Scand J Rehab Med 2: 159–163, 1970

TOPICAL CRYOTHERAPY IN SPASTICITY

E. Knutsson

From the Departments of Clinical Physiology and Neurological Rehabilitation, Karolinska Sjukhuset, Stockholm, Sweden

ABSTRACT. The effects on resistance to passive movements, clonus and maximal contraction force produced by application of local cold over spastic muscles was studied in 30 patients. The passive resistance to stretch of the chilled muscles was reduced in 10 out of 15 cases. Clonus was abolished or diminished in the 8 patients displaying this type of exaggerated stretch reflex. Contraction strength did not increase significantly in the chilled muscles but the power of the antagonists was enhanced by more than 50% in 11 of 29 patients. Such an increase was invariably observed when the resistance to passive movement was lowered and a significant degree of voluntary activation preserved. No such increase was seen when abnormal vasomotor reactions prevented a local temperature fall or when for other reasons, probably an alpha spasticity, the muscular hypertonus was not reduced.

Application of local cold over a muscle usually reduces the phasic stretch reflex in healthy subjects (13). This depression seems to be the result of a fairly complex series of events. Thus, Knutsson & Mattsson (6) showed that cold applied over the calf muscles for 20 min lowers the amplitudes of the electromyographic as well as the mechanical responses to electrical stimulation of the motor nerve fibres. As the amplitudes are reduced the responses increase in duration, and hence it seems likely that the amplitude reduction should be due, at least in part, to a desynchronization of the responses, probably as a consequence of different effects on the conduction velocity in different nerve fibres. By comparing responses to electrical stimulation of muscle afferents (H reflexes) with responses to tendon taps it could also be shown that cooling facilitates the alpha motoneurons but depresses muscle spindle excitability through inhibitory effects on the gamma motoneurons or through direct effects on the spindles proper. In good agreement with these observations, Urbscheit & Bishop (16), in studies of the effects of cooling on H reflexes and tendon jerks under reinforcement by means of Jendrassik's manoeuvre, found that local cooling applied over the triceps surae muscle inhibits the gamma motoneurons but facilitates the alpha motoneurons. Since they employed fairly brief periods of cold application, no appreciable temperature fall should have occurred in the muscle spindles, but after more prolonged cooling sufficient to lower the intramuscular temperature, the spindles or their nerve terminals are likely to be directly affected (1).

Since local cooling reduces muscle spindle excitability but facilitates alpha motoneurons, the reduction of spasticity following local cooling described by Levine et al. (8) may apply only to cases in which the hypertonus is due mainly to enhanced gamma motoneuron activity. On the other hand, in a muscular hypertonus that is comparatively independent of the inflow from muscle spindles (2), viz. so-called alpha spasticity, the reduction in tone should be very slight, and in such cases the facilitation of the alpha motoneurons may even result in a further exaggeration of the stretch reflexes.

In the following, data will be presented on effects of local cold application achieved in a group of patients with pronounced spasticity; it will be shown that in some subjects the muscular hypertonus was significantly reduced whereas in others it remained constant or increased. These results may reflect differences in the mechanisms governing the muscular hypertonus and may explain why in some cases of spasticity cooling results in beneficial effects on motor functions whereas in other cases no such improvement occurs.

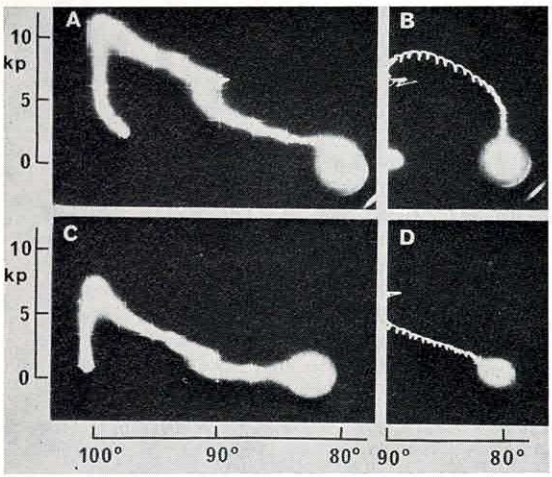

Fig. 1. Resistance to passive knee extension in patient with spastic paraparesis before (*A* and *B*) and after (*C* and *D*) 20 min cold application over the hamstrings. Resistance to manual extension (*ordinates*) recorded by transducer attached to the leg 4 cm above the medial malleolus. Angular displacement from right to left on *abscissae*. *A* and *C*, slow, *B* and *D* rapid extension. Angular displacement of knee recorded as horizontal deflection and concomitant changes in passive resistance as vertical deflection of the oscilloscope beam.

