

Intra-operative telethermographic scanning in the compressive syndrome of the wrist median nerve

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SUMMARY. The Authors report about the etiology and pathogenesis of the carpal tunnel syndrome, on the basis of the experience acquired by the use of intra-operative teletermography. They point out the importance of the vascular factor when defining this syndrome and distinguish 3 phases, among which the first 2 are functional: 1st phase: irritative (venous stasis); 2nd phase: altered metabolism (from diminished arterial supply); 3rd phase: organic.

Thus, from this pathogenetic considerations, there is no standard surgical intervention for the treatment of the carpal tunnel syndrome; each operation should be different and proportional to the damage.

Key words: intra-operative thermography, thermography, carpal tunnel syndrome, micro-surgery.

INTRODUCTION

For a better understanding of the pathogenetic events which are responsible for the syndrome of the carpal tunnel, it is important to consider:

1) the median nerve runs within an unextensible osteofibrous tunnel whose dorsal wall, being flat, does not allow any elastic adaptation of the nerve; this adaptation, on the contrary, is fairly possible in the interstices of the flexor tendons;

2) at the tunnel level, the sensitive fascicles for the thumb and the 3rd common digital nerve, as well as the motor fascicles, are located at the surface¹⁰, and so more directly exposed to the effects of compression; on the contrary, the sensory fascicles for the 1st and 2nd common digital nerves, being deeper, have more possibility of escaping these effects (thumb sensitivity is always more affected than other finger sensitivity);

2) there are three different types of vascularization⁸ of the wrist median nerve: a) from the radial and ulnar arteries (70% of cases); b) from the persistence of the median artery that, at the palm, divides into the digital branches (in this case the superficial palmar arch

is missing: 10% of cases); c) from the median artery anastomoses with the superficial palmar arch (20% of cases).

The vascularization of the wrist median nerve is almost fully of a volar size; the vessels, as well as the fascicles, are therefore particularly exposed to any compressive event. Moreover, in the median nerve, as in the other peripheral nerves, the vascularization is functionally segmental²; a localized metabolic disease of the nerve may therefore occur when there is a segmental trouble of the vascular supply.

At the moment, there are two interpretations about the pathogenesis of the compressive syndrome: a) the vascular factor could cause the local lesion of the nerve; b) the pressure itself could cause the invagination of the paranodal myelin⁷ or the segmental demyelination. Landi and Coll.^{1,6} were able to prove that, actually, in such compressive syndrome, the vascular factor is the first, appearing a really compressive factor during the regeneration of the nerve fibres; so it would be more exact to define that as an ischemic-compressive syndrome.

The purpose of this paper is to evaluate the role of the vascular factor in the compression syndrome determination.

MATERIALS AND METHOD

The investigation has been carried out by means of an AGA THERMOVISION 680 with a 15° x 15° lens and an extension ring to get the maximum magnification; an area of 4.6 x 4.6 cm was explored.

The thermographic investigations have been carried out after surgical exposition of the me-

dian nerve, at standard intervals of 10, 20, 30 minutes after removing the tourniquet.

The investigation has been performed on 10 patients where the carpal tunnel syndrome had been diagnosed both on a clinical and a neurophysiological basis, and 2 control patients with Dupuytren disease (Fig. 1A). In the last cases the intra-operative thermographic investigation, according with the anatomic macroscop-

