The differences in the times of these effects on shivering and on the subcutaneous temperature suggest that the changes in cold shivering do not take place through thermoreceptive reflexes from the integument of the body, but that they are the results of the direct action of the drugs on the central mechanism of thermoregulation. Usually all the effects of intraventricular injection of adrenergic drugs on cold shivering and on the subcutaneous temperature arose bilaterally and simultaneously, but in some experiments the effect appeared sooner on the contralateral side.

The results suggest that noradrenalin in cats can behave as the mediator in the activating and inhibitory neuronal systems responsible for the central regulation of cold shivering. The activating effect of noradrenalin in this case is mediated through α-adrenergic and its inhibitory effect through β-adrenergic receptors.

LITERATURE CITED

1. S. V. Anichkov, The Selective Action of Mediators [in Russian], Leningrad (1974).
2. K. P. Ivanov and T. M. Laryukhina, Fiziol. Zh. SSSR, 61, 1805 (1975).
3. J. Bligh, W. Cottle, and M. Maskrey, J. Physiol. (London), 212, 377 (1971).
4. B. Dhawan and P. Dua, Br. J. Pharmacol., 43, 497 (1971).
5. W. Feldberg, Proc. R. Soc. London, Ser. B., 191, 199 (1975).
6. W. Feldberg and R. Myers, Nature (London), 200, 1325 (1963).
7. R. Lomax, Int. Rev. Neurobiol., 12, 1 (1970).
8. G. Metcalf and R. Myers, J. Pharm. Pharmacol., 27, 616 (1975).
9. E. Preston, Can. J. Physiol. Pharmacol., 51, 472 (1973).
10. C. Raper and W. McCulloch, Med. J. Aust., 2, 1331 (1971).
11. K. Tangri, A. Bhargava, and K. Bhargava, Neuropharmacology, 13, 333 (1974).

DEPENDENCE OF ELECTRICAL ACTIVITY OF A MUSCLE ON ITS LENGTH DURING DEVELOPMENT OF A CONSTANT FORCE

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The dependence of the integral electromyogram (EMG) on the length of the muscle during the development of constant force was investigated in relation to the human biceps brachii muscle, while performing flexion at the elbow in a horizontal plane against a constant force of resistance. Both with a static load and during movement the integral EMG is increased several times during shortening of the muscle. The causes of the increase in electrical activity are discussed.

KEY WORDS: biceps brachii muscle; integral electromyogram; change in length.

In so far as the force developed by a muscle during contraction is a linear function of its excitation, the surface electromyogram (EMG), which reflects the level of excitation, can also characterize its strength. In most investigations [1, 4, 9] during isometric contraction of the muscle a near-linear relationship was obtained between the integral EMG and force for small loads. Attempts to extend this result to movements with a change in length of the muscle showed that the relationship is influenced in particular by the character of the movements (shortening or lengthening of the muscle) [3, 11]. The dependence of electrical activity of the

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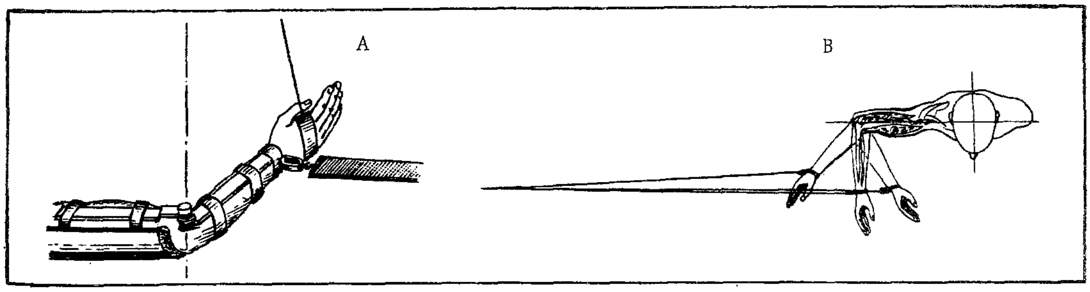

