REVIEW

Current issues in arthrogenous inhibition*

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Abstract

Joint disease commonly results in severe weakness of associated muscles. Efforts to restore strength are often unsuccessful, even in the absence of pain. This is because of the underlying inhibition of motoneurones by afferent signals from in and around the affected joint, 'arthrogenous inhibition'. This phenomenon has received scant scientific attention, but several experimental techniques are now available with which it can be studied in man. Animal studies suggest possible neurophysiological mechanisms. Selective atrophy of different muscle fibre types, perhaps implying selective inhibition of different types of motor unit, remains unexplained, however. The severity of arthrogenous inhibition can be temporarily reduced by silencing afferent traffic but none of the techniques is yet generally applicable in practice. An alternative therapeutic approach is to produce involuntary muscle contractions by electrical stimulation. The effectiveness of therapeutic electrical stimulation may depend on the frequency and other characteristics of the stimulus.

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Severe weakness and wasting of muscles acting across an inflamed or injured joint (Charcot's 'atrophic articular paralysis'[1]) is a common clinical problem. It causes disability and probably predisposes the joint to further damage. The proper management of patients with joint problems includes efforts to prevent or reverse the weakness.[2–4] The 'atrophic' element results in part from joint immobilisation (fig 1). The 'paralysis' is due to reflex inhibition of motoneurones (fig 1). The importance of reflex inhibition, although well recognised by Charcot and his contemporaries,[5] is usually forgotten in standard textbooks. After a brief summary of the investigative background, this review discusses current issues in arthrogenous inhibition, highlighting areas of potential therapeutic relevance.

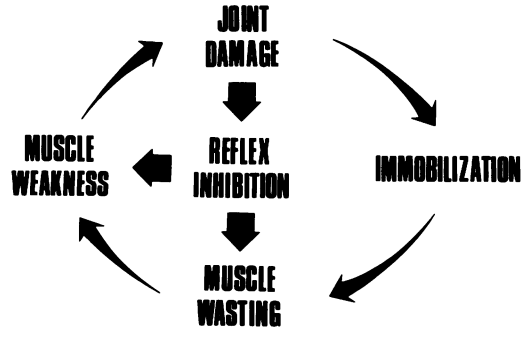

Figure 1 Interaction of immobilisation and reflex inhibition in the production of arthrogenous muscle weakness. (Reproduced, with permission, from Stokes and Young.[6])

Background

SURGICAL MODEL

Patients undergoing arthrotomy and meniscectomy provided an 'experimental model' of standardised joint damage for our earlier studies of reflex inhibition. The maximal voluntary activation of the quadriceps was measured as the integrated, rectified, surface electromyogram recorded during a maximal, voluntary, isometric contraction of the quadriceps. Bilateral measurements were made before and at intervals after the operation. Inhibition was calculated as the percentage reduction in maximal voluntary activation from the preoperative value for the same muscle. This simple approach yielded a good deal of information.[6] Voluntary activation of the quadriceps of the operated limb was severely inhibited (typically by about 70%) for at least 72 hours after the operation. Even two weeks after the operation 30–40% inhibition was not uncommon. It was not simply due to pain, but could be reduced by infiltration of local anaesthetic into the affected joint.[7] Inhibition was worse when an effusion was present and was reduced, but not abolished, by aspiration of the effusion. The severity of inhibition was less if the isometric contraction efforts were made with the joint in a flexed position than if they were made with the joint in an extended position. The phenomenon was not simply the result of prolonged, perioperative tourniquet ischaemia.[8] Later, it became apparent that inhibition was less severe after arthroscopic meniscectomy and probably owed more to the arthrotomy (or its suturing) than to the meniscectomy itself.[9]

EXPERIMENTAL KNEE INFUSION

Reflex inhibition has also been studied during the infusion of fluid into the knees of normal