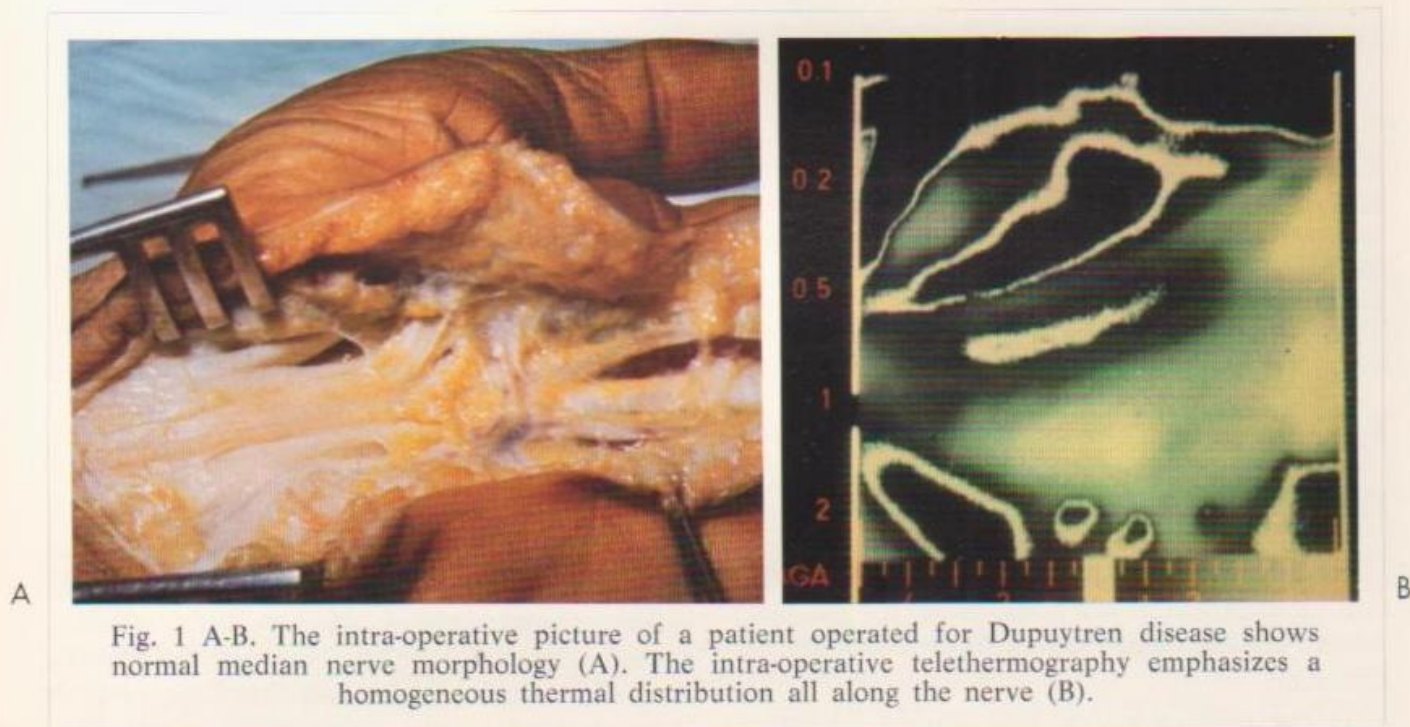


Fig. 1 A-B. The intra-operative picture of a patient operated for Dupuytren disease shows normal median nerve morphology (A). The intra-operative telethermography emphasizes a homogeneous thermal distribution all along the nerve (B).

