

TOURNIQUET PARALYSIS SYNDROME

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A TOURNIQUET is usually applied as a first-aid measure to stop bleeding when blood vessels are damaged in battle casualties as well as with injuries in civilian life. It is also widely used in orthopedic, nerve, or plastic surgery when a bloodless field is required.

Paralysis induced by a tourniquet is said to be rare, and very few cases have been reported in the literature. The statistics would probably be increased if one were more aware of the existence of the characteristic clinical syndrome which may follow the application of a tourniquet and which is the subject of this paper.

This complication can be overlooked for several reasons. First, the paralysis and the associated phenomena can be of relatively short duration. Secondly, the lesion can be a mild one and result in an incomplete paralysis, which may be interpreted as difficulty in moving the parts because of the surgery. And, thirdly, the sensory examination is usually confined to the testing of pain sensation with a pin prick and, because of the supposedly normal sensory findings, the incomplete or even complete paralysis is often erroneously diagnosed as hysterical.

In spite of extensive experimental work in the animal and numerous studies in man, the mechanism of the paralysis itself has not been established. Most of the workers have attributed the symptoms to ischemia of the nerve. Sometimes the complication induced by a tourniquet has been erroneously associated with the Volkman type of contracture, which is a completely different syndrome.

The tourniquet is obviously an instrument which can produce ischemia, but depending upon the type used, the duration of its application, and the amount of pressure it can also cut deeply into skin, muscles, and nerves.

The symptoms observed in tourniquet paralysis can be identical with those seen in some cases of pressure paralysis. The first good clinical studies on the effect of compression on nerves in man were reported by J. B. Bastien and A. Vulpian in 1855. In 1861 Duchenne de Boulogne described several cases of pressure paralysis in which the palsied muscles retained normal excitability to electrical stimulation. In 1862 A. Waller described the different symptoms of pressure on nerves in man. In 1872 S. W. Mitchell showed various forms of pressure resulting in paralysis. In 1876 W. H. Erb studied the electrical stimulation of the nerves, and he also reported cases with normal response to stimulation of the distal end of the nerve. In 1899 J. J. Déjérine and Bernheim reported one case with autopsy in which there was an injury to the radial nerve with conduction loss and in which the excitability was

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retained below the injury. In 1920 C. H. Frazier and S. Silbert described injuries in which the nerves were not affected directly but in which a bullet had caused an indirect injury to the nerve, resulting in a dissociation of motor and sensory findings. In 1923 G. Bourguignon and H. Laugier studied the disturbance in sensations and loss of conduction of motor nerves due to temporary ischemia, using both inflated cuff and an Esmarch bandage. In 1931 T. Lewis and E. E. Pochin made extensive studies on paralysis and dissociation of sensations resulting from either the experimental use of an inflated cuff or from direct pressure on nerves. Cases with tourniquet paralysis were reported by N. L. Eckhoff in 1931. A series of interesting experiments on paralysis induced in animals by direct pressure or by tourniquet were performed by D. Denny-Brown and C. Brenner in 1944; they also described one case in man. In 1945 J. Spiegel and P. Lewin reported three cases in which the nerve was damaged by the use of rubber tourniquets.

A great number of studies have been made on conduction and blood supply of nerves, which are interesting but are too numerous to be analyzed here.

The reversible disturbances in sensations and the paralysis resulting from the experimental use of an inflated cuff have been analyzed by different authors. The data is conflicting because the loss of sensations is often so rapid and the various defects are so much intermingled that it is difficult to make a thorough study of each individual mode of sensation; furthermore, the experiments are usually accompanied by some discomfort and even pain, which increase the difficulty of the study. After release of pressure the return of the different functions occurs so rapidly and the accompanying paresthesiae and tinglings are so strong that it makes the study of the sensations difficult to analyze.

CASE MATERIAL

In cases of tourniquet paralysis an analysis of the different sensations is relatively easier, because there is usually a very slow return of the different functions. The electrical and sensory tests can be repeated many times on one patient, and the findings can be checked from day to day. In some of those cases the patients have been followed for many weeks until complete recovery.

It is also interesting to point out that in none of the patients examined was there any spontaneous pain or any paresthesia that would interfere with the study of the defects in sensations.

Seven cases of complete paralysis resulting from the use of a tourniquet have been studied. Three were induced by rubber tube, two were the result of Esmarch bandages, and two were due to the use of an inflated cuff.