METHODS

The study comprised 30 patients, all with pronounced spasticity in one or more muscle groups, in 14 cases caused by cerebrovascular diseases, in 11 by multiple sclerosis and in 5 by traumatic spinal cord injuries.

Cooling was accomplished by application of chilled packs (ColPaC, −12°C) over the muscle group exhibiting the most pronounced spasticity; the packs were left in place for 15 min over the upper extremities and for 20 min over the lower limbs. The temperature was measured with thermistors subcutaneously as well as at a depth of 2 cm within the muscle. With the type of cooling employed the subcutaneous temperature rapidly drops to about 20°C and then remains fairly constant. The intramuscular temperature is as a rule slowly and gradually reduced by 3–8°C. In some cases no significant temperature fall occurred in the muscle, and in these cases the effects differed from those generally obtained, as will be shown below.

The resistance to passive movements was measured using two different methods. By one of them, the joint was flexed and extended (30°) pneumatically by a constant force corresponding to 80 kp, thus far exceeding the passive resistance to the limb movement. Movements could thus be performed at a constant rate, usually 30° per sec, and independent of possible changes in resistance. The alternative method which was employed when the patient could not stand fixation implied manual extension and flexion of the limb during concomitant recording of the rate and range of the movements. In both these procedures the passive resistance to the movements was recorded with a strain gauge transducer displaying the pressure and tension necessary to perform the test movements. The strain gauge constituted one arm of a chopper amplifier, and an oscilloscope and/or an inkwriter were used to record the signals from this amplifier as well as from an angulometer.

Maximal voluntary contraction power was measured with strain gauge transducers and voluntary range of motion with the angulometer. In cases where the patient lacked ability to perform any movements, the limb was allowed to hang suspended in a strap connected to the strain gauge transducer; in this position the weight was measured and the loss in weight during effort to lift the limb recorded. With this method, forces down to 0.2 kp can be measured with reasonable accuracy. The variability in contraction power was ±20% and in angular range ±5°.

In some experiments the electromyographic activity was recorded in order to be able more accurately to evaluate the voluntary muscle activity and the type and distribution of spasticity as well as the threshold and frequency of clonus. For this purpose surface electrodes were used and the myopotentials were rectified, time-averaged and recorded on the inkwriter as a so-called integrated electromyogram (IEMG).

The displacement patterns of walking were recorded by means of interrupted-light photography. The subjects were supplied with reflecting targets on the lateral surfaces of lower and upper limbs and photographed when walking before a camera in the illumination of a strobe-light flashing 20 times per sec (cf. 12).

RESULTS

Resistance to passive movements

The resistance to passive movements before and after cooling was measured in 15 patients. In 2 cases it remained constant, in 3 cases the resistance to stretch in the chilled muscles increased (10–60%), and in 10 cases it was reduced after cooling. In 3 of these latter cases the improvement was small or varying and hence difficult to evaluate quantitatively, but in 7 cases it was significant (mean 50%, range 35–65%).

These effects are exemplified in Fig. 1, showing the resistance to passive knee extension in a patient with spastic paraparesis and with marked muscular hypertonus in the knee flexors. The records show the resistance to slow extension of the knee joint from 80° to 100° (*A* and *C*) and to rapid extension from 80° to 90° (*B* and *D*). *A* and *B* represent pre-cooling, *C* and *D* post-cooling results. As appears from the figure, the resistance to both slow and rapid extension is distinctly reduced after cooling, viz. from maximum 12 to 7 kp on slow, and from maximum 9 to 5

kp on rapid stretch. These changes can be attributed to a marked reduction of the hypertonus in the knee flexors, but a fairly strong hypertonus persisted after cooling since with the type of stretch employed the normal passive resistance is less than 1 kp.

