Distinguishing Motor Weakness From Impaired Spatial Awareness: A Helping Hand!

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**ABSTRACT:** Our patient, aged 73 years, had background peripheral neuropathy of unknown cause, stable for several years, which caused some difficulty in walking on uneven ground. He attended for a teaching session but now staggered in, a new development. He had apparent weakness of his right arm, but there was difficulty in distinguishing motor weakness from impaired spatial awareness suggestive of parietal lobe dysfunction. With the patient seated, eyes closed, and left arm outstretched, S.A.R. lifted the patient’s right arm and asked him to indicate when both were level. This confirmed motor weakness. Urgent computed tomographic scan confirmed left subdural haematoma and its urgent evacuation rapidly resolved the patient’s symptoms. Intrigued by our patient’s case, we explored further and learnt that in rehabilitation medicine, the awareness of limb position is commonly viewed in terms of joint position sense. We present recent literature evidence indicating that the underlying mechanisms are more subtle.

**KEYWORDS:** Arm weakness, motor impairment, spatial awareness, differentiation, simple new bedside test

**INTRODUCTION**

It is difficult to distinguish true motor weakness from impaired spatial awareness, the latter indicative of parietal lobe dysfunction. We suggest a simple method to resolve a diagnostic dilemma which may lead to the correct diagnosis.

To the best of our knowledge, the technique used has not been described before; hence, we wish to bring it to the attention of colleagues.

**BACKGROUND HISTORY**

The patient (C.R.S.), aged 73 years, is a retired Acting Senior Manager at a nearby colliery. After retirement, he pursued a career in furniture restoration.

He was under the care of K.D.B. for surveillance of Barrett’s oesophagus, had undergone a right nephrectomy for a carcinoma in 1997, and later developed peripheral neuropathy. Nerve conduction studies showed large fibre sensory motor axonal peripheral neuropathy, with more severe sensory axonal loss. There was also evidence of gangliounopathy. No evidence of paraneoplastic neuropathy was found; hence, his disorder was classified as ‘peripheral neuropathy of unknown cause’.

K.D.B. had last seen C.R.S. about 2 years earlier for surveillance gastroscopy followed by clinical assessment. Neurological examination showed that the degree of functional impairment was comparable with that noted in earlier assessments. Thus, he was able to play golf regularly despite the reduced sensation in his feet. Walking was slightly difficult when on uneven ground and more so when trying to walk in the dark.

He attended a junior medical student teaching session to demonstrate his neurological problems.

**THE ACUTE EVENT**

In view of his earlier neurological assessments, it was surprising to see the patient stagger into the seminar room. It transpired that he had developed a bad headache several days earlier, mainly frontal and at the vertex. Its intensity built up gradually, interspersed with occasional periods of relief for up to an hour but later became continuous. During its early stages, he saw his General Practitioner (GP), who prescribed analgesics and arranged for review but advised C.R.S. to go to the Emergency Department if headaches rapidly worsened. Shortly afterwards, the headaches increased so a neighbour took the patient to the Emergency Department in the late afternoon. After assessment, the doctor increased the analgesics and kept him under observation before discharging him about 4 hours later and advised him to consult his GP again for review.

That evening, C.R.S. noticed difficulty with fine movement, for example, texting messages incomprehensible to his family. Over the next few days, he noticed that his right leg and foot had become ‘clumsy’, heavy, and difficult to move. He tried to get to his garage to get some items from the freezer but fell a few times on the way.

**FINDINGS**

The striking feature now was the marked change in his walking, which was laboured as he had to drag his right leg and with an unusual ‘clumsiness’ of his right hand and arm. Initially, it was difficult to distinguish between motor weakness of the upper limb and impaired ‘spatial awareness’, the latter suggesting parietal lobe damage and commonly tested for in terms of ‘joint position sense’ (JPS). These features were in marked
contrast to the clarity of his mind, speech, and recollection. The history and physical signs were compatible with a ‘stroke’ but, in view of his falls, also of an intracranial bleed.