In addition to those seven cases of tourniquet paralysis, over 100 cases of pressure paralysis of different nerves have been examined. These cases may have quite similar symptoms and will be the subject of a later report.

Two cases of tourniquet paralysis syndrome will be described in detail. The findings in the other five cases were exactly similar.

CASE 1.—A 40-year-old woman was admitted to the hospital for the removal of a ganglion of the right wrist and was operated on March 15, 1951. A towel was applied on the upper arm and over it an Esmarch bandage was tied around the arm several times. The operation lasted 47 minutes.

After the operation the patient complained of complete paralysis of the muscles of the forearm and hand and numbness of the hand. She was examined by me for the first time 14 days after the onset and about once a week at regular intervals for over three months until complete

recovery. The patient was of dark complexion. At 8 cm. above the elbow there was a small and well-defined band of very dark pigmentation of the skin, 2 mm. in width, encircling the upper arm. Apparently more pressure was applied at that level, resulting in an actual damage to the skin.

On first examination, 14 days after the onset of paralysis, the following signs were noticed (Fig. 1):

Motor function: There was complete paralysis of all muscles of the forearm and hand supplied by the median and ulnar nerves and also of all muscles supplied by the radial nerve below the brachioradialis muscle; the anconeus muscle escaped paralysis.

Light touch: When tested with cotton or von Frey's hairs, there was absence of sensation in the entire palm of the hand, in the dorsum of the tip of the fingers, and in an area between the thumb and index finger in the dorsum; the loss of touch sensation corresponded consequently to the distribution of the radial, median, and ulnar nerves.

Superficial pressure: This was also lost in the same area as touch; the patient was unable to feel the heaviest von Frey's hair.

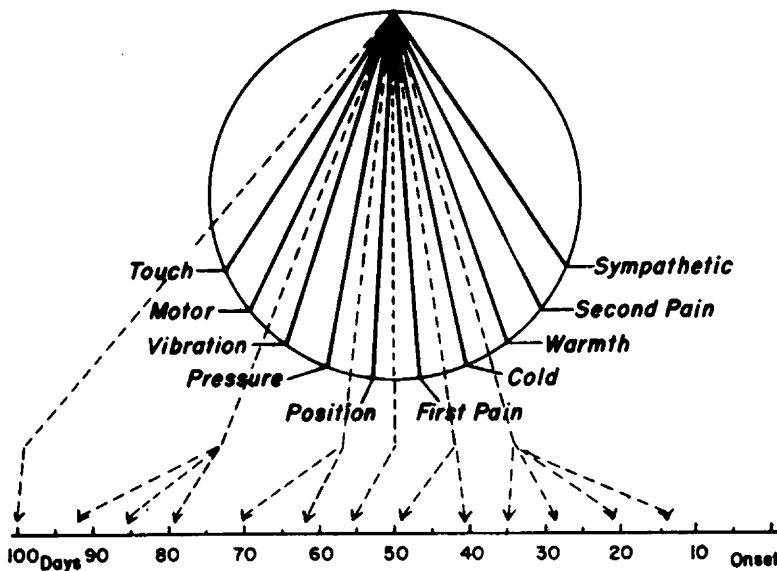


Fig. 1.—Tests were done at regular intervals after onset, as indicated by arrows. Dotted line to extreme right separates unaffected from affected modalities. Remaining dotted lines indicate time of return of affected modalities. There was a progressive return to normal, motor and touch being the last functions to recover. Syndrome in this case was typical 56 days after onset. Before this time it was slightly unusual because of severity of injury to nerve, with involvement of cold and first pain. During entire period of paralysis, stimulation of distal end of nerves resulted in normal muscular responses. There was a block of conduction where the tourniquet was applied, that is, no response to stimulation above lesion.

Proprioceptive sensation: Position sense and appreciation of passive movements was absent in all fingers and in the wrist.

Vibration sensation: This was absent in the hand and wrist.

Superficial pain: The entire palm of the hand was very painful to pin prick. In the area supplied by the median nerve there was a delay of 1 to 1.2 seconds in perception of pain for the distal phalanges of the thumb, index, and middle fingers. The threshold for pain, measured with the algesiometer, was of an average of 20 gm. in the distribution of the median nerve and 10 gm. in ulnar distribution. The sensation of pain was accompanied by a strong affective reaction and was particularly unpleasant. On the normal hand, 20 gm. for the median nerve distribution was felt as a sharp point but it was not actually painful.

