Technical Nerve Conduction

Technical Nerve Conduction:

Pitfalls and Solutions

By

Paul Seror

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ABOUT THE AUTHOR

Dr. SEROR is a doctor specializing in rheumatology and neurophysiology, with a particular focus on electroneuromyography (ENMG), specialties that he has been practicing in private practice in Paris since 1984. He is a former resident and clinic fellow at the Montpellier hospitals and was affiliated with the rheumatology department at the Cochin Hospital and the ENMG department at the Salpêtrière Hospital.

He published his first article on somatosensory evoked potentials in newborn babies as a co-author in Montpellier in 1982. He published in 1984 his first ENMG article as first author. This considered a rare, but fascinating pathology, the anterior interosseous nerve syndrome, a rare clinical form of neuralgic amyotrophy of Parsonage and Turner. Over the years, he has become one of the leading specialists in this disease and has written many articles on it. For this reason, the French Society of Rheumatology recently asked him to write an update on this condition for the Encyclopédie Médico-Chirurgicale.

He was also highly interested in, and wrote many articles about, the most common nerve entrapment syndromes, such as the median nerve lesion at the wrist (carpal tunnel syndrome) and the ulnar nerve lesion at the elbow. Additionally, he showed interest in less common forms, such as the ulnar nerve lesion at the wrist and the lateral femoral cutaneous nerve lesion at the anterior superior iliac spine. The latter, although typically regarded as rare, has been shown to be the most common nerve entrapment syndrome of the lower limbs.

On PubMed, there are 118 references with his name. He has published numerous other papers published in non-referenced journals but widely distributed among doctors specializing in rheumatology or neurology, and has delivered numerous oral presentations at rheumatology and neurophysiology conferences.

Currently, there are only two books in French devoted to ENMG, one of which is P. SEROR's book, Les Conductions Nerveuses, published in 2005. It will soon be published in English with several new chapters in 2025 by Cambridge Scholars Publishing.

PREFACE OF THE PRESENT ENGLISH EDITION IN 2025

It is a privilege to introduce this English translation of *Nerve Conduction Study* by Dr. Paul Seror, a work that embodies decades of experience in clinical neurophysiology. This text is a testament to Dr. Seror's meticulous approach, presenting both foundational principles and advanced techniques while underscoring the essential point that the electrodiagnostic examination is an extension of the clinical history and physical examination.

Whether you are a seasoned neurophysiologist or just beginning your journey in the field, this book offers techniques and insights of immense practical value, many of which may be new even to experienced practitioners. Dr. Seror's methodical breakdown of procedures, paired with clear and detailed illustrations, provides a wealth of guidance to enhance your practice and enrich your clinical toolkit.

The book is particularly notable for its inclusion of methods to study nerves that are less commonly explored, often because of the technical expertise required. By demystifying these challenging areas, he broadens the scope of what is achievable in nerve conduction studies, equipping practitioners to tackle even the most complex cases.

This English edition expands the reach of Dr. Seror's expertise beyond its original French-speaking audience, making it an indispensable resource for neurophysiologists worldwide. More than a manual, *Nerve Conduction Study* is a reflection of Dr. Seror's profound knowledge, passion, and dedication to his discipline. I am confident that this book will be a valuable addition to the library of anyone committed to advancing their expertise in neurophysiology.

Dr James Burge PhD MRCP
Consultant in Clinical Neurophysiology,
Kings College Hospital, London, UK

PREFACE TO THE FIRST FRENCH EDITION IN 2005

The electrodiagnostic examination, commonly known as the electrodiagnosis (EDX), is in fact an extension of the neurological clinical examination, and in particular the neuromuscular examination. The electromyographer or neurophysiologist must be a clinician with all the necessary knowledge to understand neuromuscular pathology. However great their knowledge and expertise, they are not immune to technical considerations. Indeed, the EDX examination requires perfect mastery of nerve and muscle exploration techniques. If knowledge of anatomy and neuromuscular pathology is an essential prerequisite for the examination, it is also necessary to perform the examination in such a way that an interpretation can be made correctly.

