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CORRESPONDENCE

Pretarsal injections of botulinum toxin improve blephospasm in previously unresponsive patients

The experience of Albanese *et al*[1] is very interesting because it is a good example of the old adage that logical treatment works. The technique as taught by Scott does involve the injection of pretarsal botulinum toxin because it is spasm of the pretarsal muscle that causes the symptoms.

Injection of the preseptal muscle should not be expected to be as effective for controlling the symptom of lid spasm. Injection of the superciliaris is sometimes required to control the brow spasm that many of these patients get.

A similar study to that of Albanese *et al* [1] has been reported locally with a similar result—the logical treatment of the pretarsal muscle is more effective (but produces slightly more side effects) than the less logical injection of the preseptal orbicularis.

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1 Albanese A, Bentivoglio AR, Galardi G, Maderna L, Colosimo C, Tonali P. Pretarsal injections of botulinum toxin improve blephospasm in previously unresponsive patients. *J Neurol Neurosurg Psychiatry* 1996;**60**:693-4.

Albanese replies:

I agree with the propositions of Kowel. In my experience, patients sometimes question the need for pretarsal botulinum toxin injections due to local pain at the time of treatment. Other side effects (for example, small local haematomas) are a minor concern.

I suspect that, after the pioneering treatments, injection sites have been gradually moved to preseptal and even to orbital locations to avoid local pain. This must be taken into account considering that many patients still do well with preseptal botulinum toxin injections.

ALBERTO ALBANESE

Rehabilitation of gait in Parkinson's disease

We comment on the recent publication by McIntosh *et al*[1] concerning rhythmic auditory motor facilitation of gait patterns in patients with Parkinson's disease. These authors have shown a roughly 10% improvement in gait velocity as a result of entrainment of cadence with a metronome embedded in background music.

Although this study is interesting, the level of improvement in gait is therapeutically small and not directed to the amelioration of the basic deficit in parkinsonian gait. Other studies have clearly shown that the basic deficit in the control of parkinsonian gait is regulation of stride length. Cadence control is intact in Parkinson's disease (Morris *et al*[2]) and cadence is used as a compensatory mechanism for the defective control of stride length. This is done to increase gait velocity above normal and preset values for each individual patient. We see very little benefit in entraining patients whose cadence is already increased above normal values to improve velocity by values of only the order of 10%. This is particularly so when it is possible to restore normal gait velocity, normal cadence values, and normal stride length by use of either visual cues and more importantly by attentional strategies that are not dependent on any visual cues whatsoever. Under these circumstances it is possible to restore normal gait indices in patients with Parkinson's disease even though they may be totally off their medication or when medication is totally ineffective.

The percentage improvement in gait velocity associated with auditory stimulation is of the same order that can be attained by patients by simply asking them to walk faster. When asked to do so, patients with Parkinson's disease increase their cadence rate, and to a lesser degree stride length, by similar amounts to improve their velocity. This requires no complex equipment—only a gentle prompt to walk at a faster rate.

However, to obtain normal indices patients need to be shown the correct stride length that they are to attain and then told to use attentional strategies to attain that stride length. Again with very minimal equipment and with a modicum of encouragement it is possible to allow patients with festinant gait to walk normally.

We do not disagree with what the authors have demonstrated in their paper, nor do we necessarily disagree with their conclusions; however, we merely wish to put these findings into some form of perspective and of relevance, particularly in the sphere of rehabilitation. We have now used stride length normalisation as a strategy for restoring gait to normal values in our multidisciplinary programme and have found this very effective. The problem is that it requires constant vigilance and continual reinforcement for it to be applied in a consistent manner. For that reason we have used this approach as a back up for medication failure or medication ineffectiveness and not as a mainstay in treatment.

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1 McIntosh GC, Brown SH, Rice RR, Thaut MH. Rhythmic auditory-motor facilitation of gait patterns in patients with Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1997;**62**:22–6.
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Thaut replies:

We entirely concur with Iansek and Morris in regard to the importance of stride length regulation in gait rehabilitation of patients with Parkinson's disease. In regard to their comments about our paper some apparent errors in reading our data may have occurred:

(1) Iansek and Morris state that our study found a 10% increase in velocity. Most of their argument against the rehabilitation relevance of our paper is based on this increase. However, the actual increase in our paper (see p 24) was 36% for ON patients and 25% for OFF patients. This is potentially a very useful increase for rehabilitation.[1]

(2) The second argument against the therapeutic usefulness of our paper concerns the improvement of cadence rates which are already raised above normal. Again, this assertion does not fit the cadence rates in our study sample. The cadence for ON patients was 98 and 91 for OFF patients, which is clearly below normal age matched cadence rates and matches gait data from other studies .

(3) The emphasis of Iansek and Morris on the importance of stride length is well taken. However, our study supports that point. Rhythmic cuing improved both stride length and cadence in our study (p 24, table 2, and results section). This dual effect of rhythmic cuing on stride length and cadence in Parkinson's disease has also been reported by other researchers.[2]

(4) The error of Iansek and Morris in commenting on a 10% increase in velocity may have stemmed from mistaking it for the 10% increase in cadence found in our study. This, however, simply reflects the exact synchronisation to the rhythmic cue which was set only 10% faster than each patient's baseline to avoid hastening into abnormally high step rates. Patients were able to follow the rhythmic cue in close synchronisation without compromising their stride length.

(5) From a clinical perspective it needs to be clearly reiterated that nowhere in this study do we suggest that patients with step rates already above normal should be cued to even higher step frequencies. The assertion of Iansek and Morris in this respect is unfounded. However, patients with Parkinson's disease with slowed step rates may very well benefit from rhythmic cuing to improve velocity, or the entrainment effect may actually be used to decelerate hastened stride. Besides the apparent errors in reading our results, we fail to see how this is less relevant than or contradicts other facilitation