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volunteers.[10-14] The findings of these studies have been reviewed.[15] They confirmed that the phenomenon of reflex inhibition occurs independently of the sensation of pain. Even small volumes of infusion fluid produced substantial inhibition; just 20–30 ml of infusion produced a 50–60% reduction in the maximal voluntary activation. Our studies concentrated on isometric contractions, but others have shown that the ability to perform isokinetic contractions is also inhibited by experimental infusions.[14] It is not only voluntary activation of the quadriceps which is inhibited. The infusion of fluid into a normal knee also reduces the level of excitation of the quadriceps' anterior horn cells. This is seen as a reduction in the quadriceps' H reflex (the monosynaptic, reflex, motor response to electrical stimulation of spindle afferent fibres). This applies when the H reflex is recorded at rest[13] and when it is recorded against a background of voluntary muscle activity.[12 16] We also attempted to elucidate some of the interneuronal connections of the joint afferents excited by knee infusion. There was spatial facilitation of the responses to Ib activity from the gastrocnemius and soleus, implying convergence of the inhibitory joint afferent pathway and the Ib pathway.[12 16]

METHODS OF STUDY

Several experimental techniques can be used to study arthrogenous inhibition. The use of measurements of maximal voluntary activation has already been illustrated. The technique is easy to use and is valuable for serial measurements. A limitation is that pre-intervention and bilateral data are required for the findings to be fully interpretable. Moreover, there is always room for uncertainty about any measurement which is dependent on voluntary effort, especially in situations where pain may be present or may be feared.

Changes in the measured strength of maximal voluntary contractions can also be used to follow changes in the severity of inhibition, but are subject to the same limitations as measurements of maximal voluntary activation. In addition, their interpretation is complicated by the effect of the degree of atrophy which is present. Their use is limited to short term serial measurements (when muscle size is presumed not to change) or to situations where bilateral strength measurements are compared in combination with bilateral measurements of the cross sectional areas of the individual muscles. This demands the use of a transverse imaging technique: measurements of limb circumference are not sufficient as the atrophy may be highly localised.[17-19]

The twitch superimposition technique,[20 21] modified for use in large muscles,[22 23] is a valuable addition to the techniques available for studies of arthrogenous inhibition. Electrically stimulated twitches are superimposed on a voluntary contraction; the resulting increments in the force record are inversely proportional to the completeness of voluntary activation. A major advantage is that this can be calculated from a single measurement, with no need for comparison with the other limb or with data from before the operation. Nevertheless, problems of interpretation remain; incomplete activation may be due to incomplete voluntary effort or to inhibition.

The severity of inhibition can be examined as inhibition of the H reflex. This has the advantage that it removes the problems of interpretation created by doubts about motivation in voluntary contractions. The recordings, however, can be technically difficult and so are probably best used for studying acute responses, and even then, only in the context of an experiment.

Whichever technique is used, it is essential to consider whether the measurements are made with the joint at the most appropriate angle for the purpose of the study. Isometric knee extension is more severely inhibited with the knee in extension than when it is in flexion.[6 24]

What neurophysiological mechanisms contribute to arthrogenous inhibition?

There are several different mechanisms by which articular and periarticular pathology might reflexly inhibit anterior horn cells. They are not mutually exclusive. Indeed, there may be a good deal of overlap between them.

INCREASED JOINT AFFERENCE

There is no doubt that reflex inhibition can be caused by activity in joint afferents excited by joint inflammation or increased tension in the joint capsule, or both. Dorsal root section prevents the muscle wasting produced by experimental joint inflammation in animals.[25 26] Intra-articular anaesthetic increases quadriceps maximal voluntary activation after open meniscectomy,[6] increases both maximal voluntary activation[10] and isokinetic torque[14] after experimental intra-articular infusion, further increases force after aspiration of an effusion,[27] and prevents suppression of the H reflex by experimental intra-articular infusion.[13] Furthermore, quadriceps strength is not reduced by the infusion of a large volume of fluid into a Charcot joint.[10] The precise characteristics of the receptor(s), however, remain uncertain. Reflex muscle inhibition provoked by periarticular pathology can also be relieved by selective infiltration of the source with local anaesthetic.[6]

DECREASED JOINT AFFERENCE

It seems likely that rupture of the anterior cruciate ligament may result in the loss of afferent input from receptors in the cruciate ligament itself. As argued by Newham *et al*,[28] such a change in sensory input might contribute to the arthrogenous inhibition seen in these patients, but there is no direct evidence.