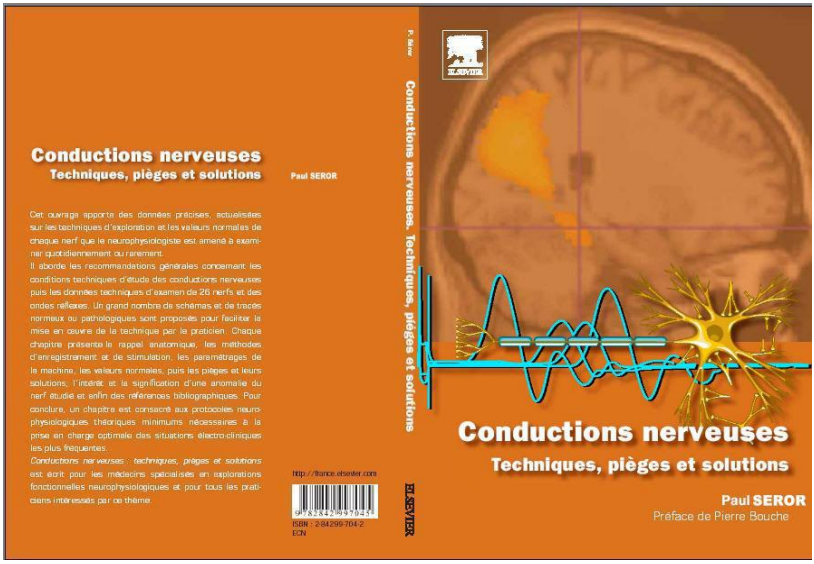
The book written by Paul SEROR responds perfectly to the concerns of the examiner. Paul SEROR transmits here his know-how and his great competence, providing us the diagnostic approach to follow and the best tests to use for the pathology of each nerve. The book begins with a chapter of recommendations that must be read and reread. It is indeed essential to be aware of the pitfalls ingeniously set for the electromyographer and all the parameters and factors that can modify the results of an examination. The main part of the book is then devoted to the techniques, ranging from the simplest to the most sophisticated, proceeding nerve by nerve, and for each nerve, the techniques are given according to the pathologies that may be encountered. In each case, a clear illustration allows the technique described to be clearly visualized. For each technique proposed, the author gives us the values obtained, the limits of the normal, and thus proposes a clear interpretation of the results.

The final chapter deals with the minimum examination necessary for the diagnosis of each electro-clinical situation. This rather new approach seems to be very interesting, as too often textbooks unroll indiscriminately the catalogue of all the technical modalities for exploring a nerve, without giving the logical procedure to follow in order to get to the diagnosis, using the most reliable and sensitive methods.

This wealth of knowledge must not make us forget that the electromyographer is a clinician and that clinical common sense must always be present. We must remain humble and accept that even with the most sophisticated techniques and the most powerful devices, the disease and the patient sometimes escape our technological sagacity.

In the end, the EDX examination only gives us what we bring to it. It is therefore up to the examiner to improve this contribution as much as possible.
This book is primarily intended for EDX practitioners and should be their bedside book to which they will constantly refer; that is all we wish for it.

BOUCHE Pierre MD
Head of the Department of Clinical Neurophysiology at the
Pitié-Salpêtrière Hospital Group.



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ACKNOWLEDGMENTS

To Dr. Pierre BOUCHE
He kindly welcomed me into his department in 1991 and was the first to believe in my book. He graciously wrote the preface to the first edition when it was published in 2005.

To Dr. Jean-Claude WILLER
A specialist in functional explorations of the facial nerve, he improved the corresponding chapter, provided valuable advice on avoiding pitfalls he had encountered, and allowed me to include some of his tracings.

To Dr. James BURGE
A few years ago, he recognised the quality of my work on neuromuscular diseases of the shoulder, as reflected in my articles. He invited me to participate in the King's Neuromuscular Symposium in 2022.

To Dr. Romain GAROFOLI
Before you begin reading this book, I must acknowledge that this book would not have come to fruition without his generous assistance with translation and linguistic adjustments.
Many thanks to all four of you!

And

Very special Thanks to my Wife, who accept to see me going on working during some weekends and vacations, despite I should have more taken part in the house life…Without her patience and encouragements the numerous papers I have published, and this book could not have existed.

