Short report

Validation of a diagnostic sign in carpal tunnel syndrome

WEM PRYSE-PHILLIPS

From the Division of Neurology, Faculty of Medicine, Memorial University of Newfoundland, St John's, Newfoundland, Canada

SUMMARY Of three signs in carpal tunnel syndrome, Phalen's, Tinel's and the Flick sign, the last of these was the most valid and reliable. The presence of a positive Flick sign predicted electrodiagnostic abnormality in 93% of cases and had a false positive rate of under 5% among other neural lesions in the arm. The key question consists of an enquiry as to what the patient does with the affected hand at times when symptoms are at their worst; a flicking movement of the wrist and fingers demonstrated by the patient constitutes a positive response.

Patients with carpal tunnel syndrome adopt various manoeuvres to rid themselves of the distressing symptoms of acroparaesthesiae, pain, heaviness and numbness which they feel most acutely by night or on awakening.^{1,2} These include hanging the arm down beside the bed, slapping it against the thigh, massage using the unaffected hand, and shaking the wrist. The last of these has been commented upon but has not been emphasised as a diagnostic sign^{2-5,21} and, in several major reviews of the condition,^{1,3-12} it has been ignored. Noticing the frequency with which patients with carpal tunnel syndrome shake their wrists when describing how they react to symptoms at their worst, I assessed the frequency and diagnostic validity of this sign in a series of patients referred to a neuromuscular diagnostic clinic.

Patients and methods

All patients had been referred to a neuromuscular/EMG clinic for complaints of arm pain, weakness and/or paraesthesiae and sensory deficit and diagnosed by clinical and electrical means as suffering from carpal tunnel syndrome. These included all patients seen since 1978 and a further group retrospectively surveyed who had been seen since 1972. A sample of patients diagnosed as having other

Address for reprint requests: Dr WEM Pryse-Phillips, Division of Neurology, Memorial University of Newfoundland, St John's, Newfoundland, Canada A1B 3V6.

Received 3 November 1983 and in revised form 25 January 1984.
Accepted 30 January 1984.

Fig Male subject with bilateral carpal tunnel syndromes demonstrating Flick signs bilaterally.

median nerve pathologies, thoracic outlet syndrome, ulnar nerve lesions or cervical radiculopathy between 1979 and 1983 was included as a control group.

The Flick sign is characterised by a flicking movement of the wrist and hand, similar to that employed in shaking down a clinical thermometer, and demonstrated by the patient (fig) in response to such a question as "What do you actually *do* with your hand(s) when symptoms are at their worst?" which was the question posed in all cases.

Occasionally as a variant the wrist of the dependent arm is oscillated with the fingers semi-flexed.

The diagnostic criteria used were as follows:

1. Carpal tunnel syndrome

Clinical Symptoms of paraesthesiae, numbness and/or weakness in the hand in digits I–III or I–V, with or without hand and arm pain, usually with nocturnal or early morning accentuation, +/- clinical signs of thenar motor or median nerve territory sensory deficit.

Electrical Distal motor latency >4.5 ms or 1 ms R/L difference or 1.5 ms median/ulnar difference. Median SAP amplitude < ulnar (orthodromic) or <10 μ V or latency to onset >3.5 ms at 13 cm, in the absence of a Martin-Gruber anastomosis.

2. Cubital tunnel syndrome

Clinical Symptoms of hand weakness, +/- digit V (IV) hypoaesthesia, not extending into palm: and/or electrical signs of interosseous or hypothenar wasting, with proportionate weakness.

Electrical Eisen Score¹³ greater than 5/10.

3. Other median nerve pathologies

Clinical Digital neuropathy affecting digits I–III or arm pain/paraesthesiae without nocturnal predominance, or clinically apparent weakness of long forearm flexors, +/- palmar hypoaesthesiae. (Cases of anterior interosseous syndrome were excluded.)

Electrical EMG evidence of acute/chronic denervation in forearm flexor muscles, +/- delay in motor conduction speeds across the point above the wrist with absence of electrical evidence of median nerve compression at the carpal tunnel.

4. Thoracic outlet syndrome

Clinical Symptoms of arm pain and paraesthesiae mainly in the postaxial limb border increased by carrying or other manoeuvres elevating the shoulder and accompanied by hand (C8-T1) muscle weakness and wasting +/- Raynaud's phenomenon, supraclavicular bruit or positive Adson's test or other tests to obliterate the radial pulse.

Electrical¹⁴ Reduction of thenar muscle CMAP amplitude compared with hypothenar, and of ulnar SAP amplitudes compared with median, with F-Wave delay (median), with or without evidence of denervation of intrinsic hand muscles.

5. Cervical radiculopathy

Clinical Digital numbness/paraesthesiae, mainly nocturnal, with pain in centrifugal radicular distribution; objective radicular hypoaesthesiae and selective weakness of muscles innervated by appropriate roots; and features of cervical spine pathology (pain, grating, decreased range of movement, postural deformity etc).

