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EDITORIALS

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Neuropathies in Hansen's Disease

One of the basic features of *Mycobacterium leprae* is its neurotropism. As a consequence of this, the structures of the peripheral nervous system are one of the main targets of this disease[1, 2, 3, 4]. The thickening of the peripheral nerves in certain anatomic areas and the recognition of some of the most evident signs (anesthetic patches, deformities such as claw hand or "main en griffe," lagophthalmos) have been for many years the only known characteristics of the pathology generically designated as "leprous neuritis."

The discovery of sulfones and the consequent possibility of neutralizing the epidemiological risk through chemotherapy put medical science up against the need for returning to society those patients who, until that time, had been isolated in leprosaria[5].

Treatment and rehabilitation of the disabilities, considered part of the natural history of leprosy in the past and now identified as complications arising from the involvement of peripheral nerves[6, 7], has in the last few years included the surgical treatment of damaged nerves in a search for more appropriate solutions. The contradictory results of the first surgical efforts in this regard[3, 8, 9, 10, 11, 12, 13] showed the need to be very selective in the cases to be submitted

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for surgery and the technique to be used. Furthermore, it has been shown that so-called "leprous neuritis" is by no means a single nosological entity with individual pathological characteristics. The identification of different pathogenic mechanisms whose common final result is damage to peripheral nerves in Hansen's disease, and where inflammation is one, but not the only, causal factor involved, leads us to postulate abandoning the traditional generic designation "neuritis" as incorrect. We prefer the less exclusive term "neuropathy."

In our opinion, many of the discussions involving the neuropathy of Hansen's disease are due to the lack of a common unequivocal language as well as the lack of standardized guidelines for the evaluation of the therapeutic results. In this paper we have attempted to avoid these difficulties by defining with clarity the terms we use and presenting a classification of the disorders which we have used for several years with good results[14]. Paraphrasing an old aphorism which still holds true, "All leprosy is neuropathy." Therefore, the peripheral neurological study of Hansen's disease patients as well as treatment and prevention of nerve damage are the primary objectives of contemporary leprology.

PATHOGENESIS

Every clinical form of leprosy involves, to a greater or lesser extent, the peripheral nerves. Nevertheless, curiously, and contrary to what a first impression would lead us to assume, nerve destruction is more rapid and more severe in patients having better immune defenses (tuberculoid leprosy) than in patients whose immune status is more depressed (lepromatous leprosy). This occurs because the bacillary infiltration of the nerves is accompanied in the tuberculoid patient with an intense inflammatory reaction unleashed by the immunologic conflict within the nerve tissue. Consequently, edema is produced as well as vascular lesions, ischemia, necrosis, and eventual caseation of the lesions of the parenchyma (neural abscess)[5, 8, 15].

From the point of view of evolution, it is important to bear in mind that peripheral neural damage and its side effects frequently follow a course which is independent from the systemic manifestations of the basic disease. This means that in "leprologically inactive" patients a progressive exacerbation of their neuropathies may occur. Furthermore, in cases such as those with reversal reactions showing clinical improvement as well as bacteriological and histological improvement, the inflammatory neuritis may become exacerbated. From the practical point of view, this implies that "dermatoleprological discharge" does not necessarily coincide with "neurological quiescence" of the patient. Therefore, periodic checkups and neural reevaluation and rehabilitation should be continued for prolonged periods of time in leprosy patients.

The basis of the lesion in peripheral nerves is the bacillus' neurotropism which causes its localization in nerve fibers of different diameters, ranging from the thin, subcutaneous branches through the main nerve trunks of the upper and lower limbs, to areas of plexus formation, such as the branches of the cervical plexus. These single or multiple mononeuropathies ("oligoneuropathies") do not constitute a universal or systemic pathology, however. Therefore, in spite of involving multiple nerve trunks in some cases, it is incorrect to use the term "polyneuritis"[16, 17].

The presence of the bacillus and certain epiphenomena of the disease, together with local conditions in certain anatomical areas of the peripheral nerves[4, 18], unleash a variety of different lesional mechanisms, involving inflammation, ischemia, mechanical compression, etc.