Fig. 1. Scheme of experiment (A) and points of attachment of biceps brachii muscle (B).

muscle on its length during the development of a constant force has not been investigated quantitatively, for most workers used a constant external moment applied to the muscle, whereas the force changed with a change of angle at the joint, i.e., with a change in length of the muscle [14]. Only in one investigation, on muscles of the stump of a human arm, in which skin and muscle canals had been formed, was it shown that during isometric contraction with maximal force the electrical activity of the shortened muscle was considerably greater than that of the stretched muscle [6]. Since that investigation was performed under static conditions on traumatized muscles, it was decided to obtain experimental data on the amplitude of the EMG in relation to muscle length during the development of constant force both under static conditions and during movement.

The results of such experiments using the biceps brachii muscle are described in this paper.

EXPERIMENTAL METHOD

Flexion of the arm at the elbow, performed in the horizontal plane, was studied. The subjects were seated with the arm resting on a support (Fig. 1A). The absence of hinges for the forearm reduced the inertia of the system. The elbow itself acted as the hinge. For convenience of the subject the forearm was supported by a special sling around the hand. The point of suspension lay above the axis of the joint. During the experiment the subject's hand was in a position of semipronation, in which flexion is produced mainly by the biceps brachii muscle [8, 12]. A strain gauge (ring-shaped) was secured to the hand to record the force. So that during flexion of the elbow the force, and not the moment acting on the muscle, remained constant, rubber bands about 6 m in total length, creating a resistance of 1 kg force, were attached so that in all investigations of the static positions and during movement the direction of the force acting on the muscle was virtually parallel to the direction of the muscle tendon (Fig. 1B). In reality, because of the nonlinear properties of rubber, the force varied by 20%. The angle at the elbow was measured by means of a potentiometric transducer. Movement began from an angle of 170° and ended at an angle of 50-55°. Measurements under static conditions were carried out every 20°. Changes in length of the biceps muscle were determined from the known relationship [8]. For the amplitude of movements specified above the changes in length were about 20%. The EMG was derived from flat surface electrodes, amplified, and led to an integrator. For the amplitudes of input signal studied, the properties of the integrator were linear. In some experiments activity of the antagonist muscle – triceps – also was recorded. Eight clinically healthy subjects took part in the experiments. For each subject no fewer than 20 recordings under static and 50 to 60 under dynamic conditions were made.

EXPERIMENTAL RESULTS AND DISCUSSION

The great variability of the data required a large number of recordings, which were grouped on the basis of speed. Very rapid (ballistic) movements were not investigated, for they have a different type of activity from the slow movements: Two volleys of activity, separated by a period of silence, are observed in the agonist, and the volley of activity of the antagonist appears during the period of silence of the agonist [2].

Under static conditions, with a decrease in length of the muscle the integral EMG rises (Fig. 2A). For the length of the muscle corresponding to an angle of flexion at the elbow of 55°, the integral EMG was not less than three times greater in amplitude than when the length of the muscle corresponded to an angle of 160°. With an increase in the load the curve was steeper and the scatter of the experimental points greater. The integral EMG as a function of length of the muscle (angle at the joint), averaged for speeds from 26 to 43 deg/sec (bottom curve) and from 74 to 88 deg/sec (top curve) is shown in Fig. 2B. Clearly the electrical activity of the shortened muscle when developing the same force was 5 to 10 times greater than that of the stretched muscle; the curve corresponding to higher speeds of movement also was steeper. A typical record of flexion at the elbow is given in Fig. 3A. The dependence on speed for all lengths of the muscles is shown in Fig. 3B.