DECREASED DESCENDING TONIC INHIBITION

A small increase in cutaneous or subcutaneous afferent activity can inhibit anterior horn cells.[29 30] This effect is usually held in check by descending tonic spinal inhibition.[31] Studies in decerebrate animal preparations raise the possibility that joint pathology may reduce this tonic descending spinal inhibition, thus allowing the inhibition of anterior horn cells by what would normally be trivial afferent activity.[32] This possibility is increased by the fact that some ascending tract cells with a receptive field in the knee joint have convergent input from other tissues.[33] The convergent input may even be from the contralateral knee;[33] unilateral knee inflammation can cause increased responsiveness of ascending tract cells to ipsilateral and contralateral passive knee flexion.[32] This is of particular interest as Newham *et al*,[28] using twitch superimposition, have produced results which suggest a degree of contralateral arthrogenous inhibition in patients with unilateral chronic rupture of the anterior cruciate ligament. (It would have been difficult to show this with other techniques, which are dependent on comparison of the affected and unaffected limbs.)

ENHANCED FLEXION REFLEX

The flexion reflex includes activation of the hamstrings and reciprocal inhibition of the quadriceps.[34] Thus anything which facilitates the flexion reflex may provoke inhibition of the quadriceps. In the decerebrate spinal rat, the responsiveness of the hamstring α motoneurones to a standardised pinch of the ipsilateral toes was greatly and persistently increased after the intra-articular injection of an irritant.[35] It seems possible that this kind of mechanism might play a part in some examples of clinical reflex inhibition. Clearly, however, it cannot explain all examples of arthrogenous inhibition as the flexion reflex response was not affected by an intra-articular injection of saline (fig 2).

In our studies of the suppression of the human quadriceps H reflex by the intra-articular infusion of saline, convergence with putative flexion reflex afferents was shown in one of two subjects in whom it was sought.[15]

UNPERCEIVED 'PAIN'

Although the phenomenon of reflex inhibition can be independent of the conscious appreciation of pain,[7 10 13 14 24 28 36 37] that does not exclude the possibility that afferent traffic from nociceptors may yet be an important contributory factor, even if it does not rise to consciousness. This possibility is linked with the earlier discussion of a possible role for the flexion reflex, as the prolonged facilitation of the flexion reflex by intra-articular mustard oil is possibly mediated by unmyelinated C afferent fibres.[35]

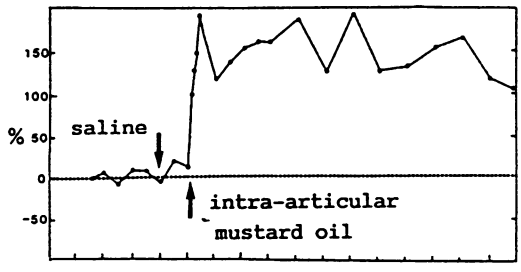

Figure 2 Effects of intra-articular saline and intra-articular mustard oil on the α motoneurone response of the hamstrings, of the decerebrate spinal rat, to pinching the ipsilateral toes. Time markers every 10 minutes on the abscissa. (Reproduced, with permission, from Woolf and Wall.[35])

Which motor units are inhibited?

Motor units differ in their pattern of recruitment and in the mechanical and histochemical characteristics of their muscle fibres. Is arthrogenous inhibition selective in the motor units which are inhibited and, if so, what are the determinants of its selectivity? This question is relevant to understanding the functional consequences of arthrogenous inhibition and perhaps also to the choice of therapeutic interventions to reverse its effects.

