The role of skin areas adjacent to extensor muscles in motor neurone excitability: evidence bearing on the physiology of Babinski's response¹

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Cutaneous afferent nerve fibres fall into two categories of fibre sizes, group II (6 to 16 μ) and group III (2 to 3 μ) (Ranson, Droegemueller, Davenport, and Fisher, 1935). Afferent fibres from the skin, together with high threshold joint afferents and groups II and III muscle afferents, are classed as flexion reflex afferents, part of the nociceptive reflex system of Sherrington (1906) subserving the basic function of protective withdrawal from potentially noxious stimuli. Yet flexion is not an invariable response to stimulation of cutaneous nerves and exceptional instances of ipsilateral extension were already compiled by Brown and Sherrington in 1912. Creed, Denny-Brown, Eccles, Liddell, and Sherrington (1932) later described the occasional reversal of reflex pattern with change of the reflex stimulus parameters, concluding that the reversal of response indicates that one of the reflexes is ordinarily concealed. Clearly this inversion is apposite to the problem of the pathophysiology of Babinski's reflex, in which the normal plantar flexion of the toes on stimulation of the skin of the lateral planta alters to extension of the large toe with fanning of the other toes.

A peripheral extension from the concept of mutually antagonistic reflexes evocable from the same cutaneous nerve is that of preferential facilitation of either flexor or extensor motor neurones from stimulation of certain specific skin areas. This concept would provide the substrate for the occasional reversal of reflex on stimulation of a cutaneous nerve, composed of an intermingling of nerve fibres from diverse skin areas. Hagbarth (1952) first reported that extensor muscles were inhibited by stimulation of most skin areas of the hind limb in spinal and decerebrate cats, but were excited by stimulation of cutaneous areas over the extensor muscle; whereas flexor muscles were excited by

stimulating most skin areas of the hind limb but inhibited by that located directly over the antagonistic extensor muscle. Kugelberg, Eklund, and Grimby (1960) also observed that extensor responses were evoked preferentially from certain skin areas in humans; stimulation of the buttock resulted in extension of the toes, ankle, hip, and trunk; whereas stimulation of the plantar surface of the heel resulted in extension of the toes and ankle. Kugelberg and Hagbarth (1958) reported that reflex discharges, recorded electromyographically, altered gradually when progressing from one stimulating point to another at a distance, although the overall pattern of discharge suggested an interaction of conflicting reflexes at any given cutaneous area. Gassel and Ott (1969) found reliable changes of excitability in triceps surae motor neurones, mediated by group II fibres, with serial alterations in localization of cutaneous stimuli on the dorsal and plantar surfaces of the distal foot. Evidence was advanced to support the concept of an inherent and intricate organization of the receptive fields of extensor facilitatory and extensor inhibitory cutaneous areas, with considerable overlapping and interaction.

Hagbarth's (1952) report of characteristic effects on extensor or flexor motor neurone excitability evoked by stimulating adjacent skin areas could well represent a fundamental rule regulating the pattern of cutaneous influence. It was therefore decided to submit the thesis to test in normal humans.

METHODS

Fifteen normal subjects were investigated. The subjects included paid volunteers and laboratory personnel selected from a total group of 27, with a capacity to remain relaxed in a fixed position throughout the investigation. The subjects were placed in a prone position with legs extending over the end of the couch. The leg was supported firmly in a heavy metal frame which was fixed to a metal stand separated from the couch. The leg frame

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was adjustable in height and angle. An automatic solenoid hammer was attached to the leg frame by a metal supporting device with two complex joints. These joints permitted movement of the hammer in all directions and made it possible to position the hammer at a fixed distance (3 cm) directly over the Achilles tendon (Gassel and Ott, 1969). The solenoid was triggered from the stimulator (DISA Multistim) and the delay to the tendon tap was 55 msec. This delay was measured before each experiment. The solenoid driven reflex hammer had a built-in microswitch which closed the circuit on percussion and triggered the sweep of the electromyograph. A regular and stabilized blow was delivered. Electromyographic activity was recorded with two non-insulated needle electrodes placed 0.5 cm apart over the distal belly of the triceps surae muscle in order to minimize stimulus artefact. Stimulus artefact might block the amplifiers on stimulation at the posterior leg position and give a spuriously low average when led into the computer (see below). It was important to display the reflex at short conditioning-test intervals in order to control the source of error. The potentials were amplified and displayed on the DISA 3-channel electromyograph.