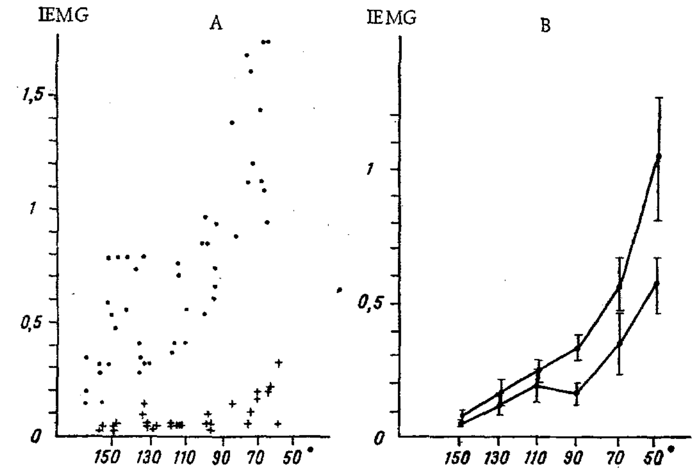

Fig. 2. Integral EMG (IEMG) as a function of length of muscle under static conditions and during movement. Forearm completely extended at the elbow, i.e., maximal length of muscle corresponding to an angle of 180°. In A: static conditions, crosses – load 1 kg, points – load 4 kg; in B: movement at speeds of between 26 and 43 deg/sec (bottom curve) and between 74 and 88 deg/sec (top curve).

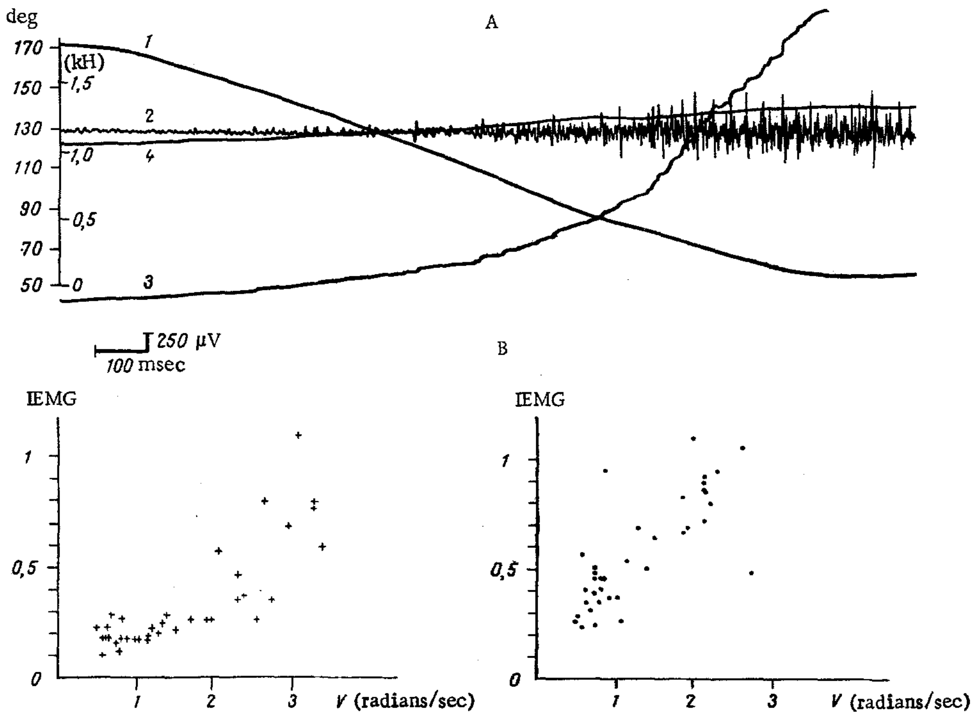

Fig. 3. Typical record of flexion at elbow (A) and of IEMG as a function of speed of movement (B). In A: 1) record of movement (angle at elbow); 2) EMG of biceps muscle; 3) IEMG; 4) force developed by muscle; in B: on left – length of muscle 342 mm (angle at elbow 110°); on right – length of muscle 332 mm (angle 90°).

Both during static loading and during movement the integral EMG thus increases in amplitude several times with shortening of the muscle.