Temperature sensation: The appreciation of cold was slightly disturbed in the hand; the answers were irregular. Warmth sensation was normal.

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Skin resistance: Comparison with the normal hand showed no difference except for a small patch of increased resistance of about 1 cm. square in the thenar region.

Electrical testing: Stimulation of the median and ulnar nerves either at the elbow or at the wrist resulted in normal responses in all muscles innervated by these nerves. Stimulation of the same nerves above the elbow just below the line of pigmentation resulted in a normal response. Stimulation of the radial nerve at that level also resulted in a good response. When the stimulating electrode was applied to either of those three nerves just above the pigmentation line, there was no response. There was complete block of conduction even with very strong current.

Direct stimulation of the motor points of the different muscles supplied by the median, ulnar, and radial nerves showed normal excitability. Chronaxie figures and strength-duration curves were normal with the exception of the thenar and hypothenar muscles and the first and fourth dorsal interossei muscles which showed some denervated muscle fibers. Denervation was characterized by a slow response to stimulation with a current of long duration, a shift of the strength-duration curve to the longer time. There was an increase of the chronaxie figures up to 10 times the normal value. The stimulation of the ulnar and the median nerves at the elbow and at the wrist with a constant current failed to give the usual tingling sensation referred to the palm of the hand that they supply. The sensation of tingling was also absent when the stimulation was applied to the nerve below the small band of skin pigmentation but was felt when the stimulating electrode was applied just above that area; the tingling was referred to the hand. It is interesting to note that this patient at no time had experienced any paresthesia or any spontaneous pins-and-needles sensations.

Pilomotor reflex was strong in the entire forearm when electrical stimulation was used. Plethysmographic recordings of the fifth finger were also taken, and they showed the same readings as on the left side. The hand was warm, pulse was good, and oscillometric readings in the forearm were normal. One week later, that is, 21 days after onset, there was no significant change in the clinical findings.

Testing 28 days after onset of paralysis gave the following results:

Motor function: There was still complete paralysis of all muscles.

Proprioceptive sensation, vibration sense, light touch and pressure sensation were still completely absent.

Superficial pain was increased, as mentioned previously, but a pressure of 10 gm. when using the algometer was very painful in the distribution of the median and ulnar nerves. The threshold of pain was diminished. The same delay in the perception of pain was noticed, that is, 1 or 1.2 seconds.

Heat and cold sensations were normal.

Skin resistance was normal.

Electrical testing showed the same findings; conduction block for the median, ulnar, and radial nerves.

Testing 41 days after onset gave the following results:

Motor function: For the first time there was a trace of contraction in the first dorsal interosseous muscle, but there was still complete paralysis in all the other muscles.

Light touch with cotton or with the von Frey's hairs showed complete anesthesia for the median, ulnar, and radial nerves.

Superficial pressure, proprioceptive, and vibration sensations were still absent.

Superficial pain: There was a delay of 1 to 1.2 seconds when the tip of the thumb, index and middle fingers were tested for pain either with the pin prick or with the lowest pressure of the algometer, that is, 2 gm. Pain sensation was still increased and was accompanied by a strong affective reaction.

Heat and cold sensations were normal. Skin resistance was normal.

Electrical stimulation showed no change. There was a normal response below the lesion and no response above the pigmentation line, with the exception of a slight response in the first dorsal interosseous muscle.

Testing 49 days after onset gave the following results:

Motor function: With voluntary motion there was a slight contraction in the first and second dorsal interossei and a trace of contraction in the flexor carpi ulnaris and also in the palmaris longus. Other muscles were still completely paralyzed.

Touch sensation: There was complete anesthesia when tested with cotton-wool and von Frey's hair. Vibratory sense showed some return in the hand but was absent in the index and middle fingers.

Proprioceptive sensation and appreciation of passive motion were disturbed in the fifth and middle fingers but were normal in the other fingers as well as in the wrist.

Superficial pressure was painful in the thenar muscles but not elsewhere.

Superficial pain: Pin prick sensation as well as 2 gm. with the algometer was very painful. There was no delay in the perception.

Electrical stimulation of the nerves above the tourniquet line resulted in response in the muscles that the patient could contract with voluntary motions.

Subsequent examinations showed a slow return of the different modes of sensations and of motor functions. When the patient was able to contract certain muscles with voluntary effort, they could always be stimulated when the active electrode was applied above the line of pigmentation. Below the line the response was always good.

