As a follow-up to the historical introduction, it seems appropriate and useful to present the complete translation of Tinel's study on the well-known "tingling" sign observed in nerve regeneration.

The "Tingling" Sign in Peripheral Nerve Lesions

by J. Tinel

Translated by Dr. Emanuel B. Kaplan

It is recognized that it is frequently difficult to make a precise diagnosis in lesions of peripheral nerves.

Is there a division of the nerve, a compression, a tear, or an irritation? Is the nerve in a state of regeneration? Is a palpable neuroma penetrated by axons? Was a nerve suture successful? These are the problems which confront the clinician and are of major importance in diagnosis and treatment.

Pressure applied to an injured nerve trunk frequently produces a sensation of tingling transmitted to the periphery of the nerve and localized to a precise cutaneous region.

We consider that the systematic study of the tingling produced by pressure on a nerve may bring precious help for the solution of the

problems.

It is important to differentiate this frequently present tingling from a sensation of pain which may also be produced by pressure applied to the injured nerve. *Pain* is a sign of *irritation of the nerve*; *tingling* is a sign of *regeneration*, or more precisely, tingling reveals the presence of regenerating axons.

The pain of nerve irritation is almost always present as a localized pain felt at the point where pressure is applied. If it extends along the nerve trunk, it is most intense at the point of pressure. It is associated almost constantly with pain produced by pressure of the muscles, and most frequently the muscular pain is more pronounced than the pain along the nerve trunk.

^{*}Tinel, J.: Le Signe du "Fourmillement" dans les Lésions des Nerfs Périphériques, Press. Med., 47:388-389, Oct. 1915.

The tingling of regeneration is not a painful sensation. It is a vaguely disagreeable feeling; the patient compares it with a sensation of electrical shock. This is hardly felt at the point of compression but is felt most frequently in the corresponding skin distribution. The muscles adjacent to the nerve where tingling is found are not painful.

The two types of sensation produced by pressure of the nerve – pain and tingling – are easily differentiated in almost all cases. The two sensations rarely exist simultaneously in the same nerve or, more exactly, at the same point of an examined nerve because, as we will see later, they may follow one another along the same nerve trunk. The two different signs produced by pressure applied to the nerve are similar to the symptoms which are revealed by examination of skin sensation. Regeneration of the nerve is manifested by paresthesias of a more constant type which are more painful and are associated with hypoesthesia produced by touch, by puncture and, especially, by slight friction of the skin.

However, in all cases the symptoms produced by pressure of the nerve—pain which indicates the irritation of the axons or tingling which indicates their regeneration—are much easier to differentiate than the signs of cutaneous sensibility. They are also more constant and appear much earlier; they furnish more precise, more localized and more important information.

A systematic study of tingling produced by pressure applied to the nerve frequently permits the clinician:

To establish whether there is complete or incomplete interruption of the nerve:

To determine the exact site and extent of the lesion:

To reveal early regeneration of the axons and to follow their progress and disclose their importance.

A few illustrations are presented:

 In total nerve interruption along the course of the nerve trunk a definite zone can be found where pressure produces tingling in the cutaneous distribution of the nerve.

This zone of tingling is not extended. It does not exceed 2-3 centimeters; it is constant and absolutely fixed; it persists for weeks and months; it is confined to the course of the nerve only, and there is no extension either proximal or distal to the lesion where pressure could produce tingling.

This zone indicates that at this precise point the suddenly interrupted axons have undergone local regeneration and, being unable to cross the obstacle or find the distal segment, they wound up into a more or less large neuroma.

In complete interruptions of the nerve produced by very tight entrapment, the same characteristics of fixation, of permanence and precise limitation are found, but the zone of tingling is more extended; it may reach 6, 8 or 10 centimeters more over the course of the nerve. For instance, in frequent compressions of the radial nerve associated with fractures of the humerus, it is possible by studying the tingling along the entire length of the nerve to determine if the nerve is caught in the bone callus or is interrupted at the level of the superior or inferior level of the fracture.

It is necessary to mention that simple pressure of a nerve caught in a bone callus does not produce tingling easily. The tingling is better re-

vealed by percussion applied to the callus.

In any case, if the zone of tingling remains fixed and does not pass the inferior limits of the callus, or, if after many weeks tingling does not spread distal to the callus, it means that the entrapment produced a sufficient stricture of the nerve to interrupt the regeneration of the axons and prevents them from passing through.

It is possible in certain instances to find along the course of the same nerve two different sites of tingling corresponding to two different

lesion levels.

For instance, we have seen two wounded men with radial nerve paralysis in the upper part of the arm. There was one zone of tingling of the radial nerve at the level of the bullet exit over the posterior aspect of the arm; there was another zone of tingling, more extended, over the lateral side of the arm at the level of a very large fracture callus; these two zones were fixed and limited. No tingling could be produced distal to the fracture site. Surgical intervention showed that the nerve was partially destroyed by the passage of the bullet and that a few fibers which escaped injury were found more distal and were compressed and interrupted in the fracture callus.

It is also possible to observe partial tingling of the nerve. Pressure of the sciatic nerve, for instance, may show a lesion limited to the medial or lateral side which will accordingly produce a tingling sensation localized in the cutaneous distribution of the common popliteal or the posterior tibial nerve.