Electrical Chronic denervation in cervical paraspinal muscles or radicular muscle neurogenic change +/- delayed median/ulnar F-wave responses.

Results

Among 505 subjects with carpal tunnel syndrome

Table Frequency of appearance of Flick sign in various pathologies

Diagnosis	(n)	(+) Flick	(%)	(-) Flick
Carpal tunnel syndrome	212	197	(93)	15
Other median n lesions	44	1	(2.3)	43
Ulnar n lesions	67	4	(6.3)	63
Thoracic outlet syndrome	41	3	(7.8)	38
Cervical spondylosis	32	0	(0)	32
(Clinical carpal tunnel syndrome, no electrical confirmation)	(5)		(100)	(0)
$\chi^2 = 313.9$ df = 4 p = <0.001				

(including 293 patients seen before 1978 and included for this statistic only), the right arm was affected alone in 154 (31%), the left alone in 115 (23%) and both were affected in 236 (47%). Bilateral involvement was more common in patients who were diabetic, hypothyroid or pregnant.

The diagnoses of patients surveyed and the frequency of occurrence of the Flick sign with these pathologies are recorded in the table.

The validity of the sign was measured by determining the false negative rate, that is, the proportion of patients with carpal tunnel syndrome who did not show the Flick sign: this was 15 of 212 (7.1%). Its reliability was assessed using the false positive rate, that is, the number of patients with sensorimotor problems in the arms not due to carpal tunnel syndrome who did display a Flick sign. This was eight of 176 (4.6%). The presence of a Flick sign was not related to age, sex, side affected or severity of symptoms, but the sign was less commonly seen in patients with carpal tunnel syndrome who were diabetic, hypothyroid or pregnant.

Given a patient with a neural problem affecting the arm, if the Flick sign is positive, the change of carpal tunnel syndrome as a cause is:

$$\frac{\text{CTS Flick (+)}}{\text{All Pathologies Flick (+)}} = \frac{197}{205} = 96\%$$

If the Flick sign is negative, the chance of carpal tunnel syndrome as a cause is:

$$\frac{\text{Flick (-) CTS}}{\text{Flick (-) All Pathologies}} = \frac{15}{191} = 7.9\%$$

Discussion

In many areas, electrodiagnosis is unavailable and a reliable clinical diagnostic test would be valuable for this common and distressing condition. A positive Tinel's sign was found in 89%,⁶ 60%,⁵ 45%,¹⁵ 44%,¹² 27%¹⁶ and 0%² of cases and may occur in normal subjects.¹⁷ Phalen's sign has been found to

be present in 73%,⁹ 48%¹² and 80%⁵ of patients with carpal tunnel syndrome. These are thus somewhat unsatisfactory as conclusive diagnostic tests. The Flick sign is at least as valid and reliable in diagnosis. The population of patients reported here is comparable in all respects to those described in other series.^{6, 11, 12}

Why this movement is employed so often in carpal tunnel syndrome and so seldom in other conditions is not easily explained. "Flicking" (as also rubbing or beating the hand or repeatedly flexing the wrist and fingers) increases thick fibre proprioceptive input, increases venous pressure and volume, might briefly increase arteriolar pressure, and moves the nerve underneath the free distal edge of the flexor retinaculum. This last factor may be of importance if it "untethers" a nerve which had formerly been unable to stretch during displacement.¹⁸ Increasing proprioceptive input might "close the gate" and thus reduce symptoms since paraesthesiae may reflect the relative reduction in the number of thickly myelinated fibres in the median nerve which occurs in carpal tunnel syndrome.¹⁹ Since more sensory fibres exist in the median than in the ulnar nerve,¹⁹ this technique for symptomatic relief may thus be employed more readily with median than with ulnar nerve lesions but does not explain why the manoeuvre is not common with proximal median nerve lesions.

Involvement of sympathetic fibres has been documented in carpal tunnel syndrome.²⁰ This might reduce vasoregulatory ability and lead to the swelling, coolness and dryness of the affected hand. By increasing arterial pressure, the centrifugal flicking movement might improve perfusion of sympathetic fibres and thus reduce any abnormal afferent impulses within those fibres. However, such autonomic involvement was only demonstrated in less than 40% of Aminoff's patients with carpal tunnel syndrome.²⁰

Sunderland²² suggests that due to the pressure gradient across the fibres, stasis in the epineurial veins and decreased intrafunicular circulation are the first effects of increased pressure in the carpal tunnel. The large rather than the smaller myelinated fibres are thus metabolically embarrassed and become hyperexcitable, clinically manifest as paraesthesiae. Flicking then would increase arteriolar pressure, blood flow and venous return (this latter aided by mobilisation of the nerve beneath the transverse carpal ligament). This in turn might reduce oedema and normalise the electrical stability of the formerly ischaemic fibres, thus providing a degree of symptomatic relief. Since similar pathologic conditions are not known to occur at other sites of nerve damage in the arm, this theory

or that of "untethering" the nerve appear the most attractive.

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