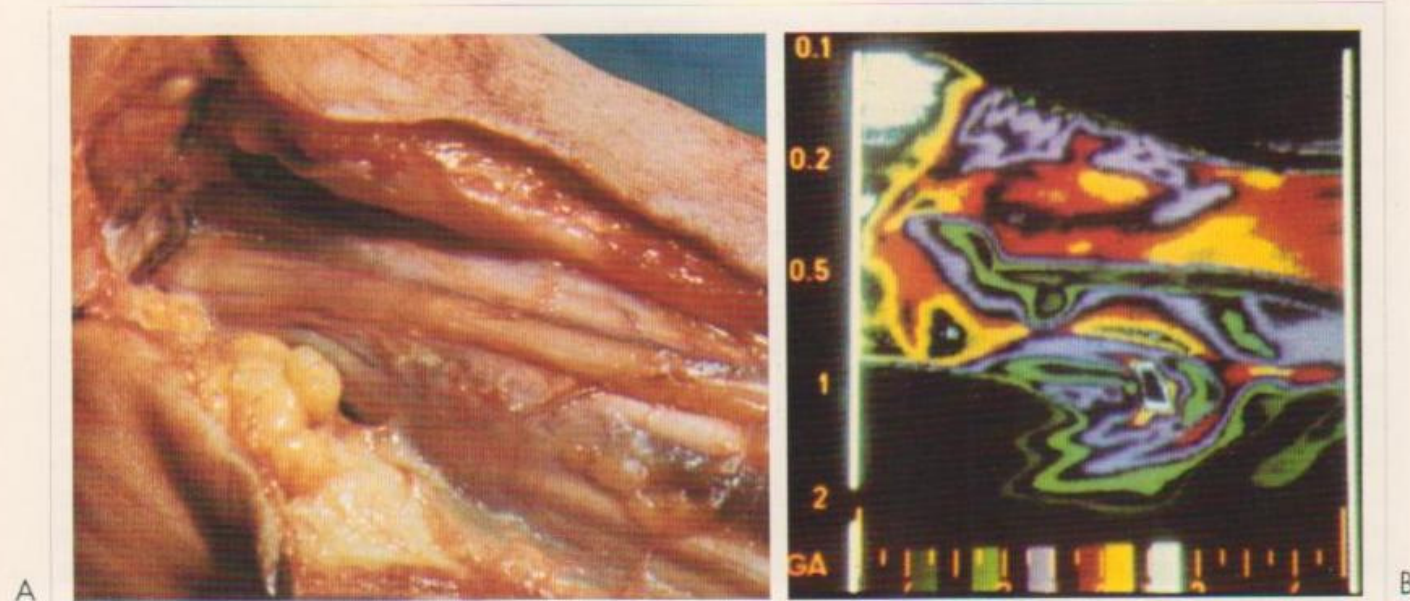


Fig. 2 A-B. Carpal tunnel syndrome in its irritative phase. Normal median nerve morphology (A), and homogeneous thermal distribution (B).

pic report, pointed out a homogeneous thermal distribution all along the section of the median nerve, between the palmar cutaneous branch to the forearm and the terminal ramifications to the palm (Fig. 1B).

Since 6 out of the 10 cases are practically identical as to extent of the anatomic, pathologic, neurophysiologic and telethermographic damage, and correspond to the most frequent type of lesion, showing a proximal bulb-like swelling of the nerve, we shall here detail only 4 cases, showing different stages of acuity.

1st case. Symptomatology featured night pain disappearing for 5 to 6 days only under ACTH treatment. At the physical examination, the only positive sign was the Gilliat's tourniquet which presented a particular susceptibility of sensitive fibres to ischemia. The electromiographic examination was negative except for a poor fibrillation of the thumb opponent.

The anatomo-pathologic appearance of the nerve was normal (Fig. 2A). The thermographic distribution was homogeneous (Fig. 2B); vascularization was the «C» type. The operation was limited to an exploratory epineurotomy.

A 6 months follow-up evidenced the complete disappearance of subjective phenomena and confirmed the normality of the electromiographic results.

2nd case: the patient had a history of 4 months duration with subjective symptoms such as night pain and paraesthesia at the 2nd and 3rd fingers; there was no atrophy at the physical examination. Superficial tactile hypoaesthesia was noticeable at the 2nd and 3rd fingers. The neurophysiologic investigation already showed a lengthening of the terminal latency whereas the S.A.P. of the median nerve was absent. Under operation the nerve was normal (Fig. 3A) and vascularization was of the «A» type. The telethermographic investigation showed a warmer area at the palm and a colder one at the wrist (Fig. 3B). An exploratory epineurotomy of the median nerve did not show any thickening of the connective component.

The clinical follow-up after 2 months showed the disappearance of hypoaesthesia at the 2nd and 3rd fingers. The instrumental investigation showed a normalization of the terminal latency (2.9 msec) and the new appearance of the S.A.P. of 30 μ v magnitude and normal latency.