The pathophysiological mechanisms producing deterioration of nerves in Hansen's disease patients can be reduced to three: 1) intrafascicular lesions, 2) intraneural-extrafascicular lesions, and 3) extraneural lesions. In our experience this pathogenic

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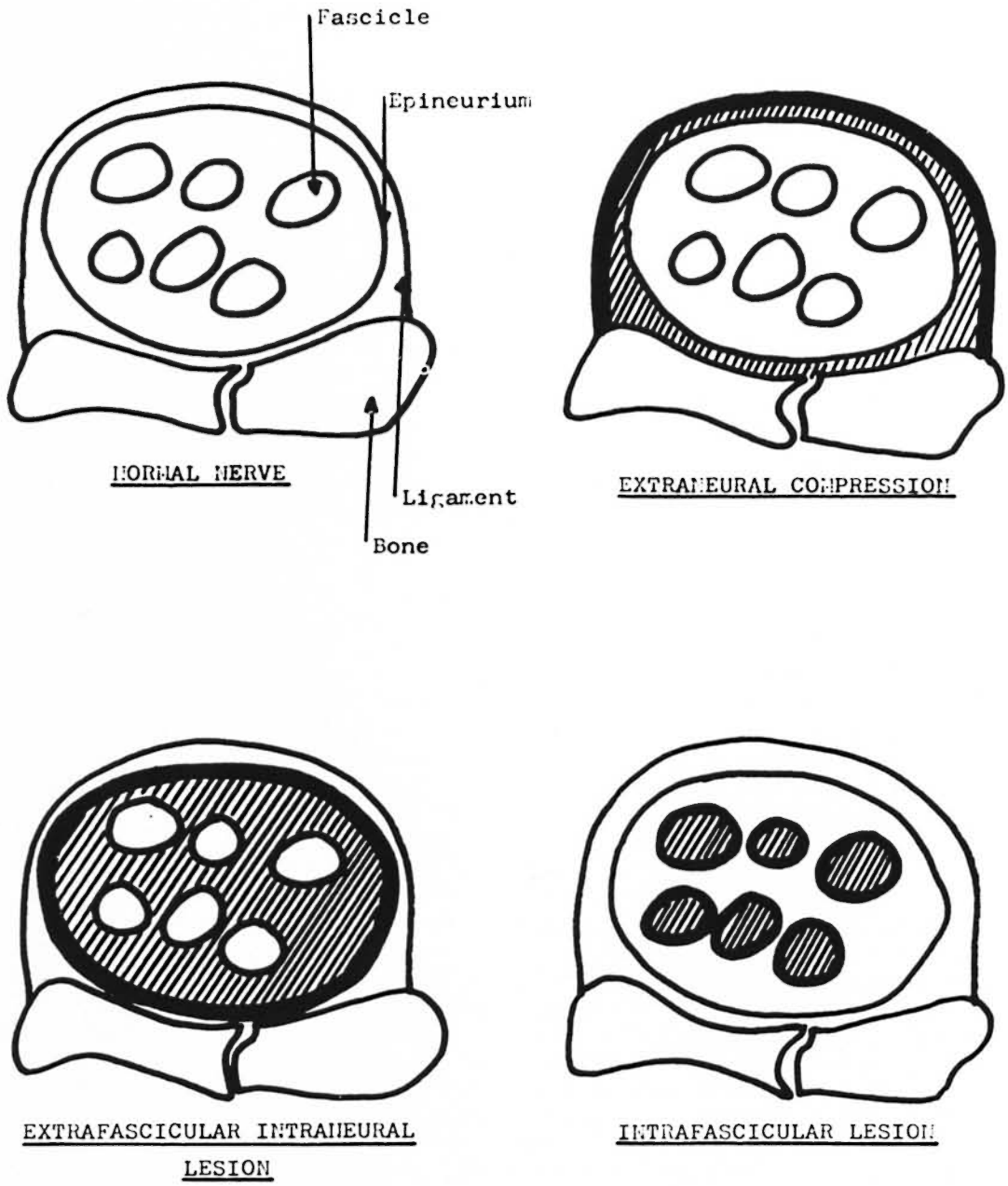

THE FIGURE. Neural lesions in Hansen's disease, pathophysiological mechanisms.

classification is useful in the interpretation of clinical events and surgical findings in the peripheral neuropathies of Hansen's disease. It allows us to select the most appropriate operative technique, i.e., that which best corrects the pathophysiology responsible for the disturbance in each case. Of course clinical reality is often more complicated than the theoretical framework we have just outlined and, in many cases, it shows that lesion mechanisms of two or more types co-exist.