INDEX OF ABBREVIATIONS

Figures
S: stimulation
R: bipolar recording
C: cathode monopolar recording
a: anode monopolar recording
thin line: anode
thick line: cathode
R: right
L: left

Text
5O: 5th toe
AAEM: American Association of Electrodiagnostic Medicine
ADM: adductor digiti minimi
AIN: anterior interosseous nerve
ALS: amyotrophic lateral sclerosis
Amp: amplitude
APB. abductor Pollicis Brevis
ASCV: antidromic sensory conduction velocity
ASIS: anterior superior iliac spine
C3: 3rd cervical root
C4: 4th cervical root
C5: 5th cervical root
C6: 6th cervical root
C7: 7th cervical root
C8: 8th cervical root
CIDP: chronic inflammatory demyelinating polyneuropathy
cm: centimetre
CMAP: compound motor action potential
CMT: Charcot-Marie-Tooth disease
PQ: pronator quadratus muscle
CTS: carpal tunnel syndrome
D3: 3rd finger
D5: 5th finger
DC/1 cm: conduction delay for 1 cm
DC/2 cm: conduction delay for 2 cm

DML: distal motor latency
DSL: distal sensory latency
EMG: electromyographic examination
ENMG: electroneuromyographic examination
EDX: electrodiagnostic examination
FDI: 1st dorsal interosseous muscle
FPL: flexor pollicis longus muscle
FDP2: flexor digitorum profundus of the 2nd digit
FP: facial palsy
GBS: Guillain-Barré syndrome
HNPP: hereditary neuropathy with liability to pressure palsy
Hz: hertz (unit of frequency per second)
IPN: interdigital plantar nerve
IT: inching test (or centimetric test)
L2: 2nd lumbar root
L3: 3rd lumbar root
L4: 4th lumbar root
L5: 5th lumbar root
LABCN: lateral antebrachial cutaneous nerve
LLN: lower limit of normal
m/s: meter per second
mA: milliampere
MABC: medial ante- brachial cutaneous nerve
MAP: motor action potential
MCN: musculocutaneous nerve
MCP: metacarpophalangeal joint
MCV: motor conduction velocity
MEP: motor evoked potential
MMN: multifocal motor neuropathy with persistent conduction block
ms: millisecond
ms/D: millisecond per division
mV: millivolt
mV/D: millivolt per division
NAP: nerve action potential
NAPT: neuralgic amyotrophy of Parsonage and Turner
NCS: nerve conduction study
OSCV: orthodromic sensory conduction velocity
PIN: posterior interosseous nerve
PIP: proximal interphalangeal joint
TIL: terminal index latency
S1: 1st sacral root

S2: 2nd sacral root
SD: standard deviation
SCM: sternocleidomastoid muscle
SCV: sensory conduction velocity
SEP: somatosensory evoked potential
SNAP: sensory nerve action potential
T1: 1st thoracic root
TLI: terminal latency index
ULN: upper limit of normal
μV: microvolt
μV/D: microvolt per division

NERVE CONDUCTION STUDIES

Introduction

The aim of this book is to provide the reader, a specialist in electrodiagnostic examination (EDX), with precise and up-to-date data concerning the techniques for studying each nerve, whether it is examined daily for some or rarely for others. The content of the book is simple: 1. One chapter is dedicated to the common basic recommendations regarding the technical conditions for the Nerve Conduction Study (NCS). 2. Thirty chapters are dedicated to the techniques of NCS for 31 different nerves. 3. Five chapters expose methods for studying some nerve functions: the reflex waves, the proximal NCS, the autonomic nervous system, the neuromuscular junction, and the continuous activity syndrome. 4. Finally, a chapter is devoted to the theoretical minimum neurophysiological protocols necessary for the optimal EDX management of the most common electro-clinical conditions. Then, the clinical significance of anomalies detected is proposed, and finally a few bibliographic references are provided.

General Basic Recommendations

The study of motor and sensory nerve conductions is a central element of the electroneuromyographic (ENMG) or formerly electromyographic (EMG) examination. It is usually carried out before the detection examination and must be done under the same technical conditions for all nerves.