Clonus

Eight patients had clonus in an upper or lower limb. In 2 of these cases the clonus was abolished when the spastic muscles had been chilled. In 6 cases the threshold, duration and/or frequency of clonus were reduced, in good agreement with previous observations of cooling effects on clonus (3, 11, 13). Most of the patients in whom a foot clonus was thus abolished or reduced reported gait facilitation as a result of the cooling but in most cases it proved to be difficult to find any significant objective changes in walking pattern that might explain these subjective assessments. In some cases, however, the dorsiflexion of the foot at the end of the swing phase was significantly enhanced after cooling. This is exemplified in Fig. 2, showing the walking pattern of a young paraplegic patient with toe-dragging, immediately before (*A*) and after (*B*) cooling applied to the calf muscles. As seen, cooling resulted in a marked improvement of the toe elevation late in swing, which provided better foot–floor clearance. The result thus obtained resembles the gait facilitation by plantar punctate pressure stimulation reported by Mårtensson, Knutsson & Hindmarsh (9) even though the mode of action is quite different in the 2 cases.

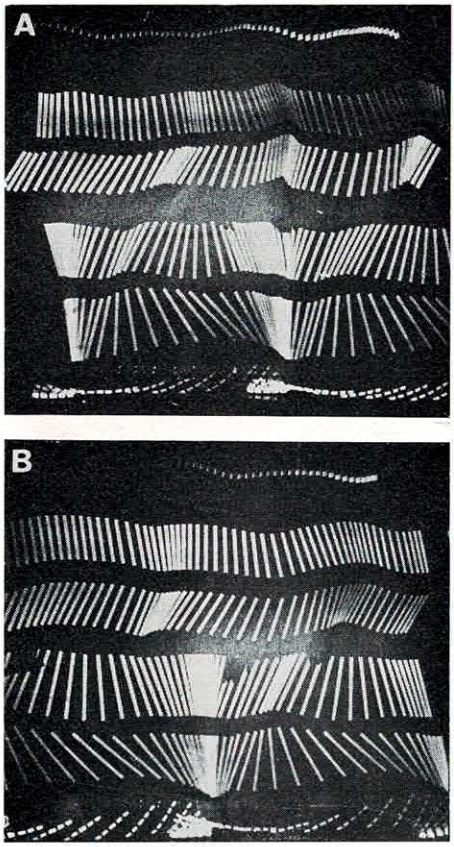

Fig. 2. Intermittent-light photographs of walking pattern in patient with spastic paraparesis before (*A*) and after (*B*) 20 min cold application over the triceps surae muscles. Reflecting targets from top to bottom located on head, upper arm, lower arm, thigh, leg and lateral lower surface of shoe. Strobe-light frequency 20/sec. Patient walking at free speed.

Voluntary range of motion

Twelve patients had restricted voluntary range of motion. In 8 of these cases the range increased 15–70° (mean 35°) after cooling. In 2 patients willed effort to extend the knee joint resulted in an unintentional flexion which was abolished by cooling. The 2 remaining cases had no power whatever of voluntary limb movement either before or after cooling.

Maximal voluntary contraction force

In the muscle groups exhibiting the most pronounced spasticity, the strength of isometric maximal voluntary contraction (MVC) varied between 0.2 and 18 kp (mean 7.6 ± 5.1, $n = 27$). The mean value after cooling was 8.2 ± 5.2 kp and the mean relative increase 11 %; this change is insignificant.

In the antagonists of the chilled muscles, MVC varied between 0.2 and 16 kp (mean 7.3 ± 5.3, $n = 29$). After cooling, the mean value had increased to 9.9 ± 5.3 kp. The mean relative increase was 98 % and the improvement highly significant ($P < 0.01$). The effects obtained varied however considerably among the different patients. Thus, no improvement occurred in 11 cases and a reduction by about 50 % was observed in one case, but in 11 patients the maximal voluntary contraction force showed an increase exceeding 50 %. The remaining 7 cases displayed a moderate increase, the significance of which was uncertain.

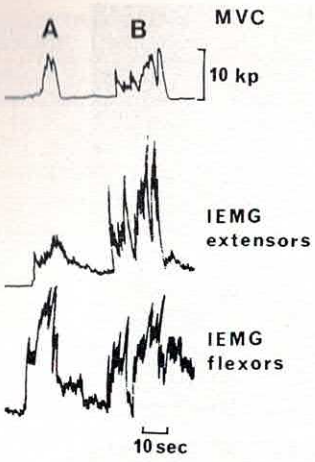

Fig. 3. Force and integrated electromyographic activity in extensor and flexor muscles of a patient with spastic paraparesis during maximal effort to flex (*A*) and extend (*B*) knee. Force recorded isometrically at 90° between thigh and leg, with transducer at ankle. Electromyographic activity recorded with pairs of surface electrodes 7 cm apart and 10 cm proximal to the patella over the quadriceps and hamstrings respectively.

