J Neurol (2014) 261:1340–1343
DOI 10.1007/s00415-014-7329-4

ORIGINAL COMMUNICATION

The tibialis anterior response revisited

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Received: 28 February 2014 / Revised: 23 March 2014 / Accepted: 24 March 2014 / Published online: 24 April 2014

Abstract The idiomuscular response to direct percussion is rarely tested nowadays because of its uncertain mechanism and significance. While performing neurological examination, we observed a brisk ankle dorsiflexion response on direct muscle percussion of *m. tibialis anterior* in patients with acute inflammatory demyelinating polyradiculoneuropathy (AIDP). In contrast, in patients with upper motor neuron lesions, an ankle inversion response was seen. In this article we describe our findings in patients with bilateral lower limb weakness. We assessed 73 consecutive patients with bilateral lower limb weakness. A strong dorsiflexion response to percussion of *m. tibialis anterior* was seen in 11 out of 14 patients with AIDP (sensitivity 78.6 %). None of the other patients showed a strong dorsiflexion response (specificity 100 %). An inversion response was seen in 11 out of 13 patients with UMN involvement (sensitivity 92.3 %). It was also noted in two of 46 patients without proven UMN involvement (specificity 96.7 %). The idiomuscular response to percussion of *m. tibialis anterior* can be useful in the assessment of patients with lower limb weakness of unclear cause.

Keywords Percussion · Guillain–Barré syndrome · Spasticity · Clinical examination

Introduction

In 1858 the German physiologist Moritz Schiff described what he called "idiomuskuläre contraction" (idiomuscular contraction), the activation of skeletal muscle after direct percussion [1]. Twenty-three years later in his monograph, The diagnosis of diseases of the spinal cord, Gowers described a phenomenon he named "front-tap contraction" seen in patients with spasticity where percussion of "the muscles on the front of the leg" causes ankle dorsiflexion [2]. This response has been described as diminished in myopathies, but increased in neuropathies [3]. More recently, it has been linked to conduction block [4, 5]. Today, the idiomuscular response to percussion is rarely tested because of its uncertain mechanism and significance.

Whilst performing neurological examination, we observed a brisk ankle dorsiflexion response on direct muscle percussion of *m. tibialis anterior* in patients with acute inflammatory demyelinating polyradiculoneuropathy (AIDP). In contrast, in patients with upper motor neuron lesions, an ankle inversion response was seen. We believe this sign to be clinically useful in the assessment of patients with lower limb weakness of unclear cause. This article summarises our findings.

Patients and methods

We enrolled 73 consecutive patients (mean age 52.8 years, 38 male and 35 female) with bilateral lower limb weakness from June 2012 to May 2013 from the Princess Alexandra

Electronic supplementary material The online version of this article (doi:10.1007/s00415-014-7329-4) contains supplementary material, which is available to authorized users.

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Hospital and the Royal Brisbane and Women's Hospital in Brisbane, Australia. Of these, we examined 65 patients at their initial presentation prior to confirmation of the diagnosis; in eight patients the diagnosis was known at the time of testing.

All patients underwent a full neurological examination. To test the idiomuscular response, reflex hammer percussion of *m. tibialis anterior* was performed several times to obtain the briskest possible response [5]. For this, the patient was in prone position with the knee flexed at about 15 degrees. As per visual observation, the response was graded as either "absent or mild dorsiflexion", "strong dorsiflexion" or "inversion" (see supplementary video 1–3). All patients with clinical signs of upper motor neuron involvement had magnetic resonance imaging (MRI) of the whole spinal cord and brain. Most, but not all patients underwent nerve conduction studies (NCS) and electromyography (EMG). If there were clinical features of pure upper motor involvement or clear evidence for an underlying pathology, (such as proven genetic mutations for certain neuropathies or myopathies) then NCS were omitted (in 19 patients).

Electrophysiological studies were performed on a Viking EDX or Synergy EMG apparatus using standard procedures, and MRI was performed on a 1.5T Siemens machine.

Statistical analysis was performed using Excel (Microsoft) and SPSS (IBM). Fisher's exact test was used to examine the significance of the association.

This publication did not require ethical approval. Verbal consent for publication was obtained from the patients involved in the submitted videos. Only the lower limbs were visible in any video recording.

Results

The diagnoses at discharge were: fourteen patients fulfilled clinical and electrophysiological criteria for the AIDP variant of GBS, and three fulfilled the criteria for the acute motor axonal neuropathy (AMAN) variant [6, 7].

Nine patients presented with pure upper motor neuron pathologies [MS (4), compressive cervical cord lesion (2), HSP (1), spinal cord infarct (1) and primary lateral sclerosis (1)]. Four patients had clinically definite amyotrophic lateral sclerosis (ALS) with upper and lower motor neuron involvement at the time of testing [8].

The remaining patients were divided up as follows:

Peripheral neuropathies other than GBS were seen in 27 patients; of these nine patients met the EFNS/PNS criteria for definite chronic inflammatory demyelinating polyneuropathy (CIDP) [9]. Nine patients had myopathies and six patients functional lower limb weakness. One patient presented with a typical history and clinical picture of GBS, including weakness, areflexia and raised CSF protein, but normal nerve conduction studies.

