 1947; Mueller and Mead, 1952; Moritz, 1963). In summary, the frequency and extent of myositic involvement in the intrinsic muscles have been insufficient to explain the shortening and atrophy of these muscles in the rheumatoid hand.

The possibility suggested itself of the formulation of a unifying concept that could explain the intrinsic weakness and atrophy and the increased intrinsic muscle tension secondary to contracture without invoking a persistent muscle spasm or the need for a diffuse contracting rheumatoid myositis. It was postulated that painful stimuli originating during motion in the inflamed MCP joint reflexly induced an antalgic (pain-avoiding) contraction of the intrinsics, placing the MCP joint in a neutral position of mild to moderate flexion (Harris and Riordan, 1954; Petersén and Stener, 1959; Gardner, 1950; Palmer, 1958). Extension of the MCP joint and, therefore, stretching of the intrinsics would be avoided as an acquired pain-avoidance mechanism (Blockey, 1954). Prolonged inhibition of lengthening of the intrinsics would lead to their contracture in a shortened position and would thus passively increase tension on the extensor tendon during active extension of the MCP joint with the interphalangeal joints simultaneously extended (intrinsic plus) (Fig. 2). This would be accompanied by disuse atrophy of the interossei, again through reflex inhibition and acquired pain-avoidance movements (Lippmann and Selig, 1928; Ekholm, Eklund, and Skoglund, 1960;

FIG. 2 'Swan-neck' deformity. Coiled spring represents absence of muscle contraction. Interosseous tension is a result of failure of the shortened fibrotic muscle to elongate.

Swearingen and Dehner, 1964; Beswick, Blockey, and Evanson, 1955). The key to this hypothesis would entail the demonstration of evidence of inappropriate early contraction of the interossei during active MCP extension of the fingers with the interphalangeal joints in extension—a motion previously shown to be accompanied by little interosseous activity, the lumbricales playing the primary role of interphalangeal extensors (Thomas and Long, 1947; Landsmeer and Long, 1965; Long, 1968; Boivin, Wadsworth, Landsmeer, and Long, 1969; Close and Kidd, 1969). Support for the premise that pain in the MCP joints leads to the intrinsic contractures was obtained by the uniform presence of MCP inflammation or a prior history of such involvement in all cases in which we have observed this deformity.

Material and methods

Fifteen patients with active rheumatoid arthritis (ARA definite or classical) with tenderness on palpation over the MCP joints and a positive Bunnell's test for increased intrinsic tension were tested. All but two patients could actively fully extend the MCP joints. The age span in the

experimental group was 30 to 70 years (average 51) and there were three males and twelve females.

Ten control subjects without arthritis or painful joints including four males and six females ranging in age from 16 to 64 years (average 37 years) were tested in the same manner as the experimental group.

Electromyographic recordings were made with a Teca Electromyograph, Model B-2. Skin electrodes were placed over the mid-portion of the first interosseous muscle and the third dorsal interosseous muscle proximal to the lumbrical. Contact paste was applied and the electrodes were taped into position in a manner which would not restrain finger motion. The ground wire was similarly affixed to the mid-dorsum of the proximal part of the hands. Proper placement of the electrodes was confirmed by the brisk firing of motor units recorded on abduction or flexion of the tested finger.

The hand was placed at the edge of a table with the fingers relaxed and fully extended and with the MCP joints approaching full flexion (90°). The patient was asked slowly to extend the MCP joint (towards 0°) of the index finger, all four fingers, the middle finger, and again all four fingers, while maintaining extension in the interphalangeal joints and avoiding abduction at the MCP joints. Recordings were taken at 100 μ V/cm., utilizing the electrode over the first dorsal interosseous as the exploring electrode when that muscle was examined, while the electrode over the third dorsal interosseous muscle served as an indifferent electrode. The proximal attachments of the exploring and indifferent wires were then reversed for recording over the third dorsal interosseous muscle. A baseline of electrical silence on recording from the relaxed muscles was obtained with the hand in the initial testing position.