Intrafascicular lesions. The intrafascicular damage to the nerves is produced by

direct action of the bacillus on Schwann's cells. It is virtually pathognomonic of the lepromatous forms of the disease[2, 3, 19]. This is a slow and insidious form of neural aggression. Although its clinical manifestation is late and not very spectacular, it commences very precociously and leads to severe function disablement of the affected nerve. This gradual deterioration may take place without the pain, which serves as a premonitory alarm, and characterizes acute neuritis. The nerve trunks suffering this process of specific bacillary infiltration may develop internal scarring, a fibrosis which transforms them into rigid cords. They are not necessarily thickened but they are equally deprived of any conductive capacity. There is no known surgical treatment which is helpful for this direct bacillary invasion or its residual consequence, secondary hyalinization.

It is important to bear in mind that not all neural damage in a lepromatous patient is due to bacillary infiltration. In periods of reaction, particularly, no matter what the pre-existent degree of deterioration in a nerve trunk, painful alterations of an acute nature may be produced due to the relatively rapid development of compressive intraneural or extraneural phenomena.

Intraneural extrafascicular lesions. In leprosy patients, the external fibrous sheath of the peripheral nerves—the epineuron—is a frequent localization of a proliferative fibrous process which transforms it into a thick and rigid cover, incapable of stretching under pressure from edema or any other endoneural collection. Reactional episodes imply an entire constellation of immune phenomena, and in the nerve trunk this is manifested as an inflammatory event with swelling and interfascicular edema. Clinically it produces a peripheral neurological syndrome which is intensely painful. From the functional point of view, sudden neural deficits may be observed. It is simple to understand that fibrosis of the epineural sheath prevents secondary expansion of the interfascicular edema, increasing intraneural pressure and producing damage due to simple mechanical compression. On the other hand, this very same intraneural pressure imbalance may produce vascular obliteration. It becomes, therefore, an added factor of neurological deficit, since it creates new causes of ischemia of the nerve trunks in which circulation is frequently compromised beforehand due to a specific leprous vasculitis.

In the tuberculoid patient, violent immune phenomena and focal ischemia of the involved nerves have been postulated as the factors responsible for the caseous necrosis which is called "nerve abscess."

Apart from the processes already mentioned—acute neuropathy due to edema caused by a reactional episode and neural abscess—there is another process also produced by intraneural extrafascicular phenomena. This is an acute focal neuritis of nonfunctional nerve trunks with longstanding fibrosis. These totally sclerosed nerves, useless from the functional point of view, may nevertheless suffer inflammatory processes which are very painful, to the point of occasionally making surgery necessary. Logically, the symptomatology of these patients is limited to inflammation of the focus, with pain, swelling, and an increase of local temperature, but without distal neurological symptoms.

Extraneural lesions. The third and last mechanism of lesion in the large nerve trunks of the leprosy patient refers to the possible compression in osteofibrous corridors and narrow anatomic passages such as the epitroclear-oleocranean and Guyon's canal for the ulnar nerve, the carpal tunnel for the median nerve, the supinator tunnel of the forearm for the radial nerve[20], the medial retromaleolar groove of Richet for the posterior tibial nerve, etc. This pathophysiological mechanism has been discussed extensively in the literature[1, 9, 11, 21, 22] and is one of the best known. In our opinion, however, its importance in clinical leprol-

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ogy has been overrated. In fact, although extrinsic compression of the nerves is frequently observed, the preferred topographic localizations of the neural pathology in these patients are proximal to the zones of the osteofibrous tunnels.