Another instance presented a man with paralysis due to a wound over the middle of the thigh which presented a double area of tingling. Pressure applied to the nerve at the wound level produced tingling over the sole of the foot which corresponded to the posterior tibial, but pressure applied distal to the wound produced tingling in an area which reached the popliteal space and extended over the dorsum of the foot in the zone of the lateral peroneal nerve. In this case, a complete interruption of the posterior tibial component of the sciatic nerve with nonadvancing tingling was found. In addition, an incomplete interruption of the lateral part of the nerve was present with a progressive transmission of tingling through the regenerated axons down toward the popliteal space.

 In effect, incomplete interruption of a nerve or, more exactly, lesions permitting the passage of regenerating axons are characterized by

progressive extension of the tingling.

Accordingly, tingling can be seen to appear distal to the lesion, extending progressively toward the periphery along the course of the nerve. A nerve which demonstrates tingling below the lesion is a partially or totally regenerating nerve. It is possible to see the progress from week to week and thus follow the slow progress of regeneration of the axons. One may assess the speed of regeneration of the nerve and possibly judge its importance according to the intensity of tingling and the extent of the area of cutaneous response.

This also pertains to nerve sutures where it is possible by observing the progressive extension of tingling to quickly determine the success of

the intervention.

Gradually, as the tingling extends and increases in intensity toward the periphery it tends to decrease and even to disappear at the site of the original injury. Thus, tingling extends eccentrically, always preserving a wide area.

Therefore, it is always necessary to explore the nerve along its entire course. The following case illustrates this principle. We examined a man who had a complete paralysis of the sciatic nerve. Five months previously he had been wounded at the proximal end of the thigh. A complete paralysis had developed since then, but the muscle tone was not completely gone and the zone of anesthesia appeared somewhat reduced. Tingling could not be found over the sciatic nerve at the level of the injury or distal to it. We thought that this indicated a poor prognosis. But on further examination we found some tingling over the popliteal space and the upper half of the leg corresponding to the branches distal to the level of injury of the nerve. This indicated that the nerve was in a stage of advanced regeneration. In effect, we could find some contractility under faradic stimulation over some fibers of the gastrocnemii, peronei and tibialis anterior.

5. The same progressive extension of the tingling zone is found in

incomplete interruption with nerve irritability.

It appears that sometimes, however rarely, the signs of irritation and of regeneration are found in the same nerve; the interpretation is then quite difficult. However, generally speaking, the patient complains of a radiating painful numbness or painful pricking rather than a tingling.

In the majority of cases, however, tingling progressively replaces the pain produced by compressing the nerve. Tingling, figuratively speaking, pushes the pain away. In accordance with the advancement of tingling, the nerve trunk and the muscles become less painful. It is found that a sciatic nerve, which becomes painless, tingles on pressure of the thigh, while the nerve and the muscular masses of the leg are still painful.

It is easy to see, as illustrated by the few examples, how helpful the

sign of tingling can be.

It is obvious that the systematic search of this sign does not eliminate in any way a careful examination of motor disturbances and the electrical sensory, and trophic changes. This sign is simply used in addition to the other observations and, in the majority of cases, only confirms the diagnosis and makes more precise the clinical findings.

It would indeed be unwise to overestimate the importance of the tingling sign, especially when the sign is absent. Thus it is important to

mention the following points:

Tingling induced by pressure of the nerve does not appear generally before the 4th and even the 6th week after trauma.

In effect it is known that each nerve lesion, section or entrapment is followed by the first phase of degeneration—descending wallerian degeneration—which extends to the end of the nerve, and ascending degeneration or retrograde which does not extend beyond several nerve segments. Even when limited they are accompanied by deep changes in the cells which give origin to the nerve. It is only after this first phase of degeneration that fibrillation of the axons of the central end, and their regrowth begins. It appears that in man this phase of regeneration does not appear earlier than after 3 or 4 weeks; it appears earlier or later according to the age, the state of health and the regenerative factors of each individual. It is this period of new axon formation which corresponds to the first appearance of tingling.

2. Equally the tingling disappears as soon as the nerve returns to its normal structure and the newly formed axons become mature. Generally, it is at the end of 8 or 10 months that tingling appears to stop; wide variations are naturally observed according to individuals, lesions and the length of degenerating nerve. We have already mentioned that tingling disappears in an eccentric manner running progressively toward the

periphery (distal end) of the nerve.

3. Finally, tingling may be absent in certain rare cases. Then, it indicates either that the lesion is very mild and that it did not produce any deep destruction of the nerve fibers or, on the contrary, that no regeneration occurred, as is observed at times in older individuals and patients with long illnesses or with disturbing nutritional problems.

Tingling therefore, does not constitute an absolutely constant fixed sign which is easy to interpret. It cannot replace precise and repeated examinations of the patient. It is valuable only in association with the total

sum of other clinical symptoms.

With all these reservations, nevertheless, tingling appears to help clarify at times certain diagnostic neurological problems and to furnish precise indications for prognosis and treatment of peripheral nerve lesions.