SELECTIVE ATROPHY OF FIBRE TYPES

Muscle biopsy samples from patients with joint pathology may show selective atrophy of type I or type II muscle fibres (presumably indicating selective inhibition of low or high threshold motor units respectively). In a review of the histochemical characteristics of arthrogenous muscle atrophy we concluded that it was not possible to identify the clinical features which determine whether an individual patient has selective type I atrophy, selective type II atrophy, or equal atrophy of the two principal fibre types.[38] Despite further histochemical studies of arthrogenous wasting since that review,[39–45] the conclusion remains much the same. The only, limited, exception is that surgical biopsy samples from vastus lateralis are unlikely to show evidence of type I fibre atrophy in patients with isolated injuries of a meniscus, whereas type I atrophy (with or without type II atrophy) is common, but not invariable, in patients with chronic rupture of the anterior cruciate ligament.[46] Future studies should probably be designed to test whether selective atrophy of one or other fibre type can be explained by clinical features such as the chronicity of the disorder, the nature of the therapeutic exercise which has already been performed, the presence or absence of pain, the presence or absence of effusion, or the degree of immobilisation. For the moment, however, we still do not know how to make the jump from clinical features to microscopic findings.

CONTRACTION SPEED AND SEVERITY OF INHIBITION

An alternative approach to identifying selectivity of arthrogenous inhibition is to examine the effect of contraction speed on the severity of inhibition. Greater inhibition at slow speeds of contraction might imply selective inhibition of motor units comprising (the slower contracting) type I muscle fibres.

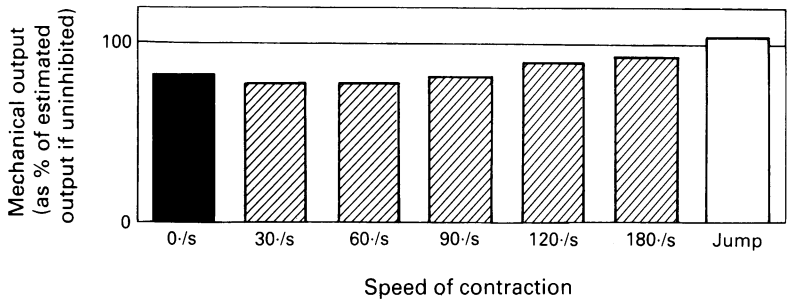

Figure 3 Effect of speed of contraction on apparent severity of arthrogenous inhibition. Drawn, after recalculation, from published data of Newham et al.[28]

This has been done for patients with a chronic rupture of the anterior cruciate ligament.[28 47] The two studies considered patients with chronic rupture of the anterior cruciate ligament and both found greater inhibition of slow contractions (e.g. fig 3). This might be expected to be associated with selective type I fibre atrophy, a common (but not invariable) histochemical finding in such patients,[46 48 49] but there was no evidence of this in the only study to combine biopsies with measurements of inhibition at different speeds of contraction.[47]

Prognostic significance of arthrogenous inhibition

The presence of a moderate degree of arthrogenous inhibition does not preclude improvements in quadriceps strength with the performance of voluntary rehabilitative exercise in patients with isolated rupture of the anterior cruciate ligament[50] or with early osteoarthritis of the knee.[51] Nevertheless, it seems likely that severe arthrogenous inhibition limits the potential response to voluntary therapeutic exercise. Conservative treatment has an important place after cruciate ligament rupture, but it is difficult to predict which patients will not do well.[52 55] It has been suggested that, after cruciate ligament rupture, perhaps the severity of arthrogenous inhibition of the quadriceps might predict which patients are not going to improve with conservative treatment,[50] so that they could proceed, without delay, to reconstructive surgery. A prospective trial is indicated.

Therapeutic afferent blockade?