Results

The angle at which motor unit activity was first detected was measured on a protractor placed adjacent to the finger tested. The records are shown in Tables I and II.

To document these data more accurately, one patient (Case 14) and one control subject were studied by the method of Close and Kidd (1969). Plastic-coated copper wire electrodes 100 μ in diameter were implanted in the first lumbrical and the first dorsal interosseous muscles. The action potentials of these muscles were photographed simultaneously with the finger motion, utilizing a dual-lens rotating prism-shutter camera (Hycam). Representative segments of the 16 mm. film are shown in Figs 3 and 4 (overleaf).

Discussion

Electromyographic studies have clearly demonstrated the functional role of the intrinsic muscles (Backhouse, 1968; Landsmeer and Long, 1965; Long, 1968; Boivin and others, 1969; Close and Kidd, 1969). The dorsal interossei and the abductor digiti quinti are abductors of the fingers and the volar interossei are adductors (Backhouse, 1968.)

Table I Angle of metacarpophalangeal joint at onset of intrinsic activity. Control group.
Zero degrees represents full extension.

Control no.	Age (yrs)	Sex	Angle (°)	
			Index	Fourth
1	25	F	5	5
2	26	F	0	5
3	59	F	0	0
4	30	F	0	0
5	16	F	0	0
6	64	F	5	10
7	26	M	0	15
8	46	M	0	0
9	33	M	5	5
10	45	M	0	5

Table II Angle of metacarpophalangeal joint at onset of intrinsic activity. Experimental group.
Zero degrees represents full extension.

Patient no.	Age (yrs)	Sex	Angle (°)	
			Index	Fourth
1	58	F	20	20
2	72	F	30/5 ^[2]	30/10 ^[2]
3	54	F	30	30
4	52	F	15	30
5	30	F	10	20
6	45	F	10	10
7	38	F	20	30
8	64	F	5	5
9	39	F	10	20
10*	51	F	30	30
11†	58	F	0	20
12‡	34	F	0	5
13	70	M	30	30
14	57	M	30/15 ^[2]	30/20 ^[2]
15	48	M	10	30

* Patient unable to extend MCP joints beyond 10°.

† Postoperative silastic implant with uncorrected swan neck deformity of fourth finger.

‡ Residual MCP subluxation, but no pain or evidence of active synovitis at time of study.

[²] Examination repeated one month later at time when subjective and objective MCP synovitis was minimal.

The dorsal and volar interossei acting together flex the metacarpophalangeal joints when the interphalangeal joints are extended. By virtue of their attachments to the extensor hoods and, thus, to the long extensor tendons of the phalanges, the interossei (particularly palmar interossei) also assist the lumbricals in extending the proximal and middle phalanges (Backhouse, 1968; Landsmeer and Long, 1965; Long, 1968; Boivin and others, 1969).

Active extension of the MCP joints with the interphalangeal joints extended produces little if any activity in the interosseous muscles of the normal hand (Long, 1968; Close and Kidd, 1969).

The findings were most consistent and definitive in the recordings from the first dorsal interossei. This is presumably due to the fact that skin electrodes,

FIG. 3 LEFT UPPER *Control. Relaxed (beginning tension in the lumbrical can be seen).*
1 Dis = first dorsal interosseus skin electrode. 1 D = first dorsal interosseus muscle implanted electrode. Lumb 1 = first lumbrical.
RIGHT UPPER *Patient. Relaxed. 60-cycle artefact in 1 Dis is present.*
LEFT LOWER *Control. Partial extension. Only the lumbrical* is showing motor unit action potential.*
RIGHT LOWER *Patient. Partial extension. Motor unit action potential in the lumbrical are present, and inappropriate activity in the interosseus is seen.*

although easy to apply and less traumatic than needle electrodes, do not as readily distinguish the lumbrical action from the lumbrical-like action of the palmar interossei (Long, 1968; Boivin and others, 1969; Andersson and Stener, 1959). As there is no palmar interosseus muscle underlying the first dorsal interosseus, and as the first dorsal rarely inserts into the extensor hood, the action of the first dorsal interosseus recorded by skin electrodes is less confused. This was confirmed in the single case in which electrodes were implanted into the first

dorsal, interosseus, the first lumbrical, and the skin over the first dorsal interosseus muscle, and simultaneously recorded by the method of Close and Kidd (1969) (Figs 3 and 4).