Table 1 Examination results

Response	AIDP ($n = 14$)	UMN ($n = 13$)	All others ($n = 46$)
Strong dorsiflexion	11	0	0
Inversion	0	11	2
Absent/mild dorsiflexion	3	2	44

AIDP acute inflammatory demyelinating polyradiculoneuropathy, *UMN* upper motor neuron involvement

A strong dorsiflexion response to percussion of *m. tibialis anterior* was seen in 11 out of 14 patients with AIDP (sensitivity 78.6 %; Fisher $p < 0.001$). On lower limb NCS, ten of these 11 patients had either a F-wave latency longer than 150 % of the normal mean or absent F-waves with normal compound muscle action potential (CMAP) amplitudes. All AIDP patients had areflexia at the knee and ankle. Of the patients with a negative response, one had AIDP on the background of a pre-existing axonal neuropathy; the two others, whilst meeting criteria for AIDP, only showed prolonged distal latencies without evidence of proximal demyelination on NCS. None of the other patients showed a strong dorsiflexion response (specificity 100 %).

An inversion response was seen in 11 out of 13 patients with UMN involvement (sensitivity 92.3 %; Fisher $p < 0.001$). These subjects had hyperreflexia and moderate weakness but only 36 % had extensor plantar responses. An inversion response was also noted in two of 46 patients without proven UMN involvement (specificity 96.7 %). Of these, one patient had an axonal neuropathy on NCS, but also an extensor plantar response on the side of testing of unclear aetiology (MRI of the brain and spinal cord normal). The other patient had clinical and CSF features of GBS, but normal NCS, as previously noted. None of the other patients with neuropathies or myopathies had an inversion response.

An overview of the examination results is given in Table 1.

Discussion

This study expands the spectrum of the response to direct muscle percussion [5]. The response appears to be a useful clinical test in two clinical scenarios:

1. The patient with flaccid lower limb paralysis, where a strong dorsiflexion response can help to support a diagnosis of AIDP. This result is consistent with the observations of Magistris et al. [5], who described an increased

idiomuscular response in the presence of conduction block with a specificity of 100 %.

It is not clear how long it takes for the strong dorsiflexion response to appear and how long it persists for. In this study we haven't performed any serial testing, but in patients with focal conduction block, an increased response could be seen as early as three days and as late as 100 days [5]. However, none of the CIDP patients we examined had a strong dorsiflexion response.

2. The patient with lower limb weakness and brisk reflexes, where an inversion response could indicate upper motor neuron dysfunction. This is an extension of the knee/ankle reflexes, and might be helpful if these reflexes cannot be easily performed, e.g., after knee surgery, if there is difficulty with access for examination or where the plantar response is uncertain.

Historically it was believed that the idiomuscular response also aids the differentiation between myopathies and neuropathies [3]. We didn't find the response helpful in this setting, as it can be very difficult to judge on subtle differences in the response (i.e., absent vs. mild dorsiflexion), and we did not have sufficient patients in the groups to separate this. It is possible that the previous observation of a dorsiflexion response in patients with neuropathy was largely due to inclusion of patients with AIDP in this group.

The underlying mechanism of the idiomuscular response to direct percussion remains poorly understood. Direct percussion elicits two separate bursts of action potential in a muscle, one immediately after the tap and the other one after a latency of about 40 ms [10]. Brody and Rozear [10] showed in a series of experiments in humans and animal models that the delayed but not the immediate response depends on innervation by a peripheral nerve and has characteristics of a spinal reflex. They also showed that the immediate response is not due to activation of intramuscular nerves, but due to direct stimulation of sarcolemma. However, other authors believe that the response is due to indirect excitation via motor nerve depolarisation [5]. In patients with persistent conduction block, ectopic muscle activity is often seen, which is probably due to axonal membrane hyperpolarisation distal to the block [11]. This would be consistent with the increased response seen in patients with AIDP.

It is more difficult to interpret the inversion response seen in patients with upper motor neuron pathology, but one explanation could be a disinhibited spinal reflex akin to the plantar response. *M. tibialis posterior* is the main muscle causing foot inversion and is typically overactive in patients with spasticity, thus, contributing to equinovarus deformity [12]. If there is an increased delayed response in people with spasticity as described by Brody and Rozear [10], this could explain an inversion response triggered by an overactive *m. tibialis posterior*. Although a disinhibited spinal reflex seems to be the most likely explanation, other possibilities exist. For example, the shockwave produce by percussion of *m. tibialis anterior* could activate multiple muscles directly (i.e., as direct sarcolemma activation) and not via the spinal cord. The final vector of the contractions of these activated muscles could produce ankle inversion.

In summary, the idiomuscular response to *m. tibialis anterior* is simple to perform and may be helpful in assessing leg weakness. Our findings are limited by small numbers, the heterogenous nature of the diseases and the performance of the response at only one time-point and without interobserver rater comparison. Further research could define the comparison with normal subjects, and when and where the response is useful, e.g., if the strong dorsiflexion response has clinical utility early in the course of AIDP, or in subjects with multifocal motor neuropathy. Another point of interest is if the inversion response is potentially helpful when subtle UMN signs are present, for example, in the setting of early ALS.

Acknowledgments The authors thank Emeritus Professor Mervyn Eadie for his valuable contributions to this article.

Conflicts of interest Alexander Lehn: none, Sasha Dionisio: none, Caroline Airey: none, Helen Brown: none, Stefan Blum: none, Robert Henderson: none.

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