Matters arising

Hypothesis relevant to defective position sense in a damaged knee

Sir: Detailed descriptions of personal clinical experience following a knee injury and subsequent surgical intervention1 are of great interest in light of recent morphological findings related to the innervation of mammalian knee joints. The general controversy which has surrounded this subject for several decades can be attributed, in part at least, to the dearth of interdisciplinary studies. Morphologists have concentrated almost exclusively on the detailed structure of conventional receptors2 while physiologists have given increasing attention to “single unit recording”.3 The likelihood that either of these research approaches will arrive at a satisfactory explanation of the problems1 seems quite remote.

Our current investigation has been based upon a comprehensive, holistic approach to peripatellar structures and their neural components. Traditional methods, applied initially, became increasingly unacceptable and entrenched theories of distinguishing between “articular” and “muscular” afferent mechanisms were abandoned. Large specimens for histological study were preferred, comprising, for example, the entire anterior apparatus of mammalian knee joints. These portions of tissue, having the patella as the central feature, were serially sectioned at an optimum thickness of 150 microns for subsequent impregnation with either gold or silver salts using standard procedures for the demonstration of neural components.

A conspicuous peripatellar plexus was present in every species examined including mice, rats, rabbits, guinea pigs, cats and man. This consistent finding led to quantitative studies in adult male rats which provided three-dimensional evidence that this network is composed of nerve fibres ranging in diameter from 2 to 8 μm which are arranged in a series of polygonal patterns and extend to a depth of 1-5 mm from the superficial fascia.4 In view of the similarities of arrangements in all the species examined a hypothesis was formulated that the nerve plexus may constitute an integral part of the hierarchy of afferent mechanisms. Its function may be to modulate afferent feedback by acting as a “transducer” in response to distortion during joint movement.5 Functional studies were postponed pending further quantitative analysis subsequent to selective partial denervation experiments.

The most dramatic observation in these investigations was the widespread proximodistal, Wallerian degeneration of fibres within the plexus following transection of the tibial division of the sciatic nerve. This was confirmed in a series of five animals establishing that a substantial majority of afferent fibres which constitute the peripatellar plexus form accessions which join the posteriorly placed tibial division of the sciatic nerve.6

Almost coincidental with these structural studies electro physiological investigations6,7 provided confirmation that mechanical stimulation of the peripatellar region gives rise to recordable responses in recognised articular nerves. The calculated calibre of these fibres, based on conduction velocity, is compatible with the above mentioned morphological findings. Taken together, these results8,9 offer an explanation for the unexpected and controversial physiological finding10 which did not explain the absence of recordable afferent discharges from certain conventional receptors during the mid-ranges of joint movement. The units identified in the more recent study8 are shown to be active throughout the entire range of joint movement.

It seems reasonable to assume that some of these recent observations may assist in explaining clinical defects following knee surgery. Previous attempts to collect sufficient data to explain these defects10 suggest that abnormal sensory feedback may be caused by surgery. Coincident with the advent of modern arthroscopic techniques there has been a significant reduction in the incidence of complications of this kind.11 The circumstantial evidence suggests that the choice of surgical procedure is a crucial factor in the production of clinical problems after operation. The lucid description1 acts as a catalyst to stimulate further careful investigation of the aetiology of these defects.

The peripatellar plexus presents a new focus of attention and may be the hitherto unrecognised feature which has prevented satisfactory explanations of postarthroscopy defects. The bundles of nerve fibres which provide connections for afferents from the plexus and project as accessions towards the posteriorly situated nerve trunks are vulnerable and likely to be interrupted when incisions as large as 4 cm are made. Given that the morphological arrangements in man are comparable to those in other mammals it is possible to visualise the effects of damage to these components. Conduction of position-sense from conventional receptors, whether encapsulated or not, will be damaged with resultant functional defects including modality imbalance. In addition to fibres which arise in periarticular tissue, those which emerge from intra-articular structures such as menisci1,11 and cruciate ligaments2 may have been severed. There is some evidence accumulating also that secondary afferents from adjacent muscle spindles pass through the peripatellar plexus “en route” for entrainment as accessions to the nerve trunks in the popliteal space.2 Interference with this function through surgical damage may well impair position-sense in the affected limb. It is also reasonable to expect in normal physiological circumstances, that the plexus may contribute as a transducer and as a modulator of protective information which is passing through its meshwork.

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References


Matters arising


Swash replies:
Wilson suggests that damage to the peripatellar nerve plexus could account for the defective movement sensation I have described after open lateral meniscectomy. This defect in sensation is not accompanied by any discernible change in static sense of position in the joint, although there is an associated alteration of motor control. These features are difficult to explain by dysfunction of any single class of sensory receptor. The concept that interruption of nerve pathways in the peripatellar plexus could result in interference with the accession of afferents emerging from intra-articular receptors, and with secondary afferents from muscles acting across the knee, is attractive and may be susceptible to experiment by local anaesthetic blockade of this plexus. These matters are important not only for theoretical reasons, but in the management of sports injuries, and in the understanding of falls in elderly persons with osteoarthritis joints.