These observations were confirmed in our control subjects. The early and inappropriate activity of the interossei (contracting during partial extension of the MCP joints) noted in our rheumatoid subjects can

* The lumbrical is normally active during active interphalangeal extension.

FIG. 4 LEFT UPPER Control. Full extension. Motor unit action potentials are seen only in the lumbrical.
 RIGHT UPPER Patient. Full extension. Lumbrical and interosseous are active.
 LEFT LOWER Control. Attempted hyperextension. Interosseous and lumbrical are now both active.
 RIGHT LOWER Patient. Attempted hyperextension.

best be explained on the basis of a 'protective' pain reflex (Wramner, 1950; Petersén and Stener, 1959; Gardner, 1950; Palmer, 1958; Blockey, 1954; Lippmann and Selig, 1928; Ekholm and others, 1960; Swearingen and Dehne, 1964; Beswick and others, 1955). Evidence for reflex muscle activity due to excessive stretching or traction of ligaments was not found (Petersén and Stener, 1959; Andersson and Stener, 1959). Stimulation of pain fibres in ligamentous and capsular structures of a joint, however, has been shown to cause reflex contraction of the flexor muscles acting on that joint as well as inhibition of its extensors (Cohen and Cohen, 1956).

Wramner (1950) studied fifty cases of rheumatoid arthritis with pain and stiffness on joint motion and noted spontaneous electromyographic activity in the muscles undergoing passive stretch which could be relieved by sufficient shortening of the muscles. He called this phenomenon the 'pain-relief contraction'. In thirty patients with rheumatoid arthritis tested, no evidence of involuntary muscle activity could be demonstrated in muscles without these signs. Morrison and others (1947) noted a similar subsidence of persistent resting electromyographic activity in the muscles about a painful knee when it was supported on a cushion in a position of comfort.

In two of our patients (Cases 11 and 12) who had no pain in the MCP joint, there was no electromyographic activity during MCP extension. In Case 14 a repeat examination during partial remission related to chrysotherapy showed a marked reduction in the angle at which intrinsic activity was observed, again suggesting the importance of pain in the genesis of the interosseous muscle action potentials.

The possibility that the interosseous muscle activity reflected a stabilization action against ulnar drift was considered, but was not supported by the finding of interosseous activity occurring during the test conditions in five of our patients who had no evidence of ulnar deviation (Cases 1, 4, 5, 6, 9). Nor was there any correlation with interosseous activity during finger extension and the presence or absence of PIP joint disease. Cases 5, 6, and 9 had no clinical signs (redness, pain, tenderness, heat, or swelling) or radiological evidence of PIP joint disease and Cases 4 and 14 had *boutonnière* deformities, while Cases 11 and 13 had swan-neck deformities, and all of the other patients showed some clinical or radiological evidence of PIP joint disease.

Although it could be argued that there was an inconsistency in the 'protective action' of the intrinsic in preventing full extension of the MCP joints while acting as extensors of the PIP joints when the latter were also painfully involved, this is in fact what we have observed. It is explicable if one considers that all finger movements are delicately integrated and that therefore the long finger flexors would maintain the PIP joints in the antalgic partially flexed position, while the interossei and lumbricals maintained sufficient tension on the extensor mechanism during finger extension to permit the hand to open for grasping or pinching. In the presence of flexion contractures or pain limiting extension of the PIP joints, even less shortening of the intrinsic muscles would be required to induce an intrinsic 'plus' state, as the long finger extensor mechanism to which they attach would then be extended distally.