Diagnosis is based on the detection of painful or irritative neural symptoms (paresthesia, Tinel's sign, etc.) in the topographic areas corresponding to the aforementioned corridors. This clinical picture can be caused by a decrease of the container (fibrosis of ligaments, traumatic microhematomas, etc.). However, this situation is infrequent in clinical leprology. It is caused more frequently by an increase in the content (edema or tumefaction of the nerves). Whichever is the case, this sudden pressure imbalance inside the tunnels interferes with blood circulation in the nerves producing, first, passive congestion with an increase of the edema and, finally, an ischemia. From the functional point of view, the neurological syndrome would be irritative at the beginning and would later achieve a stage of deficiency.

In some cases, surgical exploration of the nerves within these narrow osteofibrous passages presents the surgeon with a dilemma which must be solved on the spot. If the nerve shows a swollen epineural sheath which is soft and without signs of fibrosclerosis, the preoperative diagnosis of extrinsic compression secondary to neural edema is confirmed, and simple perineural neurolysis—unroofing of the tunnel—will solve the problem. But if the osteofibrous passage is opened and the nerve is found to be thickened, with epineural fibrosis, it is impossible to determine at first sight whether the neurological picture depends on extrinsic compression or intraneural compression. If the latter were the mechanism responsible for the lesion, the necessary operation would no longer be simple peripheral neurolysis. An epineurotomy with endoneural dissection would have to be performed.

The selection of the appropriate surgical technique depends upon this difficult intraoperative differential diagnosis. We have found intraoperative neurography to be of great value in those cases. This is a radiographic contrast technique in which a radiopaque substance is injected inside the nerve and produces images which allow a clear differentiation between intraneural and extraneural processes. The procedure is innocuous if it is carried out with proper care. It has the advantage of allowing a differential diagnosis which enables one to restrict the indications for epineurotomy. These operations, however, are not exempt from risk and consequently they should be reduced to the indispensable minimum.

CLASSIFICATION

The scientific literature of the last few years shows that intense discussions and differences of opinion have been registered on the topic of categorization and classification of neural lesions. In our opinion, many of these discussions are due to the lack of a standardized and unequivocal common language. It is imperative that we systematize classification guidelines and that we define clearly the terms employed in order to avoid misunderstandings and tacit meanings that produce mistakes.

Taking these ideas as a point of departure and accepting the complementary value of diverse classification criteria, we postulate the following system of classification by multiple and simultaneous, non-contradictory guidelines.

Clinical
- Acute
- Subacute
- Chronic

Functional
- Irritative
- Deficitary

Anatomical
- Ulnar
- Median
- Radial
- Posterior tibial
- Lateral popliteral nerve
- Facial

Topographic
- Single lesion
- Multiple lesions

Level
- Subcutaneous branches
- Nerve trunk
- Nerve plexus

Other elements allowing for a better iden-

tification of the neurological disturbance include their relation with the natural history of the disease as well as the clinical form for each patient. This group of data allows us to postulate and identify the pathophysiological mechanism of the neural disturbance and also permits us to indicate appropriate treatment.

Clinical

In regard to clinical guidelines for classification, we have used an arbitrary scale in which we designate as "acute neuropathy," that which produces spontaneous pain; "subacute neuropathy," that which only shows tenderness; and "chronic neuropathy," that which is painless and only manifests itself by a palpable thickening or functional disturbance.

- Clinical
  - Acute = spontaneous pain
  - Subacute = provoked pain (tenderness to palpation)
  - Chronic = painless

Functional

A functional classification of neuropathies depends on the identification of the signs and symptoms and determining whether they are of the irritative or deficit type in each of the functional areas or modalities of the involved peripheral nerve.

