Inverted knee jerk: a neglected localising sign in spinal cord disease

R. S. BOYLE, R. A. SHAKIR, A. I. WEIR, AND A. McINNES

From the Glasgow University Department of Neurology, Institute of Neurological Sciences, Southern General Hospital, Glasgow

SUMMARY Two cases are described in which inversion of the quadriceps jerk was observed. It is suggested that the finding of an inverted quadriceps jerk is evidence of a spinal cord lesion at L2, 3, 4 segmental level and has for the lumbar cord the same localising value as the inverted supinator jerk for the cervical cord.

Inversion of the supinator jerk is a widely known clinical sign, localising spinal cord pathology to the C5/6 spinal segmental level. Less well recognised as a useful clinical sign is “inversion” of the quadriceps (knee) jerk, in which a brisk tap of the quadriceps tendon results in contraction of the hamstring muscles and flexion of the knee. We have recently observed this sign in two patients.

Case reports

CASE 1
A 62 year old woman presented with a six month history of gradually progressive weakness of the lower limbs. There was no bladder dysfunction. Examination of the patient showed that she had marked weakness of hip flexion and knee extension, with a pyramidal tract type distribution of weakness affecting the remainder of the muscle groups in the lower limbs. Both quadriceps jerks were inverted. Ankle jerks and hamstring jerks were pathologically brisk bilaterally, and bilateral extensor plantar responses were obtained. Sensation was impaired from L2 to L3 dermatomes, with sacral sparing bilaterally. Myelography was performed, and a total block to the flow of contrast medium was observed opposite the body of L1 vertebra. In addition, a large paraspinal mass opposite the bodies of T11 to L1 vertebrae was noted on the left side (Fig. 1). Biopsy of this paraspinal mass showed it to be a secondary carcinomatous deposit.

Address for reprint requests: Dr R. S. Boyle, Department of Neurology, Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF.

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Fig. 1 Myelogram of case 1, showing obstruction to flow of contrast medium opposite L1 and paravertebral mass at T11–L1 vertebral level.
CASE 2
A 28 year old woman had been known to have lumbosacral spina bifida since birth. She gave a six year history of increasing difficulty with gait and of a tingling feeling in the anterior aspects of both thighs, with numbness down the outer aspect of the left leg. Examination revealed bilaterally wasted quadriceps together with wasting of the left gluteal muscles, hamstrings, anterior tibial compartment, and foot. The left knee jerk was absent and the right was inverted. The right hamstring jerks were exaggerated while the left were absent. Both ankle jerks were pathologically brisk, and ankle clonus was present bilaterally. There was sensory impairment in L5 segmental distribution in the left leg. Plain radiographs showed lumbar sacral scoliosis with wide spina bifida occulta of L3 and L4 vertebrae. Myelography showed a large posterior meningocele with numerous septa crossing it.

ELECTROMYOGRAPHY
Electromyography with surface electrodes recording over quadriceps femoris and hamstring muscles was performed during elicitation of the monosynaptic tendon jerk with the knee at 90° flexion. The oscilloscope traces shown in Fig. 2 were obtained by triggering with an inertia switch on the tendon hammer at the point of impact with the patellar tendon. In the control subject (Fig. 2A) a compound action potential was recorded from quadriceps (a) while the only electrical activity in the hamstrings (b) was an attenuated mirror image of that potential. The absence of muscle activity in the hamstrings was confirmed by concentric needle electromyography. In case 2 (Fig. 2B) the predominant potential was that from the hamstrings (b) with a much smaller contribution from quadriceps (a). Similar results were obtained in case 1.

Discussion
Absence of knee extension on testing the quadriceps jerk indicates interruption of the L2, 3, 4 monosynaptic reflex arcs. Flexion of the knee on testing for this tendon jerk indicates that the L5, S1 reflex arcs are intact. Readily visible flexion suggests excessive reflex activity in the hamstrings (L5, S1 segmental innervation). The presence in these two patients of exaggerated ankle and hamstring jerks, together with extensor planter responses, also indicates pyramidal tract dysfunction below the spinal level of L4 segment. Patient 2 also had clear evidence of L5 segmental dysfunction on the left side, explaining the total lack of reflex activity on testing for the quadriceps jerk on that side.

Theoretically, any interruption on the afferent side of the L2, 3, 4 reflex arcs in the presence of intact L5, S1 reflex arcs will lead to inversion of the quadriceps jerk. This is the result of mechanical stimulation of the primary sensory endings of the muscle spindles in the hamstrings, and of the absence of normal inhibitory influences on the hamstrings because of interruption of the afferent side of the L2, 3, 4 reflex arcs. However, facilitation of the L5, S1 reflex arcs will be necessary if this inversion is to be readily visible. Thus, clinical inversion of the quadriceps jerk could be caused by any extrinsic or intrinsic lesion of the spinal cord which will both interrupt L2, 3, 4 reflex arcs and damage the pyramidal tracts supplying segments below L4. A single lesion giving rise to this...
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Sign would, in the presence of normal anatomy, be situated at T11–L1 vertebral level. However, it must be realised that, as with inversion of the supinator jerk, inversion of the quadriceps jerk may be associated with dual rather than single pathological lesions—for example, a combination of cervical myelopathy and a femoral neuropathy will lead to inversion of the quadriceps jerk.

This abnormality of the quadriceps jerk has not been widely reported in English reports. Wartenberg (1945) mentions it in his comprehensive monograph, but previous references are confined almost entirely to the European literature. Guttmann (1976) mentions it in reference to spinal cord trauma but strangely relates it to a conus lesion. We suggest that the finding of an inverted quadriceps jerk alone is evidence of a spinal cord lesion at L2, 3, 4 segmental level and has for the lumbar cord the same localising value as the inverted supinator jerk for the cervical cord.

References


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