3rd case: the symptomatology dates back to 18 months with pain at the cervical spine spreading to the arm, and paraesthesia at the hand. Physical examination revealed considerable tenar hypotrophy and hypoaesthesia of the first three fingers. Fibrillation was abun-

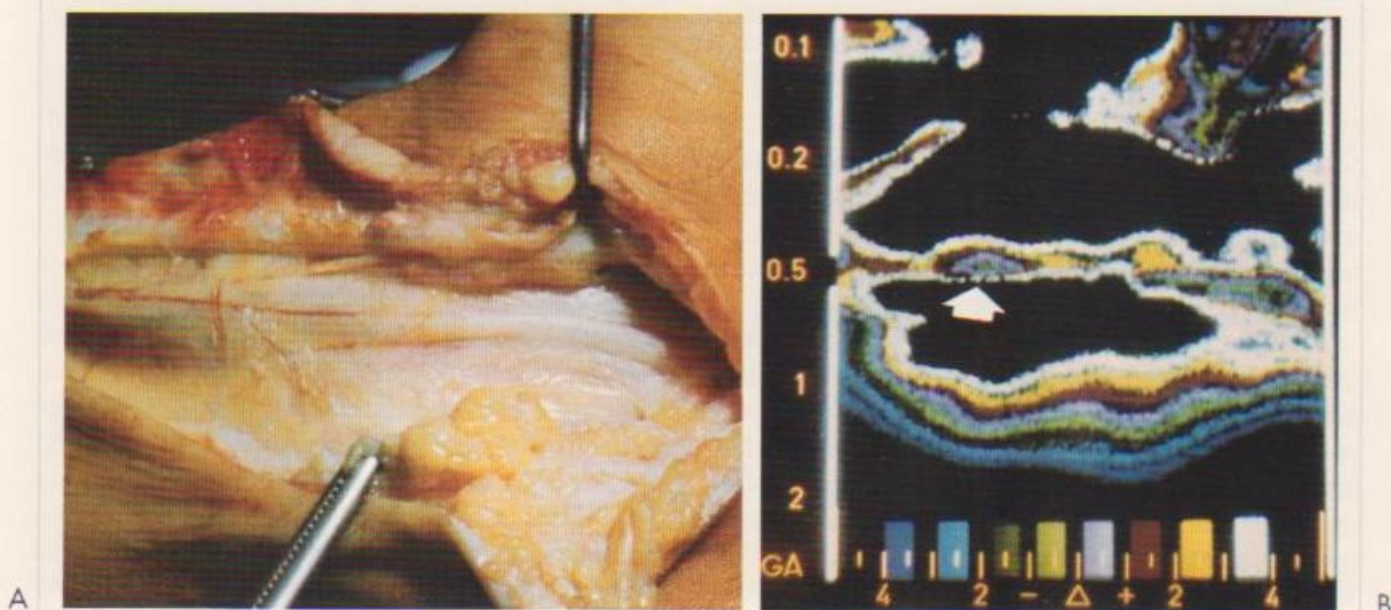


Fig. 3 A-B. Carpal tunnel syndrome operated in the metabolic phase. The intra-operative examination shows (A) a normal structure, while thermography (B) emphasizes a clear hypothermic area (arrow) in the nerve section under the cross ligament.

dant in the opponent. Terminal latency was 4.2 msec, the S.A.P. was absent. The anatomic-pathologic picture showed the typical bulb-like swelling of the nerve (Fig. 4A) and a severe tenovaginalytic lesion; vascularization was of the « A » type. Telethermography showed hyperthermia of the bulb, while the section of nerve under the cross ligament appeared

graphic picture clearly showed an area of hypothermia corresponding to the stenosis of the nerve (Fig. 5B). A lateral epineurotomy without internal neurolysis and a tenovaginallectomy were carried out.

An examination performed 9 months later did not show any motor recovery, while a considerable hypoaesthesia was still present, espe-