Signs and symptoms of the irritative period include:

- Sensory
  - Dysesthesia
  - Paresthesia
  - Hyperalgesia
- Motor
  - Fibrilation of muscles
  - Cramps
- Autonomic
  - Excessive sweating

Signs and symptoms of the deficit period include:

- Sensory
  - Hypesthesia
  - Anesthesia
- Motor
  - Pareses
  - Paralysis
    - Functional incapacity
    - Muscle atrophy
    - Deformities through muscular imbalance
- Autonomic
  - Absence of sweating due to vasomotor paralysis

The classification of a neuropathy according to the guidelines previously mentioned demands a meticulous neurological examination for which, in our experience, the application of a form or neurological protocol has been found to be useful.

Besides the history and physical examination, we carry out some special tests because prevention demands a very early diagnosis of the deficiencies, something that traditional neurological examinations cannot accomplish. In effect, the classic signs of a functional deficiency in a peripheral nerve, such as anesthesia and paralysis, are late manifestations which indicate neural damage dating a long way back. For the identification of the most subtle signs of neural damage, it is necessary to use some of the many complementary examinations. These can be divided into several groups:

Methods for the functional evaluation of peripheral nerves

1) Examination of tactile dexterity and gnosia
2) Examination of motor function
3) Examination of sensibility
4) Examination of nerve conduction
5) Examination of autonomic functions

The detailed descriptions of all these techniques go beyond the intention and limitations of this paper. Therefore, we shall only mention the best-known tests within each group, giving brief attention to those we consider most useful.

Examination of tactile dexterity and gnosia. These are complex tests to evaluate the ultimate functional utility of the hand with the simultaneous participation of sensory and fine motor functions. These tests are applied particularly in the evaluation of the final results of neuroraphies from the rehabilitative viewpoint. However, in progressive deficiency-type neuropathies, their

return to normal occurs late and this makes these tests of little value in leprology.

The better-known tests within this category are:

a) Seddon test (1954) of recognition of coins
b) Graduation of sandpaper (1958)
c) Moberg's picking-up test (1958)
d) Porter's recognition of letters test (1956)

Examination of motor function. From the point of view of simple physical evaluation, this implies examination of the contractile capacity of muscles, the detection of function impairment, the identification of substitution movements or "trick movements," the search for muscular atrophy, and the eventual appearance of deformities secondary to muscular imbalance.

In the evaluation of muscular contraction, clinical practice has used noninstrumental quantitations, such as Daniels' scale or similar methods. If more accurate quantifications are needed, dynamometers of different designs are available although their use is usually restricted to research studies. In any case, motor manifestations of neural deficit in Hansen's disease patients appear late in regard to the diagnostic needs of preventive medicine.

Sensory determinations. We may break down these tests into two large groups. They evaluate deep sensibility on the one hand, superficial sensibility on the other.

Superficial sensibility

a) Swab and pin test
b) Temperature discrimination test
c) Weber's compass or two-point discrimination test
d) Von Frey's test
e) Omer's test (identification of stimuli with nylon monofilaments of different diameters)

The majority of these tests, although they are widely used in clinical practice, do not offer reliable and reproducible results due to their mode of application which is cumbersome, slow, and depends, to a large extent, on the force with which the examiner applies the stimuli.

Deep sensibility tests

a) Tuning fork test
b) Neilson's sensogram (curve of sensitivity to vibrations)
c) Barosensitive gloves

These are experimental studies without clinical application for the time being.

Examinations of nerve conduction. These are bio-electric examinations of the differences in electric potential detected in peripheral nerves. They comprise several tests which have already been widely used in the study of the pathology of the peripheral nerves[1, 23, 24].

The most frequently used determinations are:

a) Cronaxia curves
b) Conduction velocity
c) Latency times
d) Electromyogram

These tests frequently become positive prior to the appearance of clinical symptomatology of neural deficits. Therefore, they are valuable diagnostic tools for detecting early nerve damage.

Examination of autonomic function. These are a group of determinations based on the identification of skin areas where perspiration is normal, indicating the satisfactory function of the autonomic nervous system, as opposed to those areas where deterioration of the neurovegetative fibers contained in the peripheral nerve have led to diminished or suppressed sweating.