In the patients in whom the resistance to passive movements was not reduced or increased after cooling, the MVC in the antagonists of the chilled muscles remained constant (4 cases) or was considerably lowered (1 case).

All patients in whom the passive resistance was reduced after cooling also displayed a marked increase in strength during effort to contract the antagonists of the chilled muscle group. This improvement should hardly have been due to a change in contraction strength proper but should rather be attributable to the reduction in spastic resistance in the chilled muscles. Fig. 3 shows a typical example of the activity pattern on extension and flexion of the knee in a patient with pronounced muscular hypertonus in the knee flexors. The upper curve shows the strength on flexion (A) and extension (B), the lower curves the IEMG from extensors and flexors. As appears from the figure, the electromyographic activity in the flexors is nearly as large during effort to extend as during effort to flex the knee joint. Thus, extension is strongly counteracted by the active flexors. When the flexors had been chilled the unintentional activation of these muscles during effort to extend was markedly reduced and at the same time the effective extension force increased to 22 kp as against 9 kp before cooling. Primarily, the improvement of the maximal voluntary contraction strength observed thus seems to have been the result of a reduction of the muscular hypertonus in spastic antagonists.

In four other patients no significant temperature fall in the muscles was achieved during the cooling. In one case, preganglionic sympathectomy had been performed. In another patient a partial injury of the peripheral nerves supplying the limb was verified in spite of highly exaggerated stretch reflexes, thus indicating preserved coarse nerve fibres. In still another of these patients a causalgic reaction to cold (cf. 14) was observed. These 3 patients may consequently have had vasomotor disturbances preventing a normal reaction to cold. In the fourth case no explanation could be found for the abnormal temperature reaction.

DISCUSSION

From the results reported above it is evident that application of local cold over spastic muscles may reduce their tonus and thus enhance the effect of the contraction in the antagonists (cf. 3, 8). For obvious reasons, no such effects on the contraction strength can be anticipated in cases of lacking voluntary activity even though the hypertonus may be markedly reduced. Nor does any significant increase in strength seem to occur in cases where cooling does not induce an effective topical temperature fall in the muscles, and hence no relief of spasticity may be obtained in cases of abnormal vasomotor reactions preventing a local temperature fall.

In those patients in whom the resistance to passive stretch of the chilled muscles increased in spite of a significant local temperature fall, the muscular hypertonus might have been due to an increased alpha motoneuron activity not caused by an excessive excitatory inflow from the spindles (2, 15) since the temperature fall did not reduce the stretch reflexes (cf. 1). The increase in passive resistance observed in these cases might then be accounted for by the facilitatory effect of cooling on the alpha motoneurons (6, 16).

According to clinical observations (4, 8) and objective measurements (5, 7), repeated sessions of therapeutic exercise during cold-induced relaxation of spasticity often provide striking long-term improvements of motor functions. Thus, criteria

should be established by which the categories of patients liable to respond favorably to cold therapy could be singled out at an early stage. As contraindications, cold allergy and Raynaud's disease have previously been mentioned (10); to these should be added sympathectomy and causalgia, in which conditions cooling may cause discomfort to the patients. In cases of absent or very restricted voluntary activity no functionally significant increase in strength can be expected, and in alpha spasticity the effect is likely to be insignificant or even negative. Since it is difficult in practice to distinguish between an alpha and a gamma spasticity present and to assess the voluntary activity which may be concealed by the spasticity, tests of the immediate effects produced by cooling may serve a useful purpose in the evaluation of possible long-term effects which are often fairly similar to those obtained in the acute tests (5).

ACKNOWLEDGEMENTS

This work was supported by grants from the Swedish Research Council, Olle Blomqvist Rehab-Produkter AB and the Folksam Insurance Company, Stockholm.

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Key words: Spastic paresis, reflex, motor activity, temperature.

Address for reprints:
E. Knutsson, M.D.
Department of Clinical Physiology
Karolinska Sjukhuset
S-104 01 Stockholm, Sweden