The recovery curve of the ankle jerk following single-shock cutaneous stimulation was evaluated. A DISA multistimulator was utilized with a maximum voltage output via transformer of 250 V. A regular and unvarying single 3 msec duration square-wave pulse was delivered through subcutaneous stainless steel electrodes placed 2 cm apart. The conditioning shock, painful, but of tolerable intensity was delivered to the midleg at the antero-lateral, antero-medial, or posterior surface. The shock was followed at intervals ranging from 0 to 5,000 msec by the ankle jerk reflex. Maximal reflexes were elicited at a frequency of one every 10 seconds. Generally

eight reflexes were averaged by computer (CAT 1000) in establishing a control of test values. In some studies 20 reflexes were averaged when control variations were large. The controls were repeated at intervals during investigation and are indicated in the graphs at the appropriate test intervals.

RESULTS

The range of patterns of recovery curves of the monosynaptic reflex (ankle jerk) conditioned by a single shock stimulus delivered through subcutaneous electrodes on the antero-medial, antero-lateral, and posterior midleg are represented in Figs. 1, 2, and 3.

There is some variation among individuals in the characteristics of the recovery curve at any particular stimulating point, but no systematic difference found between any of the differently located stimuli. The individual pattern on stimulation at any of the three leg positions showed considerable consistency, with lesser differences than the range of variation at any particular locus of stimulation.

The usual onset of significant effects was at 20 to 40 msec with a mild facilitation which lasted until 90 to 100 msec. There was an additional phase of facilitation at about 100 to 250 msec with a peak at about 150 msec. This phase was sometimes followed by a mild inhibition, with gradual return of the reflex excitability to control levels at 3 to 5 sec.

The range of recovery curves from the same individual (subject R.B.) after conditioning with a stimulus to each of the three midleg locations on

FIG. 1. Recovery curves of the monosynaptic reflex (ankle jerk) conditioned by a stimulus to the midleg, antero-lateral surface, in subjects K.L. and R.B. The controls are indicated in the graphs at the appropriate time intervals.

20 March 1969 is illustrated in Figs. 1B, 2B, and 3C. A fundamental similarity is evident in these graphs. The notable features are a slight early facilitation with irregular onset followed by a prominent facilitation at 100 to 200 msec. Similarly the results of stimulation on the antero-medial and antero-lateral leg of subject K.L. (Figs. 1A, 2A) are remarkably similar.

A repeat study at the same position was performed in three subjects. The results resembled those of the preceding study in two of these. The recovery curves of the third such repeat study are illustrated in Figs. 3C and 3D. The conditions of stimulation were similar except that in Fig. 3D, performed on 3 April 1969, the stimulus was experienced as rather more painful. It is observed that there is an early inhibition from 20 to 100 msec in Fig. 3D in place of the slight early facilitation in Fig. 3C, but that the late facilitation is unchanged. There was, therefore, a reversal of the early response on repeat testing. The occurrence of significant early inhibition was indeed rare at any position, and was found in but four of 35 recovery curves and all with posterior leg conditioning stimuli.

DISCUSSION

There was no local sign to the changes in excitability of the triceps surae muscle after stimulation of the skin about the midleg. Not only was there no

systematic difference in the recovery curves conditioned by stimuli at any of the midleg positions, but the recovery curves at different stimulus locations from the same individual were usually remarkably similar. The typical response was early facilitation with onset at 20 to 40 msec and lasting 90 to 100 msec, followed by a second phase of facilitation at 100 to 250 msec with a peak at about 150 msec. A similar two-phase pattern of excitability change of triceps surae motor neurones had been found previously on stimulation of the distal foot, plantar and dorsal surfaces. However, there was strong local sign to the early change in excitability, with facilitation on stimulation of the dorsal surface and inhibition on plantar stimulation of the distal foot. The nerve fibres mediating the excitability change can be judged by a comparative relationship of the latency of the excitability change with the calculated latency for central effects of group Ia afferents in humans (Gassel and Ott, 1969). A direct relationship exists between axon diameter within the myelin sheath and the conduction velocity, making it possible to judge the fibre size when the conduction velocity is known. The early change in excitability at 40 to 90 msec was of latency compatible with mediation by group II cutaneous fibres. A prominent late increase in excitability without local sign occurred at 110 to 250 msec; this change was calculated on the basis of latency of effect, to be mediated by group III fibres. The latencies of the

FIG. 3. Recovery curves of the monosynaptic reflex (ankle jerk) conditioned by a stimulus to the midleg, posterior surface, in subjects K.K., M.D., and R.B. Subject R.B. was studied at a later date (D), with a subjectively more intense conditioning stimulus.

early and late effects in the present study are compatible with the earlier attributions; to groups II and III cutaneous afferents respectively. It needs to be recognized that this method provides only an approximation of the functional nerve fibre sizes, and there are a number of provisos. The spectrum of nerve fibres mediating an excitability change cannot be judged directly from the duration of the event, as the skin stimulus undoubtedly induces after-discharge in the skin receptors and a train of nerve volleys. Even the onset is not related precisely to fibre size as the effect on motor neurone excitability is likely to be apparent only after a period of temporal summation (Lloyd, 1942).