The best known are:

a) Minor's test (iodine and starch)[25]
b) Gutman's test (reaction of quinazarine)
c) Moberg and Aschan test (with ninhydrine)
d) Omer's test (cobalt chloride solution)
e) Richter's dermometry (cutaneous conductivity of electric current as a function of its water content)
f) Buratti's test (a variation of the previous test)

[23] Minato, J. Clinical investigation of the peripheral nerve involvement at the upper extremity of leprosy. Abstract in Int. J. Lepr. **26** (1958) 442.

[24] Petrera, J., *et al.* Estudio EMG y de la velocidad de conduccíon motora en la enfermedad de Hansen. Temas Leprol. **19-55** (1975) 8–14.

[25] Charosky, C. B. Los metodos de evaluacion sensitiva. El test do Iodo-Almidon. Acta IX Cong. Argent. Ortop. Traum. **1** (1972) 320–326.

All these tests are indirect evaluations of vasomotor control mediated by the autonomic nervous system. They are subject to a number of variables (thickness of the skin, room temperature and humidity, pre-existing skin lesions, etc.), and for these reasons are not very trustworthy and not easily reproduced. Nevertheless, the well-known precocity of damage to the sympathetic fibers contained in the peripheral nerves, observed in Hansen's disease, makes the measurement of the autonomic function desirable for the early diagnosis of leprous neuropathy.

This has led us to develop in the last few years[16, 26] the diagnostic procedure of radioisotopic scintillography in a gamma camera. Its diagnostic precocity precedes by far the clinical manifestations of deficiencies. The method is based on the identification of areas in which the functional deficit of the autonomous vasomotor system produces circulatory hypoperfusion. Using an intravenous injection of 10 millicuries of technetium 99m., we use a gamma picker camera for performing the determination. A fine collimator is coupled to a computer and allows us to convert scintillographic images into numerical quantifications, graphs, curves, etc. The system also includes a unit of registration on videotape for filing information which can be recovered later and reevaluated for comparative, statistical, or other purposes.

In summary, the two types of tests which are the most useful in early diagnosis of the deficitary signs in leprous neuropathies are: 1) electro-physiological studies (conduction velocity and latency time), and 2) radioisotopic scintillography in a gamma camera.

DIFFERENTIAL DIAGNOSIS

Diagnosis in leprosy cases with nerve involvement is simplified if skin lesions are found together with those of the peripheral nerves. The problem is more difficult to solve if only neural localization is found. Surgical biopsy of the ulnar nerve has traditionally been the fastest method employed to solve the diagnostic problem. However, even if surgical exploration is limited to sampling a fragment of the epineurum, the procedure is potentially dangerous for the functional integrity of a nerve trunk as important as the ulnar nerve and, for this reason, should be abolished[19].

The most important differential diagnoses, according to the predominant neurological manifestation[27], are as follows:

1) Diseases that produce a thickening of the peripheral nerves
   a) Dejerine-Sottas' hypertrophic neuritis
   b) von Recklinghausen's neurofibromatosis
2) Diseases producing deformities of the hand (muscle atrophy, claw hand, etc.)
   a) Syringomyelia
   b) Traumatic ulnar nerve paralysis
   c) Syndromes of the thoracic outlet (scalene, etc.)
   d) Dupuytren's contracture
   e) Sclerodactylia
3) Diseases producing atrophy of the leg muscles
   a) Polyneuritis (infectious, toxic, metabolic, etc.)
   b) Poliomyelitis
4) Diseases producing plantar perforating ulcers
   a) Tabes
   b) Diabetes
   c) Syringomyelia
   d) Myelomeningocele
   e) Thevenard's ulcero-mutilating acropathy
   f) Bureau-Barriere ulcero-mutilating acropathy
5) Systemic diseases producing acute mononeuritis (either single or multiple)
   a) Paraneoplastic syndromes
   b) Diseases of the connective tissue (systematic lupus erythematosus, etc.)
   c) Bernhardt and Roth's paresthesic meralgia

TREATMENT

The therapeutic approach to peripheral neuropathy in patients suffering from Han-

[26] Charosky, C. B., Gatti, J. C., Gabrielli, M., Turianski, L. and Costagliola, G. Angiografía radioisotópica en cámara gamma. Una nueva técnica de exploración neurológica periférica. Leprologia **22** (1980) 23–33.