Testing 100 days after the onset gave the following results:

Motor functions: The return of motor function was complete in all muscles.

Proprioceptive, vibration and superficial pressure sensations were normal.

Temperature sensation and skin resistance were normal.

Light touch was normal in the entire hand except for hypesthesia in the distal phalanx of the middle finger.

Superficial pain: Pin prick was felt as very painful and definitely sharper in the entire thumb and in the index finger as compared with the normal parts of that hand or as compared with the other hand.

The electrical stimulation showed normal responses below as well as above the pigmentation line marked by the tourniquet.

CASE 2.—A 21-year-old man had suffered from severe generalized rheumatoid arthritis since the age of 7. It was rather difficult to evaluate the muscular functions of the left arm because of the ankylosis of the elbow, wrist, and of the metacarpal joints of some of the fingers. He had good use of the index finger and thumb and had fairly good range of motion in the distal phalangeal joints. The different modes of sensations were normal. He was operated on for the ankylosis of the left elbow. An arthroplasty with excision of the radial head was performed. This was done on Nov. 7, 1949. A rubber-tube type tourniquet was applied for an hour and a half without incident.

On March 28, 1951, he was operated on again for revision of the elbow arthroplasty. The regular inflated cuff tourniquet was not available at that time, and since the patient had no difficulty with the rubber tube tourniquet in his previous operation this was used again. It was applied for 2 hours 40 minutes.

Soon after the operation all motion of the fingers was lost.

Thirty-eight days after the onset, there was complete paralysis of all muscles supplied by the radial, median, and ulnar nerves.

Electrical stimulation of those nerves proximal to the site of the application of the tourniquet failed to give any response in the muscles supplied by them. At the elbow and at the wrist there was a good response to stimulation of the nerves. A few denervated muscle fibers were found in the abductor digiti quinti and in the abductor pollicis brevis, but all the other muscles had normal excitability to direct stimulation.

Proprioceptive sensation: Position sense and appreciation of passive motion in all of the joints that were not previously ankylosed, that is, the thumb and index and distal phalanges of all fingers were completely lost (Fig. 2).

Vibratory sense was absent in the entire hand.

Light touch: There was absence of sensation to cotton and the von Frey's hair in the ulnar and median nerves. There was normal touch sensation in the radial nerve distribution.

Light pressure was not felt; deep pressure on the tip of the thumb, index, and middle fingers was painful, but not on the fourth and fifth fingers.

Warmth and cold sensations were not affected.

Skin resistance was normal.

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Superficial pain: Pin prick was very painful, and there was strong affective reaction, with withdrawal of the extremity. With the smallest pressure of the algometer, that is, 2 gm., the sensation was painful and unpleasant as compared with the normal skin. This was a real hyperalgesia, with diminished threshold of pain. There was no delay in the sensation of pain, indicating that the fast pain fibers were not affected. Stimulation of the skin of the tip of the fingers with electrical current of small intensity resulted also in a very unpleasant sensation of pain without any delay. When a constant electrical current was used on the median and ulnar nerves at the wrist, the normal tingling sensation referred to the hand was absent. There was only a shock-like sensation going down to the fingers.

Testing 74 and 88 days after onset gave the following results:

Motor function: There was partial return of motion in the extensor pollicis longus, abductor pollicis longus, and in the thenar muscles and flexor profundus for the third, fourth, and fifth fingers.

Proprioception: Positional sense was normal. Appreciation of passive motion was normal.