Vainio (1967) demonstrated at surgery the frequent presence of nodules and adhesions of interosseous tendons to the capsule of the MCP joints, and suggested that this was the mechanism by which the constraining force of the intrinsic was generated. It seems more likely, however, that the initial dynamic phase of this deformity may be related to the reflex intrinsic contractions stimulated by painful stretching of the inflamed rheumatoid MCP joint. Adhesions and secondary contractures would be presumed to form after the MCP motion had become restricted by the above reflex further reinforced by acquired pain-avoidance movements.

Summary

An abnormality of function of the intrinsic muscles associated with swan-neck deformity and metacarpophalangeal (MCP) subluxations in rheumatoid arthritis has long been appreciated, but not explained. No electromyographic evidence of spasm of these muscles or consistent evidence of sufficient myositis to explain the increased intrinsic muscle tension has been observed.

In eight consecutive rheumatoid subjects with active MCP joint involvement, inappropriate interosseous activity was demonstrated by electromyographic evidence of muscle contraction occurring at 10–30° of extension (0° = full extension) during active extension. No muscle action potentials were observed at more than 5° in the controls.

It is suggested that a protective MCP flexor response is induced in the interosseous muscles as a pain-avoidance reflex mechanism and that secondary fibrosis leads to a fixed shortening of the intrinsic muscles.

The authors wish to acknowledge the generous assistance of Dr. Robert Close and Mrs. Caroline Kidd in obtaining the simultaneous recordings of implanted electrodes and finger motions.

References

- ANDERSSON, S., AND STENER, B. (1959) *Acta physiol. scand.*, **48**, Suppl. 166, p. 27 (Experimental evaluation of the hypothesis of ligamento-muscular protective reflexes. II. A study in the cat using the medial collateral ligament of the knee joint).
- BACKHOUSE, K. M. (1968) *Ann. roy. Coll. Surg.*, **43**, 154 (The mechanics of normal digital control in the hand and an analysis of the ulnar drift of rheumatoid arthritis).
- BESWICK, F. B., BLOCKEY, N. J., AND EVANSON, J. M. (1955) *J. Physiol. (Lond.)*, **128**, 83P (Some effects of the stimulation of articular nerves).
- BLOCKEY, N. J. (1954) *J. Bone Jt Surg.*, **36-A**, 833 (An observation concerning the flexor muscles during recovery of function after dislocation of the elbow).
- BOIVIN, G., WADSWORTH, G. E., LANDSMEER, J. M. F., AND LONG, C. (1969) *Arch. phys. Med.*, **50**, 17 (Electromyographic kinesiology of the hand: muscles driving the index finger).
- BOYES, J. H. (1964) 'Bunnell's Surgery of the Hand', 4th ed., revised by J. H. Boyes, pp. 327–333. Lippincott, Philadelphia.
- BREWERTON, D. A. (1957) *Ann. rheum. Dis.*, **16**, 183 (Hand deformities in rheumatoid disease).