[27] Balina, L. M., Gatti, J. C., Cardama, J. E. and Wilkinson, F. F. *Manual de Leprologia*. Edit. "El Ateneo," Buenos Aires, 1963, 105–112.

sen's disease should surpass the purely empirical method and no longer be a matter of dogmatic application of the particular preference of each school. Selective discrimination of indications should be applied based upon pathophysiological criteria.

In our experience, the basic guidelines determining therapy are: a) the neural clinical picture (acute, subacute, or chronic); b) the functional capacity of the affected nerve (irritative period, partial or total deficit period); c) the pathogenic mechanism which is probably involved, and d) the form of the basic disease as well as its state of evolution (lepromatous, tuberculoid, acute reaction, etc.). Different situations can thus be identified, some of which are listed below.

Progressive chronic deficitary neuropathy

This is a slow, painless, progressive neural deficit, characteristic of lepromatous patients with intraneural fibrosis secondary to the gradual infiltration by Virchow's cells. Treatment should be based on specific chemotherapy with drugs indicated for Hansen's disease. Physiokinesic and orthopedic treatments are also indicated in order to prevent the disabling consequences of neuropathy. Neural surgery is contraindicated.

Nonprogressive chronic deficitary neuropathy

The patient's main complaint is a long-standing and stable neural deficit. This is practically always irreversible and has produced deformities or mutilations of limbs. This picture may appear in any of the clinical forms of leprosy and is the final result of chronic progressive neuropathy or of one or more acute episodes which have ended up destroying the nerve fibers. From the neurological point of view the damage is irreparable. From the point of view of rehabilitation, physiokinetic and orthopedic treatments are necessary to correct or mitigate the patient's disabilities. Frequently, reconstructive surgery may be indicated, such as tendon transfers, arthrodeses, etc.

Acute irritative neuropathy in reactional lepromatous patients

The usual pathogenic mechanism is an intraneural edema and/or extrinsic compression. Treatment is basically pharmacological, using drugs for the basic disease as well as drugs aimed at controlling the acute reactional episode (glucocorticoids, thalidomide, clofazimine)[28, 29, 30].

In mononeuropathies or oligoneuropathies with few localizations, local measures may be added such as immobilization with orthopedic splints, ice packs (not heat, because it increases inflammation and pain) and, eventually, perineural infiltrations with anesthetic agents and glucocorticoids associated sometimes with diffusing agents[31]. These local injections should always be performed with care to avoid intraneural penetration of the needle, since the intraneural distension provoked by the fluid could be dangerous.

Infiltration should always be perineural. In these patients frequent examinations are indispensable, allowing for the early detection of any change in the functional condition moving from the irritative to the deficitary status.

Acute irritative neuropathies in tuberculoid leprosy

The pathophysiological mechanism which produces lesions in tuberculoid leprosy patients is that of very acute and destructive inflammatory processes. Frequently they lead to ischemia and caseation of an area of the nerve with formation of so-called "neural abscesses." From the therapeutic point of view, we favor the use of high doses of systemic glucocorticoid therapy added to the chemotherapy for the basic disease.

A neural abscess is an indication for immediate surgical treatment aimed at draining the caseous material by carefully decompressing the involved area and trying to preserve the intact nerve fibers throughout the process. The natural history of untreated neural abscesses has two possible final outcomes: 1) partial reabsorption of the caseous material with late calcification of the

[28] Gatti, J. C., Cardama, J. E. and Wilkinson, F. F. *Manual de Dermatología.* Edit. "El Ateneo," Buenos Aires, 1980, 8th ed.

[29] Gatti, J. C. Presente y futuro en la terapéutica de la lepra. La Semana Médica LXXXII-147 (1975) 265–275.

[30] Gatti, J. C., Cardama, J. E., Balina, L. M. and Jaled, M. M. *Actualizaciones Leprologicas.* Buenos Aires, 1983.