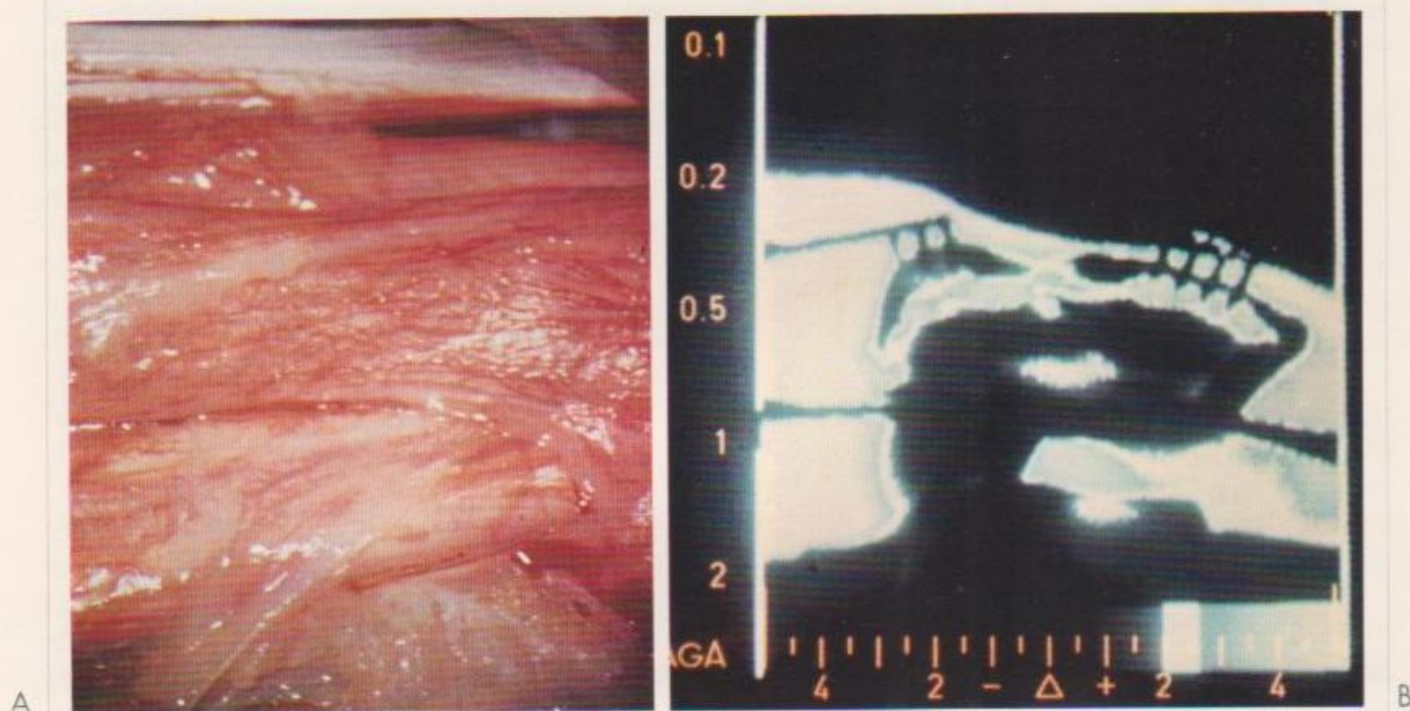


Fig. 4 A-B. Carpal tunnel syndrome in an advanced metabolic phase. The intra-operative microscope picture shows (A) a bulb-like swelling of the nerve, on the surface of which one can see a thick vascular net with a restrictive circulation. Thermography (B) shows hyperthermic bulb and hypothermia of the section under the cross ligament.

cold (Fig. 4B). After epineurotomy, the connective tissue thickening made a very limited internal neurolysis advisable.

9 months after the operation an examination showed excellent motor recovery and still a moderate hypoaesthesia, especially at the 1st finger; the instrumental parameters had improved.

4th case: symptoms were present from 26 months. There were considerable hypotrophy and hypoaesthesia. There was no sign of denervation. Terminal latency was 4.2 msec, the S.A.P. was absent. The anatomic-pathologic picture showed, as in the previous case, a bulb-like swelling (Fig. 5A) associated with a tenovaginalytic lesion of the flexors. Vascularization was of the « A » type. The thermo-

graphically at the 1st finger. Terminal latency extended to 7.5 msec.

DISCUSSION

The comparison between the various parameters defining the syndrome of the carpal tunnel, before and after operation, and especially the intra-operative telethermography, permit us to make 3 statements:

1) There are different factors in the pathogenesis of this syndrome: a) there is a first stage, of an irritative nature, emphasized by the acroparaesthesia and the night pain. This stage can be diagnosed only by means of those tests evidencing the particular sensitiveness to metabolic acidosis^{3,4}. The compression, the-

refore, at its first stage, acted on the venous back-flow system, causing phenomena of local acidosis resulting in the high irritability of sensory fibres (pain and paraesthesia) and of motor fibres. In these cases, both the clinical and the usual instrumental investigations are mute; the anatomic-pathologic appearance of the nerve is normal (Fig. 2A) and the intra-operative thermography shows an homogeneous thermal distribution in the nerve (Fig. 2B). b) there is a second stage of local diminished blood flow; at this stage the metabolic diseases

In this case the fibres, being affected by neuro-apraxia, will soon recover when the circulation becomes normal again after removing the compression.