LOCAL ANAESTHESIA

One approach to the prevention of arthritic amyotrophy is to block the afferent signals responsible for the inhibition. This can be achieved by infiltrating local anaesthetic in and around the source of the abnormal stimuli, but the effect is too short lived for practical clinical application.

EPIDURAL ANAESTHESIA

Arvidsson *et al* have shown that epidural lidocaine the day after surgical repair of the ruptured anterior cruciate ligament greatly increases the activation of the quadriceps, which can be achieved by a maximal voluntary effort.[56] They assumed that this was due to the associated decrease in pain.[57] This need not be so; it is equally plausible that it was due to a reduction in arthrogenous inhibition as a result of blockade of afferents over and above those subserving pain.

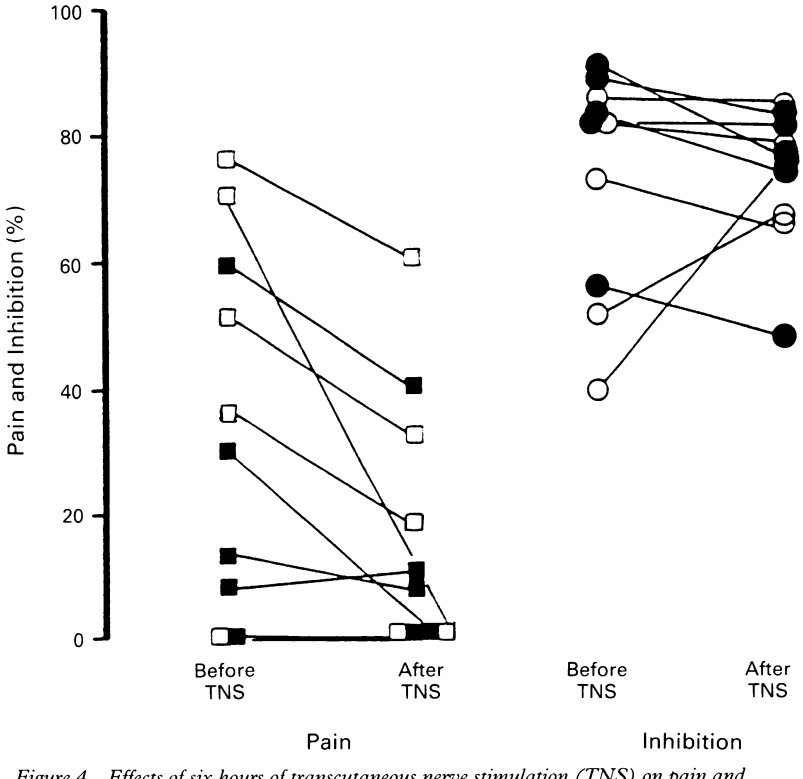

Figure 4 Effects of six hours of transcutaneous nerve stimulation (TNS) on pain and arthrogenous inhibition of the quadriceps during maximal voluntary isometric contractions two days after open meniscectomy. Previously reported only in abstract form.[58] *Open symbols = control group; closed symbols = treatment group.*

TRANSCUTANEOUS NERVE STIMULATION

Transcutaneous nerve stimulation produced a small increase in maximal voluntary quadriceps activation after surgical reconstruction of the anterior cruciate ligament[57] and after open meniscectomy.[58] Arvidsson and Eriksson ascribed this to an associated small reduction in pain.[57] Stokes *et al*, however, showed a dissociation between the effect on pain and the effect on maximal voluntary activation;[58] whereas active and placebo transcutaneous nerve stimulation reduced pain, only the active treatment resulted in an increase in maximal voluntary activation (fig 4). Unfortunately, in both studies, the effect of transcutaneous nerve stimulation was too small to be of clinical value. Nevertheless, it does suggest that this treatment modality deserves further study. Perhaps its effectiveness might be greater with different stimulation parameters or different placement of the electrodes.

ICE

The local application of ice is a technique already commonly used by physiotherapists to reduce swelling and pain. It seems possible that it might also have an effect on arthrogenous inhibition, but there is no published report of the scientific evaluation of this possibility.