A test commonly used to check for parietal lobe integrity, in particular for JPS of the upper limb, is to ask the patient to close both eyes and then simultaneously hold out arms in front at shoulder height with hands and fingers outstretched, parallel to each other and with the palms facing down. One can then check for ‘drift’. The left arm was unaffected. The procedure was modified for testing the right arm. The patient remained seated to steady himself and was then asked to perform the steps with the left arm only. The final step was to then match the position with the affected right.

C.R.S. had difficulty elevating the right arm. S.A.R. therefore suggested that he would provide the motor power by lifting the affected arm and the patient could then call out when he felt his limbs were level. C.R.S. was fully aware when the limbs were aligned, thus confirming he had a pure motor weakness.

He was admitted and urgent computed tomographic scan confirmed a large left subdural haematoma with a 1-cm shift to the right of the midline. The patient was transferred to the neurosurgical centre where the haematoma was evacuated the next day, followed by rapid improvement.

Discussion

Distinguishing impaired spatial awareness from motor weakness is not always straightforward. The Oxford Textbook of Medicine states parietal lobe lesions specifically impair ‘discriminative function’, which encompasses both JPS and 2-point discrimination. Parietal drift (which we were trying to detect) reflects impaired JPS. It is considered specific for a contra-lateral parietal lesion when the drift is up and outward, as a downward drift may also be the consequence of subtle motor weakness.

To gain additional insight, we reviewed several studies in the field of rehabilitation medicine, both older and more recent literature. Our impression was that the specialists perceived limb position awareness in terms of JPS. Recent evidence from investigation of patients with stroke, however, suggests that separate pathways may be involved for mediating kinaesthesia, the sense of body motion, as opposed to JPS.2

Several of these studies concern rehabilitation following strokes, in which robotic devices have been used to elevate the arm and include both patients and control subjects for comparison; in some investigations, the subjects were blindfolded. Robot-assisted elevation allowed measurement of specific parameters to quantify subtle changes not readily discernible by clinical means. Such techniques have been used to explore ability to ‘mirror position’,1 limb position,4–5 proprioception,6–7 track movement,8 and kinaesthesia.9 A variant on these methods is the use of an inclinometer to measure internal-external rotation of the arm and the tension generated.10

To us, outwith the field of rehabilitation medicine, a separate pathway for sensing body motion as a means of creating ‘awareness’ of the position of a limb is intuitively more appealing than to view as a construct based solely on integrating the position of its several individual joints. This, however, raises the question of how body position within the 3 dimensions is recognised.

Likely mechanisms have been discovered recently by groundbreaking advances in neurophysiology made through experimental and clinical investigations which have revealed that the entorhinal cortex, located in the medial part of the temporal lobe, has a variety of specific cells, which gives rats an ‘internal sensing’ system of spatial awareness (‘place cells’), direction (‘grid cells’), and speed of movement (‘speed cells’) for which the Nobel Prize in Physiology or Medicine (2014) was shared by John O’Keefe (London, UK) and the husband and wife team of Edvard and May-Britt Moser (Denmark).11–20 Finally, a further cell type has recently been identified which allows rats to know their position when still.21

The entorhinal cortex is affected in some patients with early-stage Alzheimer’s disease. This correlates with the well-known clinical observation that such patients can no longer navigate through their surroundings and tend to lose their way.

The analogy between the daily common experience of driving domestic cars guided by ‘global positioning satellites’ (GPS) will not have escaped the reader. GPS is made possible by multiple external satellites, whereas the complex neural circuitry of ‘internal sensing’ has evolved over time.

Speculation

We believe that such investigations will give new understanding of movement and orientation of a person or of a part of his or her body and with it bring benefit to patients who require rehabilitation following strokes and brain injury.

Conclusions

We could not find a specific reference in the literature to the manoeuvre we described. Nevertheless, we recognise that as with the wider field of medicine itself, physical examination, too, is based on science but its practice is an ‘art’. Hence, others may also be using modifications such as the one we describe but have not placed it on record. Therefore, to help colleagues who may encounter a similar clinical situation, we offer the manoeuvre as a ‘helping hand’.

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**Author Contribution**
SR and CRS conceived and designed the experiment. SR and KDB wrote the first draft of the manuscript, reviewed the literature, and agree with the manuscript conclusions. KDB, SR, and CRS contributed to the writing of the manuscript. All authors reviewed and approved the final manuscript.

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