In the altered metabolism phase, the nervous collagen already shows alterations at the histoenzymatic level⁶ which will lead to the fibrosis which is characteristic of the organic stage. In the 3rd case, in fact, the changes are no longer functional (stasis - ischemia) but having become organic over time, they affected the collagen load-bearing structure, with a

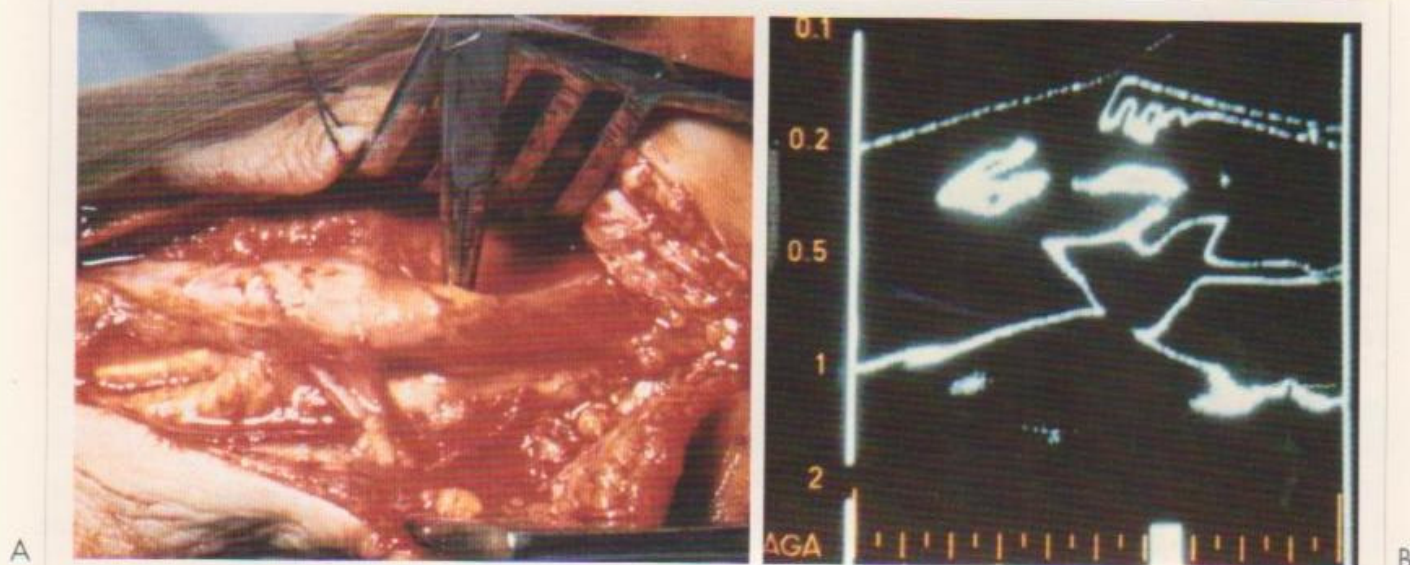


Fig. 5 A-B. Carpal tunnel syndrome, severe organic phase. Clear bulb-like swelling of the nerve and perfect correspondence between anatomic (A) and thermal (B) stenosis.

dependent on the poor circulation, mainly show themselves in the nervous fibre. The metabolism of the fibre⁵ is actually affected by the exogenous glucose, carried by the blood, or the glycogen stored by the fibre itself. The ATP produced by the oxidative phosphorylation is used both to maintain the fast axoplasmic transport and for the sodium pump that requires by itself half or more of this amount. Moreover, in Schwann's cells, the degree of myelination is governed by factors carried from the cell into the nervous fibre. A local metabolic disease, dependent on diminished blood flow, explains both the neuroapraxic lesion of some fibres, and the segmentary demyelination. In this phase, the intra-operative examination (Fig. 3A) shows a normal structure, while the telethermographic investigation emphasizes a clear hypothermic area (Fig. 3B).