[31] Garrett, A. S. Hyalase injections for lepromatous nerve reactions. Lepr. Rev. **27** (1956) 61–63.

residual cavity, and 2) spontaneous drainage with frequent retrograde pyogenic infection through the sinus. Both events are usually accompanied by both the anatomical and functional destruction of the nerve. For this reason, conservative treatment of these cases is not justifiable.

Acute and subacute deficitary neuropathies

This is a heterogeneous group of lesions that may appear in any of the clinical forms of leprosy and at different stages of their evolution. The only common characteristic is that they produce pain (which is the usual reason for seeking care). They may also show functional deficiencies of variable magnitudes.

From the therapeutic point of view, apart from chemotherapy which should not be interrupted, all of these neuropathies should be treated surgically. However, it is indispensable to subclassify this group in order to select the appropriate surgical technique according to the identified pathogenic mechanism.

Lesions due to extrinsic compression (Guyon's tunnel syndrome, the carpal tunnel syndrome, etc.). The division and partial excision of the fibrous bands or ligaments responsible for mechanical compression[20, 22, 32, 33] is indicated. In the case of the ulnar nerve, its transposition to the volar aspect of the forearm has been frequently recommended. This operation requires dissection of a relatively long section of the nerve trunk, and this implies a certain risk for the continuity of arterial circulation of the nerve. Transposition is justified in those osteo-articular processes (fracture callus, etc.) which alter the anatomy of the posterior groove of the elbow, making the placement of the ulnar nerve in a new location mandatory. However this is not the case in leprological neural surgery, where the pathology requiring operative treatment is in the nerve itself and not in its osteofibrous receptacle. For this reason, we consider routine transposition of the ulnar nerve a therapeutic excess. It is not exempt from risk and should be restricted to those few cases in which specific indications for this procedure are found.

Lesions due to intraneural edema. Surgical exploration finds a nerve with an increased diameter, swollen, and with diminished density. The surgical technique to be applied is longitudinal epineurotomy in stripes, that is, multiple superficial incisions in the exterior fibrous sheath of the nerve, parallel to the longitudinal axis of the nerve trunk. This facilitates drainage of the edema fluid. The operation should carefully respect those vessels which go through the epineurium, and the surgeon should refrain from carrying out any dissection between the nerve bundles.

Lesions due to intraneural fibrosis. The proliferation of epineural and perineural fibrous tissue strangles the nerve fascicles and produces ischemia. It may also provoke an increase in intraneural pressure when there is an inflammatory episode with edema. Clinically, the nerve appears enlarged and rigid. Frequently it presents adhesions to the neighboring soft tissues, indicating past inflammation of the epineuron.

The surgical technique to be employed in this case is extensive epineurotomy through a single incision, followed by a careful and restricted endoneurolysis. This interfascicular dissection should be carried out with microsurgical techniques (magnification and appropriate instruments) in order to avoid the division of interfascicular communications[34]. In our experience, when this type of technique is used, care must be taken to avoid the separation of the entire epineural circumference which has adhered to the soft tissues since this preserves part of the necessary circulation. Otherwise, at the end of the operation, the result would be a minutely dissected nerve which is totally ischemic and whose fate would be necrosis and complete functional destruction.

Lesions due to neural abscess. In neural abscess, whose pathogenesis has already been discussed, we employ the most radical of neurosurgical techniques—circumferential epineurectomy in the shape of a collar with interfascicular endoneurolysis. In this operation an entire ring of epineural fibrous tissue is resected in the area surrounding the

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abscess. The nerve bundles are dissected in order to drain all caseous material.

Painful, nonfunctional nerves. Exceptionally, nerve trunks with complete and permanent paralysis are, nevertheless, the seat of intensely painful inflammatory processes. Logically, these are devoid of distal neurological function. When these fibrous cords, devoid of any functional utility, are still a source of uncontrollable pain not checked by simple conservative measures, surgical treatment might be indicated. These are the only cases in which the technique of neurectomy, that is, block resection of the entire painful and inflamed nerve trunk, is indicated. It is superfluous to point out that if the slightest doubt exists regarding the irreversibility of completeness or the damage, neurectomy is absolutely contraindicated.

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