Electrically stimulated contractions

Instead of blocking the inhibitory afferent traffic, an alternative therapeutic approach is to 'bypass' the inhibited anterior horn cell, performing therapeutic muscle contractions by electrical stimulation of the motor nerve. It is difficult to activate a large proportion of the quadriceps in this way without unacceptable discomfort. Nevertheless, there is increasing evidence that this is an effective means of preserving muscle mass and muscle strength. It is also possible that the degree of effectiveness may depend on the choice of stimulation frequency.

Maximal tetanic contraction of a small muscle in response to electrical stimulation of the motor nerve trunk can be completely acceptable (e.g. adductor pollicis and the ulnar nerve at the wrist[59]). This is not so for the quadriceps; most people find it alarming and unpleasant to have high force quadriceps contractions induced by stimulation of the femoral nerve trunk. Instead, maximal tetanic contractions of a part of the quadriceps can be produced without undue discomfort. The terminal branches of the nerve are stimulated by pad electrodes placed over the muscle. Most people find this acceptable if only about 30–40% of the muscle is induced to contract.[59] Maximal contraction of up to perhaps 60–80% of the muscle can be achieved,[60 61] but can be unpleasant.[60]

To improve the strength of normal volunteers, electrically stimulated contractions offer little or no advantage over voluntary contractions.[61–64] This should not be surprising, as most people can produce maximal activation of the whole quadriceps with a voluntary effort.[22] On the other hand, if (*a*) arthrogenous inhibition prevents voluntary activation of, say, half of a muscle, (*b*) electrical stimulation activates, say, half of the muscle, and (*c*) there is no interaction between factors determining which fibres are activated in these situations, then a combination of voluntary and stimulated contractions would exercise 75% of the muscle.

Eriksson and Häggmark showed that, after surgical repair of the anterior cruciate ligament, electrically stimulated contractions of the quadriceps were associated with a better result in terms of clinically estimated muscle bulk and strength, and in terms of preservation of the muscle's oxidative enzyme activity.[65] With similar patients, Wigerstad-Lossing *et al* compared a combination of stimulated and voluntary contractions with voluntary contractions alone.[66] The addition of electrical stimulation resulted in less severe losses of quadriceps strength and cross sectional area and probably also less severe losses of activity of an oxidative enzyme and a glycolytic enzyme.

There are two reports of effective therapeutic stimulation in patients with osteoarthritis. In one study, after one hour of weak, intermittent stimulation every day for 28 days, the treated patients showed no biopsy evidence of the atrophy, of type I and type II fibres, seen in the untreated patients.[44] It is hard to understand how the stimulation had its effect as the stimulated contractions generated only 5% of the force of maximal voluntary contractions. In the other study, 10 seconds of stimulation were given during each cycle of continuous passive movement for seven days after total knee arthroplasty.[45] This prevented the atrophy, again of both fibre types, seen in patients given continuous passive movement without stimulation.

STIMULATION CHARACTERISTICS

The effectiveness of therapeutic stimulation may depend on the stimulation frequency. There is a short report of the effects of different patterns of stimulation (with and without voluntary exercise) on the discrepancy in strength between the limbs measured two weeks after arthrotomy and meniscectomy.[67] Its findings suggest that stimulation can help preserve quadriceps strength, despite the presumed presence of postoperative inhibition, but that a 1 ms square wave at 50 Hz may be more effective than a 5 kHz signal whose amplitude varies sinusoidally at 50 Hz. The importance of the choice of stimulation characteristics is also evident in a study of the role of electrically stimulated contractions of the first dorsal interosseous muscle in the restoration of the function of the hands of patients with rheumatoid arthritis.[68] The functional gains were much smaller in patients stimulated at 10 Hz than in those for whom the frequency was modelled on that recorded from a fatigued motor unit in the first dorsal interosseous muscle of a normal hand.

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