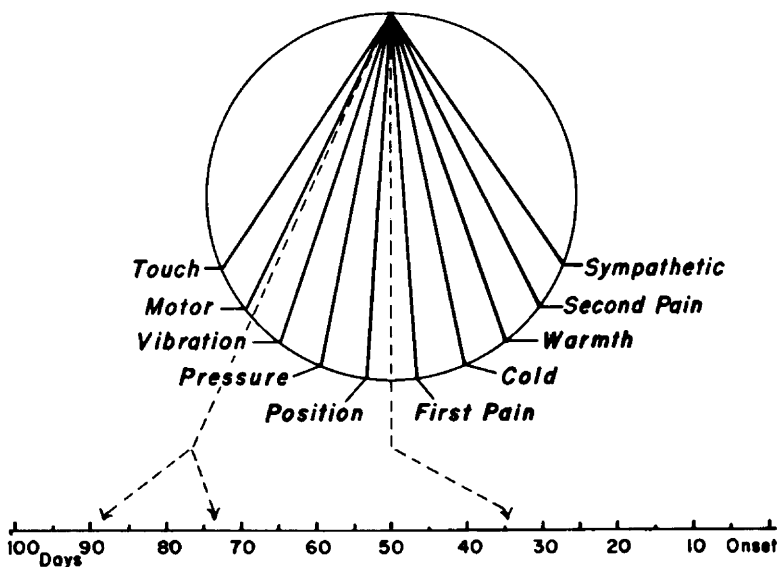


Fig. 2.—Tourniquet paralysis syndrome with typical sensory dissociation. First examination was done 35 days after onset of paralysis. There was a block of conduction at site of application of tourniquet. Muscular response to electrical stimulation of nerve distal to injury was always normal. Dotted line separates unaffected functions on right side of circle from affected ones on left. When tested 74 and 88 days after onset, position, pressure, and vibration sensations had recovered; motor and touch were still affected and they were the last functions to recover.

Vibratory sense was normal.

There was slight pain to deep pressure in the tip of the third, fourth, and fifth fingers.

Light touch: There was absence of touch sensation when tested with cotton in the entire palm of the hand. Both median and ulnar nerves were still affected. Tests with the von Frey's hair showed impairment of touch.

Light pressure was felt normally.

Sensations of warmth and cold were normal. Skin resistance was normal.

Superficial pain: Pin prick was still very painful using the smallest pressure of the algometer, that is, 2 gm. There was also a strong affective reaction.

Electrical stimulation of the ulnar and median nerves at the wrist resulted in a shock-like sensation, but there was no tingling.

Ninety-five days after onset, there was complete return of motor function; sensation was normal. There was no unusual pain to pin prick. There was a normal tingling to electrical stimulation of the median and ulnar nerves at the wrist. There was no block of conduction.

It is worth noticing that after the first operation, when a rubber-tube tourniquet was applied for an hour and a half and when there was no complication, a very strong tingling sensation followed the release of the tourniquet. After the second operation there was no tingling sensation or any paresthesia, indicating a block of the fibers responsible for the tingling.

In the other five patients with tourniquet paralysis there were similar findings.

COMMENT

The purpose of this paper is not to attempt to explain the type of tourniquet that should be used nor its mode of application but rather to give a description of the clinical symptoms that accompany a tourniquet paralysis. The study of one or more cases can be found in the literature, but none of the authors has described the existence of an actual syndrome.

It is well known that the number of cases of tourniquet paralysis has decreased since the thick rubber tube has been avoided and the Esmarch bandage or, rather, the inflated cuff has been used. This by itself indicates that ischemia most likely is not the cause of the complications resulting from the application of the tourniquet, since ischemia should be the same for any tourniquet, but that the mechanical pressure plays a greater role, if not the only one, in the etiology of the paralysis. The tourniquet should be applied where the nerves are best protected by the muscles and, if possible, should avoid compressing them against bony structures.

The two cases here reported in detail, as well as the other five cases, indicate clearly that damage to the nerves was confined to the narrow area where the pressure was applied at its maximum. The area was small and sharply localized. In the first case there was a well-defined line of dark pigmentation of the skin. There was a block of conduction which affected the three mixed nerves of the forearm—radial, median, and ulnar. This was easily demonstrated by the electrical stimulation. A very slight displacement, or about 2 mm., or a slight rotation of the stimulating electrode on the skin would give or fail to give a response in the corresponding muscles depending on whether the current was applied above or below the nerve block.

The nerves distal to the block had normal responses to electrical stimulations. This obviously indicates that the ischemia that took place below the line of pressure did not result in any change in the excitability of the nerves and the muscles. In addition, the electrical stimulation of the mixed nerves with a constant current distal to the line failed to elicit the usual tingling sensation referred to the hand. When the stimulating electrode was slightly rotated and displaced above the line of damage, a strong tingling sensation referred to the area of the skin supplied by the sensory fibers of the nerves took place. This obviously indicates that the touch fibers were intact central to the site of the injury. Furthermore, it also indicates that the sensory fibers, in the first case, for instance, were not affected above the line of pigmentation, and yet the Esmarch's bandage had been applied over a wide area of the upper arm. There is consequently no doubt that there was a damage to the nerve exactly at the level where the line of pigmentation was noticeable. Yet the ischemia due to the application of the tourniquet took place in a much larger area. Therefore, the block of conduction of the motor fibers as well as the sensory dissociation were the result of the mechanical pressure localized to a very narrow area and were not the result of the ischemia, which involved a large part of the upper arm and the entire forearm and hand. In the first case, the anconeus muscle was the only muscle of the forearm

that escaped paralysis. The nerve branch that supplied that muscle escaped injury most likely because it was deeply located and protected by the triceps muscle against direct pressure.

