NERVE COMPRESSION AS AN ESSENTIAL FACTOR IN CAUSING ISCHAEMIC AND POST-ISCHAEMIC PARAESTHESIAE

EROS ABRANTES ERHART *

While studying the disturbances of sensation occasioned by experimental ischaemia [1] our attention was drawn to the well established fact that the ischaemia produced by compression on the proximal part of the forearm and on the wrist, did not cause sensory disturbances, identical to those observed in compressions of the upper arm.

Lewis and associates [3, 4] considered the ischaemic nerve as the field of origin of the paraesthesiae, and ascribe the differences observed in compressions at various levels, to the greater vulnerability of the long fibers.

Bazett and McGlone [2] ascribe release pricking to a muscular field because this paraesthesia is much weaker when blockage of circulation is at the lower end of the forearm. These authors believe that "pins and needles" are caused by chemical agents of metabolic origin, so that exclusion of the highly metabolic muscle masses from the ischaemic territory would account for the lower intensity of the paraesthesiae.

Weddell and Sinclair [7] consider the ischaemic receptor field as the field of origin of the paraesthesiae. They explain that the intensity of the paraesthesiae is weaker when the blockage of blood flow is exercised below the level of the elbow or at the wrist, as compared to the compression in the upper arm, argueing that the cuff pressure on the distal regions, even when much higher than the systolic pressure, is not sufficient to block the circulation as well as at the upper arm, since there the vessels would be better protected against pressure.

We wonder whether or not that would apply also to the nerves.

EXPERIMENTS

The following experiments were made in order to determine whether or not compression of the nerve itself modifies the paraesthesic disturbances.

The method used was fundamentally that of Lewis and Pochin [4], and several experiments were made on ourselves and others.

Exp. I — The blockage of circulation was performed by putting the cuff of a sphygmomanometer on the proximal part of the forearm and the elbow joint, and bringing the pressure rapidly up to 200 mm/Hg while the limb was elevated. Thereafter the limb was put in the most comfortable position for the subject. About 10 minutes after the arrest of circulation a very light tingling sensation was felt

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Trabalho realizado no Departamento de Anatomia da Fac. Med. da Univ. de São Paulo (Prof. R. Locchi).

* Assistente do Departamento de Anatomia da Fac. Med. da Univ. de S. Paulo.

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beginning in the fingertips; the hand was cold and successive thermic waves were referred by the subject. After 13 minutes a numbness sensation was felt beginning also in the fingertips. At this stage it was still possible for the subject to recognize numbers drawn with a pencil on his fingertips. At the 17th minute anesthesia began in the fingertips and gradually spread to the whole hand, specially on the palmar surface. At about 19.30 minutes intense pain was felt in the hand. The cuff pressure was released after 20.30 minutes of compression. Immediately after, the subject referred heat, swelling sensation, disappearance of pain, of numbness, and strong hyperhaemia was observed. After 2.30 minutes a light tingling appeared, but was soon masked by buzzing. Pins and needles were very weak; they were reforced by tapping and diminished momentarily by firm pressure. Such sensations gradually diminished and finally disappeared about 14.30 minutes after the release of compression.

Exp. II — Conditions of this experiment were as in Exp. I, but the cuff was placed more distally, that is, without envolving the elbow. Identical sensations were observed concerning the appearance, character, intensity and duration of the phenomena.

Exp. III — The cuff was applied to the forearm as in Exp. II, and a small cushion was placed under it so as to compress the field of the median nerve trunk; the pressure was 200 mm/Hg. About 2 minutes after the arrest of circulation, a light tingling sensation was felt in the cutaneous territory of the median nerve including the thenar eminence, but chiefly in the tips of the first, second and third fingers. In the ulnar territory there was only a cold sensation. At the 10th minute a decrease of sensibility was referred by the subject, only in the field of the median nerve. After 14 minutes of compression that field became numb while the ulnar territory was practically not affected. At the 17th minute the field of the median nerve, was anesthetised. The ulnar territory showed only slight sensory disturbances. The cuff was released at the 20th minute. One minute after, buzzing, and pins and needles appeared; the latter of greater intensity in the field of the median. Pins and needles were reinforced by tapping and diminished by local pressure. A most interesting fact was observed in the fourth finger, that is, the difference in intensity of the paraesthesiae, was perfectly noticeable, being much stronger on the lateral side (median nerve) than on the medial side (ulnar nerve). About 10 minutes after release, the ulnar territory was practically normal; this only occured with reference to the field of the median at the 45th minute, that is, 25 minutes after the release of the cuff.

Exp. IV — The conditions of the experiment were as in Exp. III, but the cushion was placed so as to compress the ulnar nerve. Identical phenomena as those of Exp. III were observed, but now with predominance in the ulnar territory.

Exp. V — Compression of 200 mm/Hg with a cushion over the median nerve, at the level of the wrist, determined identical phenomena as those observed in Exp. III concerning to the appearance, character, intensity and duration of the paraesthesiae.