The examination must be carried out in a well-heated room, because the patients will be partially undressed, or even almost completely undressed, when all 4 limbs must be examined. The skin temperature of the examined area must be at least 32°C on the upper limb and 30°C on the lower limb. Indeed, the cooling of the nerves causes a slowdown in the motor (MCV) and sensory (SCV) conduction velocities; this slowdown is proportional to the cooling and may result in a conduction block (experimental in animals). Certainly, there are correction coefficients depending on cooling, but these coefficients vary from one author to another, reflecting the

heterogeneity of the nerve's response to cold in 2 different individuals. It is therefore necessary to be able to warm the extremities of patients who require it and not to use these correction coefficients whenever possible. This phenomenon is particularly crucial for the hands, to the extent that in city practice, 30 to 45% of examinations are carried out looking for carpal tunnel syndrome and that a slowdown in conduction speeds linked to cold can cause a wrong diagnosis of median nerve lesion at the wrist. Finally, how can we compare 2 examinations done 6 months apart, if the first is done in summer with hot hands and the second in winter with cold hands?...

Machine Settings

For the motor conduction study, the parameters are:
High filter: 10.000 Hz; low filter: 2 Hz.
The analysis window will be 3 ms/division (ms/D) for the upper limbs and 5 ms/division for the lower limbs. For the F waves or the H reflex, a window of 10 or 20 ms/division is used. In some demyelinating neuropathies, it is necessary to widen the analysis window to avoid missing very late F waves, which is why I prefer 20 m/division in routine.
The input amplitude (or gain) should be 5 mV/division for MCV and 0.5 or 0.2 mV for F waves.
The duration of the shock is routinely 0.2 ms; it can be increased to 0.5 or even 1 ms when the depolarisation of the nerve is difficult to obtain for anatomical reasons (Erb's point, inguinal fold), or because of oedema or obesity.
The supramaximal stimulation intensity can vary from 15 to 100 mA.

For the sensory conduction study, the parameters are:
High filter: 2.000 Hz; **Low filter**: 10 Hz.
The analysis window is usually 1 ms/D for recording the routine sensory nerve action potential (SNAP) of the median, ulnar, and radial nerves at the wrist, or the sural and peroneal nerves at the ankle. To record more proximal SNAP potentials, at the elbow, the axillary fossa, or Erb's point, a window of 2 to 5 ms/D will be used. On the contrary, over a short distance, the window can be reduced to 0.5 ms/D to obtain a greater precision in the measurement of latency, which can then be measured from 0.02 ms to 0.02 ms.
The input amplitude (or gain) is usually 20 μV to minimise artefact rejection.
Stimulus duration is routinely 0.2 ms or 0.5 ms.

The supramaximal stimulation intensity at the finger of the hand is usually achieved at 15 to 20 mA.

Measurements

Figure 1. With an analysis window of 0.5ms / division we can clearly differentiate the latency from a SNAP measured at the peak within 0.02 ms. Indeed, we can settle 4 latency sliders between 2 sliders spaced 0.1 ms.

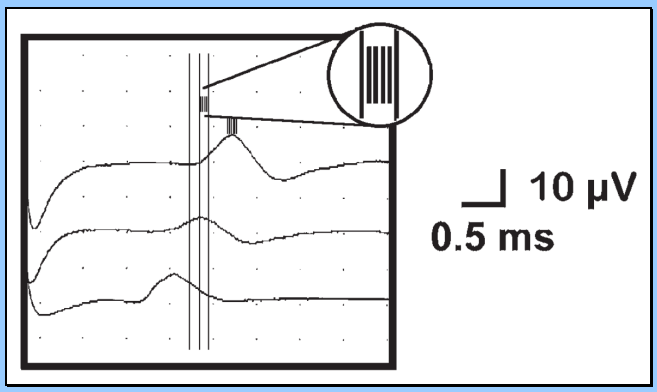
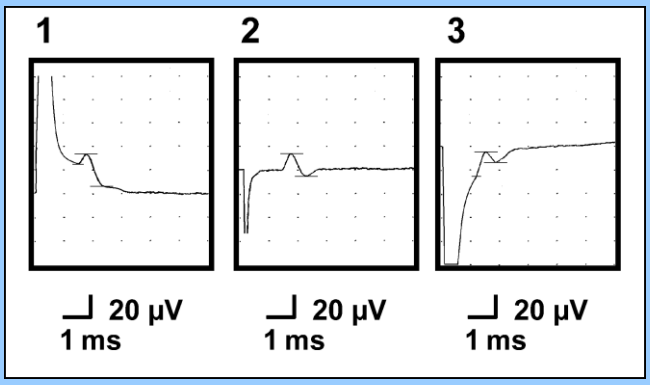