An explanation alternative to that of late facilitation mediated by small cutaneous afferents is that of long-loop, spino-bulbo-spinal reflexes (Shimamura and Livingston, 1963). These reflexes are most easily and regularly evoked from stimulating cutaneous nerves. There is an ascending arc to the bulbar reticular formation, and then descending effects, resulting in serial and bilateral ventral root discharges. However, these reflexes reflect into flexor muscles only on cutaneous nerve stimulation (Shimamura and Akert, 1965).

The inversion in sign of the early excitability change, with inhibition from 20 to 100 msec, noted on repetition of the study of subject R.B. exemplifies

the concept of concealed reflexes (Creed *et al.*, 1932). One of the antagonistic reflexes is ordinarily concealed and evoked under special circumstances. The stimulus parameters were not significantly altered, although the conditioning shock was seemingly more painful in the later study. It should be noted, however, that inversion is by no means a regular occurrence after increasing the stimulus intensity. The latency and duration of the reversal of response indicates that larger skin afferents implicated in the excitability change with local sign (see above) also mediate the reversal of response and they may well be related phenomena. The concept of nascent, cutaneously-evoked, reactive propensity, apparent under special circumstances, is fundamentally that operative in Babinski's reflex. It has long been established that supra-segmental influences affect the pattern of the response to cutaneous stimuli. For example, the flexion reflex is more active in the spinal than decerebrate preparation and more active in the chronic than acute spinal animal; and in the exceptional instances in which Brown and Sherrington (1912) observed ipsilateral extension on cutaneous nerve stimulation, it was invariably with decerebrate preparations. The neurophysiological basis of the supraspinal control has been the subject of more recent study (Eccles and Lundberg, 1959; Holmqvist, 1961; Shimamura and Akert, 1965). An altered pattern of dominance of mutually antagonistic flexor and extensor responses to cutaneous stimuli resulting from altered suprasegmental influences is a likely pathophysiology of Babinski's reflex.

It therefore appears that there are mutually antagonistic excitability changes evocable from certain skin areas. One of these influences is dominant under ordinary circumstances, but evidence of an interaction or reversal of effect can be made manifest by changing the parameters of stimulation or by special recording techniques. Certain areas of the skin, most strikingly the dorsal and plantar surfaces of the distal foot, have been demonstrated to show specific patterns of dominance. Indeed, Sherrington (1906) and Kugelberg *et al.* (1960) observed that the characteristics of the flexion reflex varied with the localization of the noxious cutaneous stimulus resulting in appropriate withdrawal. However, Hagbarth's (1952) basic conclusion that extensor muscles are inhibited by stimulation of most skin areas of the limb, but are excited by stimulation of cutaneous zones located over the extensor muscle, is not supported by this study in normal humans. Hagbarth's results are based on experiments on spinal and decerebrate cats. Investigation of patients with established lesions of the nervous system is planned to clarify the apparent discrepancy in experimental results.

SUMMARY

This study reports the pattern and range of excitability changes in triceps surae motor neurones conditioned by single-shock cutaneous stimuli to the antero-medial, antero-lateral, and posterior midleg in a group of normal subjects. The usual change was an early period of facilitation with onset of 20 to 40 msec and lasting to 90 msec. There was a second phase of facilitation at 100 to 250 msec, sometimes followed by a mild inhibition with gradual return of excitability to control levels at 3 to 5 sec. The early change in excitability is attributed to effects mediated by group II cutaneous afferents and the later facilitation to group III fibres. There was no systematic difference in the recovery curves evoked by any of the differently located stimuli, and the curves conditioned at the various midleg positions in the same individual were usually remarkably similar. It was, therefore, not possible to confirm Hagbarth's experimental conclusions (1952), based on studies in decerebrate and spinal cats, that extensor muscles are inhibited by stimulation of most skin areas of the limb but are facilitated by cutaneous zones located over the extensor muscle.

An inversion in sign of the early excitability change, with inhibition from 20 to 100 msec, occurred on repetition of the study in one of three subjects. Evidence was advanced to support the concept of mutually antagonistic extensor facilitatory and extensor inhibitory reflexes excitable from a given skin area. One of these response propensities is dominant under ordinary conditions, but evidence of interaction or reversal of response can be made manifest by altered suprasegmental influences, changes in the parameters of stimulation, or by special recording techniques. This reversal of response is apposite to the pathophysiology of Babinski's reflex in which the normal plantar flexion of the toes on stimulation of the skin of the lateral planta alters to extension of the large toe with fanning of the other toes.

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