Paresthesiae or pins-and-needles sensations are typical and characteristic symptoms in all experiments in which the inflated cuff has been applied above systolic pressure for a certain length of time. It is interesting to notice that none of the patients with tourniquet paralysis, nor any of the patients with pressure paralysis, experienced any paresthesia at any time. There was no spontaneous tingling sensation.

Obviously, in patients with either tourniquet paralysis or pressure paralysis, the tingling sensation is absent because of the block of the fibers conducting that sensation, namely touch fibers. Consequently, the absence of paresthesia becomes an important diagnostic and prognostic symptom.

Occasionally, the sensation of warmth or cold may be slightly impaired, but this is rare. There may be some denervated muscle fibers in some of the paralyzed muscles. This would depend on whether or not, in addition to the block of conduction, there has been a more serious damage to some of the nerve fibers.

Pain sensation was always present. The first case was a rather a severe one. There was a delay in the pain perception because the first or fast pain fibers were affected, but they recovered after a relatively short time. The delay for the pain sensation of the finger tip was 1 to 1.2 seconds, which would indicate that the pain fibers that were able to conduct pain had a conduction time of about 1 meter per second. They were consequently the slow conducting fibers.

All patients with tourniquet paralysis who have been tested showed evidence of real hyperalgesia. There was an increased pain sensation with a low threshold. In addition, there was a very strong affective reaction. It is interesting to notice that in some areas of the hand, when the patient was recovering, the real hyperalgesia with low threshold was still present when touch sensation had returned to normal. Consequently, the hyperalgesia does not seem to be the result, as has been sometimes postulated, of the absence of touch or other sensations.

In tourniquet paralysis the syndrome consisted of a loss of the following functions: touch, motor, light pressure, vibration, and position senses. Warmth, cold sensation, pain, and sympathetic fibers were not affected. The affected functions would correspond most likely to the larger fibers described by Erlanger and Gasser, the A fibers. The larger fibers would consequently collapse before the smaller ones. If the damage is severer, as in the first case, the fast or first pain fibers and some of the fibers conveying cold sensation can to some extent be impaired. As a rule they are not involved in the tourniquet syndrome.

In none of the cases was there evidence of appreciable atrophy, because the motor fibers were still in continuity, as evidenced by the good response to the distal portion of the nerve and also because the repair took place in a relatively short time; very few muscle fibers were denervated. None of the patients showed evidence of a Tinel's sign. The return of function that takes place is apparently the result of a local repair. None of the cases showed any neuroma sign at the site of the injury.

When the syndrome of tourniquet paralysis is complete, the duration of the impairment of motor function and sensation can be as long as three months or even longer.

SUMMARY AND CONCLUSIONS

In a tourniquet paralysis there is a disturbance in the functions of the peripheral nerves which is apparently due to the mechanical pressure of the tourniquet on the nerve and is not the result of ischemia.

There is a constellation of symptoms that justify the term syndrome. The characteristics of this syndrome are as follows:

Motor function: There is paralysis with hypotonia or atonia, but no appreciable atrophy.

Sensory examination shows a dissociation of the sensations. The fibers subserving touch, pressure, vibration, and position sense are affected, and those sensations are usually absent. Pain sensation is never lost. In most of the cases there is actual hyperalgesia, that is, the pain is felt with a low threshold. In severe cases the first or fast pain can be affected. Heat and cold sensations are usually not affected or slightly disturbed.

There is no paresthesia or tinglings after release of the tourniquet, which is indicative of a block of the touch fibers, responsible for the tingling sensation. There is no Tinel's sign. There is no sign of neuroma at the site of the injury.

Sympathetic fibers are not affected. Pilomotor reflex is normal. Skin resistance is normal. Color of the limb is normal. Plethysmographic findings are normal. Temperature of the skin is normal.

Electrical studies show that there is a block of conduction characterized by lack of response to stimulation of the motor nerve above the injury, a good response below the injury. There is no tingling sensation to stimulation of the sensory fibers distal to the site of injury, but there is a tingling sensation when the nerve is stimulated proximal to the lesion. The electrical stimulation can localize the level of the lesion.

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