Reference


Alternating unilateral jaw spasm due to matoclopramide

Sir: Thompson et al. described eight cases of unilateral jaw spasms of which three were masticatory in nature. Three of the eight cases were painful. None of the eight cases was drug induced. The following case illustrates once again that drug induced movement disorders often mimic the naturally occurring ones, even those that are rare.

A 66 year old woman was seen because of painful jaw spasms which began 2 hours previously. She described having spasms on each side in a seemingly random pattern. In the emergency room, she was witnessed by other observers to have two episodes of involuntary right jaw spasm, each lasting about 5 minutes. When I examined her no neurological abnormality was found and she had full range of motion of the jaw without any pain. She could not induce a spasm. After approximately 10 minutes she developed left masster jaw spasm with the jaw deviated mildly to the left. In addition, there was a mild dystonic contraction of the orbiculari oris and oris. During this time speech was dysarthric due to jaw clenching and the patient complained of severe pain. A repeat neurological exam was otherwise normal. There was no other evidence of dystonia and movements in other body parts did not exacerbate the jaw spasm. Intravenous diphenhydramine 50 mg was given and the episode abated over 1-2 minutes. Diphenhydramine 50 mg was given orally in addition at that time and thrice daily for the next 3 days. There were no recurrences.

The patient had taken metoclopramide 10 mg tid for one month, ending one month before this episode, along with librax (chlor Diazepoxide hydrochloride 5 mg, and Cidinium bromide 2:5 mg) two tablets per day. Two days before the spasms she had resumed metoclopramide 10 mg tid and the day before she had stopped the librax. Other medications included macrodantin, sulfamethoxizole, trimethaprim for a urinary tract infection and amiloride 5 mg hydrochlorothiazide 50 mg (Moduretic) for hypertension. Her past medical history included an ileal resection for bowel infarction, spastic colitis, hypertension, total abdominal hysterectomy and bilateral salpingoophorectomy for endometrial carcinoma 16 years previously, cholecystectomy and appendectomy 48 years previously, kidney stones and recurrent urinary tract infections. She stated that she had suffered similar jaw spasms approximately 18 years before but she could not recall any details. For several years, she had had no neurological symptoms and between episodes of jaw spasms her neurological examination was normal. She had no jaw or dental problems. The onset of the syndrome in close association to starting metoclopramide, a dopamine blocking agent, stopping the anticholinergic agent Cidinium hydrochloride and the striking reaction to diphenhydramine make the diagnosis of an acute dystonic reaction as certain as possible. The normality of her post-ictal examination also supports this.

This case is unusual for several reasons. As discussed in the paper by Thompson et al., unilateral jaw spasms are themselves rare. Acute dystonic reactions, like kinaesthesia is quite symmetric when it involves the jaw. Acute dystonic reactions are rarely painful. Oftentimes acute dystonic reactions can be overcome, at least temporarily, by volition, although once the conscious focus is lost the dystonia re-emerges. Acute dystonic reactions tend to last for hours if untreated. Finally, an intermittent alternating dystonia is (I think) unreported. An explanation for this extraordinary reaction is not apparent.

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Reference


Pseudotumour cerebri with amiodarone

Sir: The letter by Fikkers et al. prompts us to report a similar case of pseudotumour cerebri in a patient taking amiodarone.

A 52 year old man with Wolf-Parkinson-White syndrome was treated by amiodarone because of recurrent exercise-induced paroxysmal tachycardia. In November 1985 the dose was raised to 400 mg/day, five days a week. In January 1986 he noticed gradual loss of vision in the left eye; he felt tired and irritable. There was no headache, nausea or vomiting. The neurological examination on 3 February 1986 showed bilateral papilloedema; there were no focal or lateralising signs. The general examination was unremarkable. The blood pressure was 140/85 mm Hg and the electrocardiogram showed a sinus rhythm. Ocular examination revealed central deposits typical of amiodarone keratopathy. Visual acuity and colour vision were normal. There was a partial field defect in the nasal inferior quadrant of the left eye, confirmed by computerised perimetry. Pattern-shift visual evoked responses were within normal limits. Fluorangiography showed dilatation of the peripapillary capillaries and increased fluorescence beyond the edge of the papill on the late views, consistent with bilateral papilloedema.

A CT scan of the brain and orbitae was normal. A dural sinus occlusion was excluded by a normal intravenous digital subtraction angiography of the intracranial vessels. Blood and urine tests were normal. At lumbar puncture, the opening pressure was normal.