Figure 2. According to the shape of the stimulus artifact, the SNAP amplitude of 20 µV without artifact (2), will be measured at 29 µV (1) or at 10 µV (3)



The distance measurement is done with a dressmaker's tape measure. To minimise measurement errors, it is advisable to stimulate either on anatomical landmarks that cannot move or to mark the stimulation site with a ballpoint pen before rather than after the stimulation.

The latency measurement is done on the screen either automatically or manually. For sensory conduction, it is almost always necessary to refine the measurement by hand, even in automatic measurement mode. For motor conduction, the measurement is always done at the beginning of the negative wave of the compound motor action potential (CMAP) or at the onset of the first wave for CMAP. For sensory conduction, the latency measurement is better done at the onset of the negative wave of the sensory nerve action potential (SNAP), especially when velocity between the stimulating point and the recording point is calculated (ex SCV from digit to wrist), which represents > 90% of SCV. In this situation, using measurement of latency at the peak increases the conduction time and decreases the velocity, which requires the use of different normative data. Sometimes a measurement at the peak of the negative wave Is preferable and simpler, without significantly changing the SCV result. This is the case when the conduction velocity is studied between 2 points, such as for the ulnar nerve velocity at the elbow (Fig 4.7), or when the latencies of 2 nerves are compared over the same distance, such as the median to ulnar nerve latency difference of the 4th digit (Fig 2.6 & 2.7) or between the wrist and above the elbow (Fig 4.6).

The sensitivity of the measurement depends on the analysis window. On current machines, the measurement is usually made from 0.1 ms to 0.1 ms. If this sensitivity is sufficient for the measurement of motor latencies and is even fine for the measurement of late F or H wave latencies, it is quite insufficient for some sensory special tests. Indeed, there is no clinical relevance to being able to distinguish a latency of 52 ms from a latency of 52.2 ms. On the other hand, the pathological thresholds are 0.40 ms for the measurement of median to ulnar latency difference of the 4th digit (see page 30) and vary from 0.45 ms to 0.69 ms for the inching test (page 32). Thus, if the upper limit of normal is 0.40 ms, it seems important to be able to differentiate a value of 0.36 ms (normal) from a value of 0.44 ms (pathological). This is only possible if the measurement can be made from 0.04 ms to 0.04 ms or from 0.02 ms to 0.02 ms (Figure 1).

The amplitude measurement is done on the screen either automatically or manually. For the sensory conduction, it is almost always necessary to refine the measurement by hand. For motor conduction, the measurement

is always done from baseline to the peak of the negative wave. For sensory conduction, the measurement is always done from peak to peak (between the peak of the negative wave and the peak of the following positive wave). The ratio of maximum/minimum right/left amplitude is the best criterion to detect unilateral lesions, and usually, a max/mini ratio > 2 or 2.5 is considered abnormal. When the stimulation artefact is descending or ascending (Figure 2) and includes the sensory potential, the measure of the peak-to-peak amplitude is incorrect and will be increased or decreased. Then, everything possible must be done to avoid this situation by changing the orientation of the stimulation electrode.

The potential duration measurement is done on the screen, either automatically or manually. The easiest and most reproducible measurement is that of the negative wave: the duration corresponds to the time between the wave onset from the moment it first crosses (dropping) the baseline. If the potential is desynchronised, and the potential crosses the baseline several times, we will take as a reference the last time when it crosses the baseline while coming down. For sensory conduction, it is often necessary to refine the measurement by hand.

The potential area is calculated automatically apart from the amplitude and duration of the potential.

