

Pyramidal weakness

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The signs associated with an upper motor neurone syndrome vary considerably between text books. Often included are loss of dexterous, rapid and fractionated voluntary movements, variation of movement with mode of activation (prime mover, synergist, antagonist), weakness, various postural signs (eg, pronator drift), time-dependent tonal and tendon reflex changes and the extensor plantar response. Russell Brain's first edition of *Diseases of the Nervous System* (1933, pp 6-8) refers to movements of flexion being stronger than extension in the upper limb and extension movements stronger than flexion in the lower limb as one negative feature of a unilateral pyramidal (internal capsular) lesion, albeit attributing this 'probably' to the distribution of hypertonia. However, this pattern of 'pyramidal weakness' (also including shoulder abduction) has since been emphasised in many text books as being characteristic of an upper motor neurone lesion.

Early in my consultant career, I saw a man with acute pain in the low and mid back and weakness in both legs. He could barely stand, tone was normal and he had weakness of hip flexion more than extension, knee flexion more than extension and ankle dorsiflexion much more than plantar flexion: tendon reflexes were brisk, and plantar responses silent with joint position and vibration sense loss in the toes and distal cutaneous loss. I diagnosed an evolving spinal cord lesion. However within hours he had obvious facial, bulbar and arm weakness (also in a 'pyramidal pattern') and absent tendon reflexes: he turned out to have Guillain-Barré syndrome. I was dismayed by being initially misled about localisation mainly through the distribution of weakness.

When manually examining strength in specific muscle groups (figure 1), we usually encourage the patient to build up their strongest contraction in an approximately isometric manner. We then judge it to be normal or otherwise either

by comparison with the normal side (if weakness is unilateral) or from our knowledge of normal strength in different muscle groups acquired by examination of many patients. Whether or not we can 'break' a contraction depends on the muscle group, the individual patient and our own strength but does not, per se, indicate weakness. I know that I can sometimes break maximum voluntary elbow extension but not flexion in a healthy subject, rarely hip or knee extension, and virtually never ankle plantar flexion. Only the most determined examiner can generate a manual counterforce of 200–300 N or more. Thus, we expand clinical testing to use the subject's body weight to stand from sitting (hip extension) or to hop (ankle plantar flexion). The quality of the patient's effort, for example, 'give way' weakness, the rate of force build up, early fatigue or disparity between 'make' and 'break' forces may also inform diagnosis.

In healthy men and women aged 20–79 years, quantified measurements of isometric strength in positions similar to those used in clinical examination show that strength of shoulder abduction is about 0.6–0.8 that of adduction; elbow extension about 0.7–0.9 that of flexion; and wrist extension about 0.4–0.6 that of flexion. Similar ratios pertain for hip and knee flexors compared with extensors, and ankle dorsiflexion is 0.3–0.4 that of plantar flexion.¹ Thus, a 'super-strong' examiner testing a healthy individual would find a so-called 'pyramidal pattern'. If strength were reduced by 30% in *both* flexors and extensors (ie, no selective weakness), more examiners would detect a 'pyramidal pattern'.

Whether lesions engaging the corticospinal pathways cause a selective 'pyramidal pattern' of weakness was directly addressed in both upper and lower limbs using fixed dynamometry, with allowances for gravitational torques.^{2–4} Physiological flexors and extensors were equally affected in the



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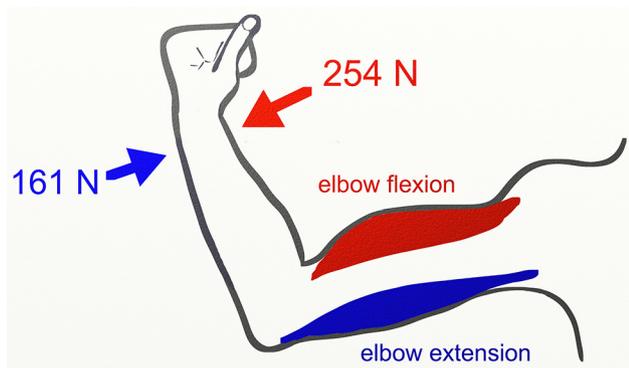


Figure 1 The examiner's counterforce (arrows) required to 'break' a maximum voluntary contraction of elbow flexion (red) or extension (blue) in a healthy male subject.

lower limbs of both hemiparetic and paraparetic subjects. In the upper limbs, some flexor groups (notably wrist and fingers) were actually weaker than extensor groups, though the pattern was variable. In another study, the ratio of flexor to extensor strength at the elbow, wrist and knee was not significantly different between patients with 'central' or 'peripheral' causation for weakness.⁵

A focused examination to ascertain (any) specific weakness of muscle groups can be an important component of diagnosing an upper motor neurone lesion.⁶ But manual muscle testing is not an easy skill for undergraduates to acquire. However, *observing* the patient (1) doing what they say they find difficult, (2) in movement (walking, finger dexterity, arm or finger rolling, foot tapping), (3) maintaining a posture (eg, outstretched arms, pronator test), and evaluating tone and reflexes (mindful of time from clinical event) may be easier to learn and is often more revealing.

'Pyramidal weakness' may be an illusion resulting from manual testing, the natural strength of muscle groups and the distributed nature of (sometimes marked) increased tone in an upper motor neurone lesion. Undergraduates, neurology trainees and even experienced clinicians should probably not make too much of it. A deeper analysis of clinical features of the upper motor neurone syndrome caused by lesions at differing levels in the corticobulbar/corticospinal pathways using modern imaging and quantification of signs would be worthwhile.

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