Exp. VI — Based on Exp. III, the compression was applied on the tibial nerve at the popliteal region. After 3 minutes, tingling started in the medial part of the sole of the foot and hallux. At about the 5th minute tingling had already spread to the whole foot, specially fingertips. At the 12th minute it was very strong in the whole cutaneous field of the tibial nerve. Tingling was so intense that it ressembled pins and needles. After about 15 minutes the numbness started followed by diffuse pain. The foot was entirely cyanotic. The pain was practically unbearable. Compression was discontinued after 20 minutes. The typical hyperhaemia, comparable to the one that usually occurs in the upper limb was not observed; here it was very slight. The subjects noticed that the heat sensation following release of compression was lesser intense, as compared to the upper limb. The fingernails began to loose the cyanotic aspect at the 21st minute, when buzzing appeared, followed soon by pins and needles. Tapping determined a very distinct spread of pins and needles to the medial malleolus and neighborhood. At the 27th minute, that is, 7 minutes after release of compression, the limb returned to its normal condition.

Exp. VII — The sciatic nerve can be compressed when the subject takes an adequate seating position. During 20 or 25 minutes of compression, sensory disturbances (tingling, numbness and anesthesia) follow each other in the whole foot and distal part of the leg. Release of pressure on the nerve results in buzzing, and pins and needles. It should be recalled that the release pricking is so strong that the patient is quite unable to stand up without support.

DISCUSSION

It is a common experience to awake with a limb, or part of it, anesthetised on account of a long compression of one or more nerve trunks. However, in these circumstances release pricking is not always felt after compression is interrupted, perhaps because the compressed portion of the nerve did not exceed 2 cm.[5], or because the compression was not intense enough. We wonder if anesthesia might be the result of long summation effect of a slight compression, in spite of its small extension.

These hypotheses and other unknown conditions might explain the findings of Weddel and Sinclair who compressed a nerve in the *sulcus bicipitalis*, without interfering with the circulation, obtaining tingling and anesthesia but not a characteristic release pricking. Sinclair [6] points out furthermore, that in this case anesthesia does not spread gradually as in ischaemic experiments, but shows some discontinuity from one nerve field to another.

All our experiments show that besides ischaemia, the nerve compression increases the paraesthesic effects of the release pricking in a very distinct way. In this respect our results differ somewhat from those of other investigators, including those recently published by Merrington and Nathan.

Concerning the field of origin of the paraesthesia, our results seem to corroborate Lewis' hypothesis that the ischaemic and post-ischaemic conditions of the nerve trunk are responsible for such paraesthesiae. Our experiments show, however, that compression of the nerve itself added to ischaemic condition, increases the paraesthesiae, in the cutaneous field of the compressed nerve.

Regarding the mechanism of the paraesthesiae, we must admit with Weddell that the several mechanic conditions on the different levels of the limbs have great importance in determining different paraesthesiae, caused by proximal or distal blocking. However, we observed, that it is not the mechanic action on the blood vessels as Weddell admits, but the mechanic effect on the nerve trunk itself that determines the typical ischaemic and post-ischaemic paraesthesiae, as shown by our results.

SUMMARY

In order to explain why ischaemia produced by compression at the proximal part of the forearm or at the wrist, does not cause sensory disturbances identical to those observed in compression at the upper arm, several experiments were made on ourselves and other subjects.

The method used was essentially that of Lewis and Pochin. Circulation was blocked at different levels of the limb by applying the pressure of 200 mm/Hg with a sphygmomanometer cuff; compression of the median, ulnar and tibial nerve trunks was made with the aid of a small cushion under the cuff.

We observed always that the nerve compression is an essential factor in causing ischaemic and post-ischaemic paraesthesiae.

SUMÁRIO

A compressão do nervo como fator essencial no desencadeamento das parestesias isquêmicas e pós-isquêmicas

Estudando as alterações da sensibilidade conseqüentes à isquemia experimental, foi notado, mais uma vez, o fato já descrito de que as isquemias produzidas por compressão, ao nível da porção proximal do antebraço e do punho, não ocasionam perturbações da sensibilidade, idênticas às verificadas em compressões do têrço superior do braço.

Com o intuito de estudar tal problema, efetuamos uma série de auto-experiências e as repetimos em outros. A técnica empregada foi fundamentalmente idêntica à de Lewis e Pochin; a circulação foi bloqueada em diferentes níveis dos membros, usando-se para isso o manguito de um tonômetro, a uma pressão de 200 mm/Hg. Sob o manguito, no entanto, era colocado um coxim de modo a comprimir quer o nervo mediano ou o ulnar, logo abaixo do cotovelo ou ao nível do punho, ou, então, o nervo tibial, no cavo poplíteo.

Contornando, assim, o problema da dificuldade de compressão adequada dos nervos por causa de suas situações diversas nos segmentos proximais e distais dos membros, foram observadas em tôdas as nossas experiências perturbações de sensibilidade, limitadas ao território do nervo comprimido e idênticas às verificadas em compressões efetuadas na porção proximal do braço.

Verificamos, portanto, que a compressão adequada do nervo é um fator essencial para o desencadeamento das parestesias isquêmicas e pós-isquêmicas.

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Departamento de Anatomia da Fac. Med. da Univ. de São Paulo. Caixa Postal 100 B — São Paulo, Brasil.