The motor conduction velocity is calculated from the recording of 2 CMAPs obtained after stimulation at 2 different points along the path of the studied nerve. The ratio between the distance (D) in millimetres between these 2 points and the time (T) in milliseconds between the 2 latencies gives the MCV in m/s $\mathbf{MCV=}\frac{\text{D (ms)}}{\text{T (mm}} = \text{m/s}$. MCV cannot be calculated from the recording of a single CMAP, even though the distal motor latency (DML) reflects the MCV. Indeed, DML of a CMAP is composite data including: 1- a nerve conduction time, 2- the chemical time of the neuromuscular junction, and 3- finally a time of propagation of the depolarisation along the muscle fibres. The muscle fibres act as an amplifier of the motor nerve response, and the amplitude of the CMAP is in the mV range, whereas the amplitude of the mixed or sensory potentials is 100 or 1000 times lower (in the µV range).

The sensory conduction velocity (Table 1) is calculated in most cases from the recording of one single sensory nerve action potential (SNAP) obtained after stimulation at one single point. Indeed, the stimulation directly depolarises the sensory fibres, and the recorded potential is that of

the sensory fibres themselves. There is no amplifier, and the amplitude of the SNAP is in the µV range. The ratio between the distance (D in millimetres) between the cathode of the stimulator and the cathode of the recording electrode and the time (T) or latency in milliseconds gives the SCV in m/s $\mathbf{SCV} = \frac{D\ (ms)}{T\ (mm} = m/s$. If 2 SNAPs are obtained at 2 different points along the nerve (below and above the elbow, Fig. 8. Chap 4) after a single point stimulation (5th digit), we can calculate the SCV between these 2 different recording points. As for the MCV, the ratio between the distance in millimetres that separates these 2 points (D) and the time in milliseconds that separates these 2 latencies (T) gives the SCV. Measurement at the peak is also recommended for calculating the latency difference for the median and ulnar nerves between 4th digit and the wrist (Fig. 7-8 chap 2) and from the wrist to above the elbow (Fig.7. chap 4).

SCV can be studied in the direction of physiological nerve conduction (orthodromic) or the opposite direction (antidromic).

Specificity of the Nerve Conduction Study

Motor Conduction (Figure 3)

The specificity of the NCS, for a nerve, is ensured either by the specificity of the stimulation or by the specificity of the recording. If at the wrist and elbow it is reasonable to think that the median nerve, the radial nerve, or the ulnar nerve can be stimulated in isolation and that the recording is therefore specific of the nerve being studied, one must always bear in mind that as soon as stimulation is applied at the axilla or at Erb's point, this specificity disappears, as the stimulation will always depolarise several nerves at the same time. In this case, the specificity of the conduction study of a single nerve is only guaranteed thanks to the specificity of the target muscle recording.

To ensure the specificity of the motor conduction study, the following two simple rules must be respected. **The first rule** is that if the stimulation is recognised as being specific to a single nerve, the recording can and should be made using a pair of disposable, repositionable, self-adhesive electrodes. **The second rule** is that if the stimulation cannot be considered to be specific of a single nerve, the recording should be achieved with a concentric needle electrode.

Table 1: Mean normal values and lower limits of normal nerve conduction velocities

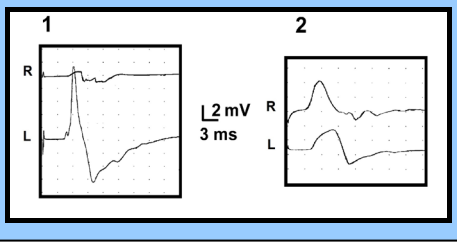
	Motor velocity		**Sensory velocity**	
	Normal	LLN	Normal	LLN
Upper limb	59 m/s	50 m/s	60 m/s	45 m/s
Lower limb	50 m/s	40 m/s	50 m/s	40 m/s

Table 2: Mean normal values and lower limits of normal of CMAP and SNAP amplitudes.

	CMAP		SNAP	
	Normal	LLN	Normal	LLN
Median	10,5 mV	5mV	36µV	12µV
Ulnar	10,5 mV	5mV	18µV	7µV
Peroneal	7 mV	2mV		
Tibial	13 mV	5mV		
Sural			< 65 years = 29µV * > 65 years = 13µV *	20 to 10µV * 5µV *

LLN: lower limits of normal; *: Horowitz 1992

Figure 3. The **specificity** of the coaxial needle recording (1), vs a pair of surface recording (2) is well demonstrated in this case of neuralgic amyotrophy with a right anterior interosseous nerve palsy. The recording in the pronator quadratus after stimulation at the elbow, demonstrates a 90% axonal loss with needle recording, whereas recording with surface electrodes (2) shows no difference between both sides [Seror 2017].