- BUNIM, J. J., SOKOLOFF, L., WILENS, S. L., AND MCEWEN, C. (1948) *Ibid.*, 7, 251 (Cellular aggregates in skeletal muscle of patients with rheumatic diseases other than rheumatoid arthritis).
- BUNNELL, S. (1953) *J. Bone Jt Surg.*, 35-A, 88 (Ischemic contracture, local, in the hand).
- , DOHERTY, E. W., AND CURTIS, R. M. (1948) *Plast. reconstr. Surg.*, 3, 424 (Ischemic contracture, local, in the hand).
- CLAWSON, B. J., NOBLE, J. F., AND LUFKIN, N. H. (1947) *Arch. Path.*, 43, 579 (Nodular inflammatory and degenerative lesions of muscles from four hundred and fifty autopsies).
- COHEN, L. A., AND COHEN, M. L. (1956) *Amer. J. Physiol.*, 184, 433 (Arthrokinetic reflex of the knee).
- CLOSE, R. J., AND KIDD, C. C. (1969) *J. Bone Jt Surg.*, 51-A, 1601 (The functions of the muscles of the thumb, the index, and long fingers).
- EKHOLM, J., EKLUND, G., AND SKOGLUND, S. (1960) *Acta physiol. scand.*, 50, 167 (On the reflex effects from the knee joint of the cat).
- EYLER, D. L., AND MARKEE, J. E. (1954) *J. Bone Jt Surg.*, 36-A, 1 (Anatomy and function of the intrinsic musculature of the fingers).
- FLATT, A. E. (1959) *Arthr. and Rheum.*, 2, 278 (Surgical rehabilitation of the arthritic hand).
- (1968) 'The Care of the Rheumatoid Hand', 2nd ed., pp. 35-42. Mosby, St. Louis.
- GARDNER, E. (1950) *Amer. J. Physiol.*, 161, 133 (Reflex muscular responses to stimulation of articular nerves in the cat).
- HARRIS, C., AND RIORDAN, D. C. (1954) *J. Bone Jt Surg.*, 36-A, 10 (Intrinsic contracture in the hand and its surgical treatment).
- HORWITZ, M. (1949) *Ann. rheum. Dis.*, 8, 258 (Muscle lesions in rheumatoid arthritis).
- KESTLER, O. C. (1949) *Ibid.*, 8, 42 (Histopathology of the intrinsic muscles of the hand in rheumatoid arthritis: a clinico-pathological study).
- LANDSMEER, J. M. F., AND LONG, C. (1965) *Acta Anat. (Basel)*, 60, 330 (The mechanism of finger control, based on electromyograms and location analysis).
- LIPPMANN, R. K., AND SELIG, S. (1928) *Surg. Gynec. Obstet.*, 47, 512 (An experimental study of muscle atrophy).
- LONG, C. (1968) *J. Bone Jt Surg.*, 50-A, 5, 973 (Intrinsic-extrinsic muscle control of the fingers).
- MORITZ, U. (1963) *Acta. rheum. scand.*, Suppl. 6, p. 65 (Electromyographic studies in adult rheumatoid arthritis).
- MORRISON, L. R., SHORT, C. L., LUDWIG, A. O., AND SCHWAB, R. S. (1947) *Amer. J. med. Sci.*, 214, 33 (The neuromuscular system in rheumatoid arthritis: electromyographic and histological observations).
- MUELLER, E. E., AND MEAD, S. (1952) *Amer. J. phys. Med.*, 31, 67 (The electromyogram in rheumatoid arthritis).
- PALMER, I. (1958) *Acta. chir. scand.*, 115, 312 (Pathophysiology of the medial ligament of the knee joint).
- PETERSÉN, I., AND STENER, B. (1959) *Acta physiol. scand.*, 48, Suppl. 166, p. 52 (Experimental evaluation of the hypothesis of ligamento-muscular protective reflexes).
- STEINBERG, V. L., AND WYNN PARRY, C. B. (1961) *Brit. med. J.*, 1, 630 (Electromyographic changes in rheumatoid arthritis).
- SWEARINGEN, R. L., AND DEHNE, E. (1964) *J. Bone Jt Surg.*, 46-A, 1364 (A study of pathological muscle function following injury to a joint).
- THOMAS, D. H., AND LONG, C. (1964) *Amer. J. med. Electron.*, 3, 96 (An electrogoniometer for the finger).
- TISELIUS, P. (1969) *Acta rheum. scand.*, Suppl. 14, p. 57 (Studies on joint temperature, joint stiffness, and muscle weakness in rheumatoid arthritis).
- VAINIO, K. (1967) *Ann. rheum. Dis.*, 26, 328 (Rheumatoid changes in the metacarpophalangeal region as a cause of Bunnell's sign).
- WEGELIUS, O., PASTERNAK, A., AND KUHLBÄCK, B. (1969) *Acta rheum. scand.*, 15, 257 (Muscular involvement in rheumatoid arthritis).
- WOZNY, W., AND LONG, C. (1966) *Arch. phys. Med.*, 47, 699 (Electromyographic kinesiology of the rheumatoid hand).
- WRAMNER, T. (1950) *Acta med. scand.*, 138, Suppl. 242, pp. 18-56 (Studies on the effect of neostigmine on muscular symptoms in rheumatoid arthritis).