consequent fibrosis. The anatomic pathologic examination (Fig. 4A) shows a typical bulb-like swelling and tenovaginal lesion, while telethermography emphasizes (Fig. 4B) hyperthermia of the bulb and hypothermia of the section under the cross ligament. The operation was a very limited internal neurolysis after epineurotomy. c) in the third stage the damage is exclusively organic: the nervous fibres, packed full by the fibrotic collagen gradually pass from the phase of axon stenosis to the one of axon cachexia. It is at this stage that the persistence of a compression lays the basis for an endofascicular internal compression putting the axons, in their regenerating phase, in the condition of looking for alternative ways of development (bulb-like deformation of the proximal section). In the 4th case the anatomic-pathologic examination showed a bulb-like

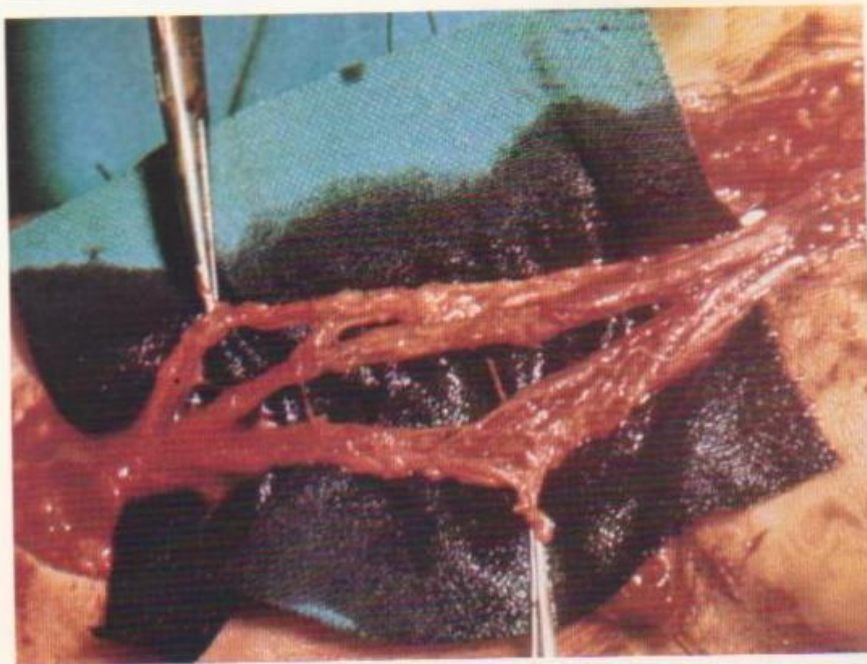


Fig. 6. The intra-operative picture of a median nerve operated for internal neurolysis. Even a serious lesion does not justify such an extended treatment because, besides the anatomic damage of the fibres there is an added surgical damage.

swelling and severe tenovaginitic lesion (Fig. 5A); thermographic picture showed an hypothermic area corresponding to the stenosis of the nerve. The operation consisted in a lateral epineurotomy without internal neurolysis and a tenovaginallectomy. In this case, when the damage is already present in the various components of the nerve, the intervention was not sufficient because it was not possible to remove the endoneural fibrosis between the various fascicles, and the patient got worse both from a clinical and an instrumental point of view.

2) Therefore there is no a single clinical form of the syndrome of the carpal tunnel, but just aggravating phases within the same pathogenetic mechanism.

3) The surgical treatment should consequently be proportioned to the damage. During the irritative phase, opening the tunnel is sufficient, if local steroidal therapy has failed. In all other cases, the lateral explorative epineurotomy, with regard for the vascularization, will permit the surgeon to decide, with intra-operative thermographic aid, whether or not a limited internal neurolysis is necessary.

On the basis of what discussed above, the anatomic dissection of the fascicles (Fig. 6) is a surgical intervention of a purposeless nature.

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