Although the experimental conditions were chosen so as to ensure constancy of the external force, because of the nonlinear properties of rubber the force of resistance created by it was 20% greater for the shortened muscle than for the stretched muscle. The force developed by the muscle may also be influenced by the activity of the antagonist, for in this case the agonist has to overcome not only the load, but also the resistance of the antagonist. However, recording the EMG of the triceps muscle in these experiments did not reveal its activity, a result which agrees with observations by other workers [8, 10]. Forces of friction in the joint can evidently be disregarded [13], but the role of viscoelastic forces during movement must be taken into account. With an increase in the speed of movement to 3.5 radians/sec, the passive forces of resistance to movement almost doubled to reach 34 N [8].

The external force of resistance to movement was thus increased by 20%, friction in the joints can be disregarded, passive forces were not more than doubled during the movement, but electrical activity under these conditions increased by several times. The results cannot therefore be described purely by an increase in the load on the muscle.

Of the five muscles contributing to flexion of the forearm at the elbow (the biceps brachii, brachialis, brachioradialis, pronator teres, and extensor carpi longus), only the biceps was investigated. However, it has been shown [10] that during flexion of the elbow against resistance the activity of the biceps, brachialis, and brachioradialis muscles show identical changes. The increase in electrical activity of the biceps muscle could therefore not be due to a redistribution of activity among the agonist muscles.

The increase in amplitude of the integral EMG during shortening of the muscle might be attributable to an increase in the potential of the muscle fibers on account of their thickening. Observations of this sort have been made on the isolated frog muscle fiber [5]. However this effect is small. The electrical activity of the fiber falls by 25-30% in response to its stretching by 50%, and under the experimental conditions used the length of the muscle changed by only 20% (from 110 to 90% of the resting length), whereas the increase in IEMG amounted to hundreds of percent.

Investigations have shown that the stronger the muscle (i.e., the greater the load it can lift), the smaller the electrical activity recorded while supporting a given load. Maintenance of the same force in a relaxed muscle is accompanied by an increase in the number of actively functioning motor units, and in the frequency and synchronization of their activity [1, 7]. Presumably during shortening of the muscle its maximal force falls. For that reason, during movement with shortening of the muscle, the involvement of additional motor units is necessary to maintain a constant force. It can be postulated that the program preceding the beginning of a movement and calculated on the basis of a constant force of resistance must incorporate the recruiting of additional motor units during shortening of the muscle, and this can take place both directly via α pathways and also with the use of the mechanism of α–γ contraction.

LITERATURE CITED

1. R. S. Person, Electromyography in Human Investigations [in Russian], Moscow (1969).
2. R. W. Angel, in: International Symposium on Human Reflexes and Motor Disorders, Brussels (1976), p. 33.
3. B. Bigland and O. C. J. Lippold, J. Physiol. (London), 123, 214 (1954).
4. S. Bouisset, in: New Developments in Electromyography and Clinical Neurophysiology (ed. by J. Desmedt), Vol. 1, Basel (1973), pp. 547-583.
5. C. H. Hakanson, Acta Physiol. Scand., 41, 199 (1957).
6. V. T. Inman, H. J. Ralston, et al., Electroenceph. Clin. Neurophysiol., 4, 187 (1952).
7. V. T. Inman, J. B. de C. M. Saunders, and L. C. Abbott, J. Bone Jt. Surg., 26, 1 (1944).
8. F. Lestienne, Programme Moteur et Mécanismes de l'Arrêt d'un Mouvement Monoarticular, Lille (1974).
9. O. C. J. Lippold, J. Physiol. (London), 117, 492 (1952).
10. J. E. Pauly, J. L. Rushing, and L. E. Sceving, Anat. Rec., 159, 47 (1967).
11. J. Sherrer, J. Physiol. (Paris), 49, 376 (1957).
12. D. J. Simons and E. N. Zuniga, Am. J. Phys. Med., 49, 253 (1970).
13. S. Tadashi and M. Hakuhiko, J. Jpn. Soc. Lubric. Eng., 18, 90 (1973).
14. J. Vredenbregt and W. G. Koster, IPO Ann. Prog. Rep., 1, 94 (1966).