This is particularly true for the anterior interosseous (Figure 3) (Seror 2017), suprascapular, and long thoracic nerves, but also the radial nerve. Indeed, all monopolar recordings (surface or needle) always correspond to a composite of the several potentials of different muscles innervated by one or several nerves.

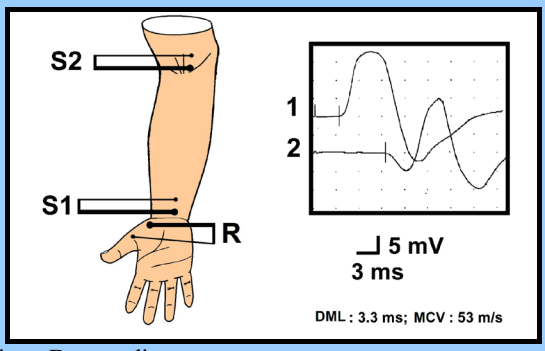
Even if the monopolar needle is isolated, except for the distal 2 mm, the so-called neutral reference electrode is never really neutral and always records the electrical activities of the closest muscles. Thus only

The coaxial needle whose active and reference electrodes are in the studied target muscle can claim to record only the electrical activity of this muscle, excluding any other.

Co-stimulation. Any (slightly excessive) nerve stimulation may result in co-stimulation of an adjacent nerve, even when the 2 nerves appear to be sufficiently separated to be stimulated in isolation. If there is no response to stimulation of a particular nerve, the physician will increase the stimulation to try to obtain a response. If this increase is too great, the adjacent nerve may be activated, and a response may be recorded that does not match that of the nerve being studied. This is especially true in overweight subjects where the anatomical landmarks are masked and the stimulations are of high intensity. This can occur for the median and ulnar nerves at the wrist as at the elbow, but also for the peroneal (PN) and tibial (TN) nerves at the popliteal fossa. These co-stimulations are constant for all proximal stimulations in the upper limb (at Erb's point) and in the lower limb (sciatic trunk above the knee).

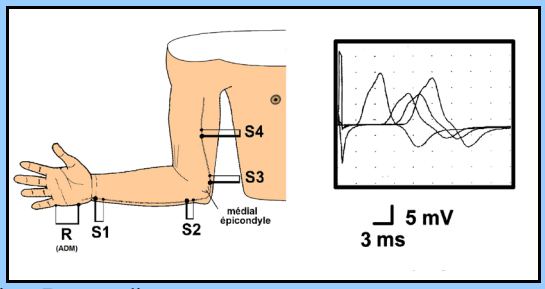
Nerve anastomosis. They are particularly frequent between the median and ulnar nerves in the forearm. They are less common for the peroneal nerve. The best-known median-cubital anastomosis is the one described by Martin and Gruber in 1763. In this case, ulnar fibres are found in the median nerve at the elbow and in the ulnar nerve at the wrist, so they join the ulnar nerve during their forearm course. Therefore, when we stimulate the median nerve at the elbow, we also stimulate ulnar axons or nerve fibres. It is therefore a particular form of nerve co-stimulation, but not related to a technical error. For the median nerve, after stimulation at the elbow, we note the presence of a positive wave before the usual negative wave of the CMAP (Figure 4). This positive wave prevents the calculation of an accurate nerve conduction velocity from the median nerve to the forearm. For the ulnar nerve, the consequence is more important, as it is often responsible for an erroneous diagnosis of conduction block at the elbow (Figure 5). Indeed, the ulnar fibres that are in the median nerve at the elbow are not stimulated, and the CMAP is of lower amplitude than after stimulation at the wrist, where all nerve fibres are present. This abnormality is more common for nerve fibres that supply the 1st dorsal

Figure 4. The pattern of the « normal » conduction of the median nerve when there is a Martin-Gruber median-ulnar anastomosis. The positive wave onset at elbow is due to the stimulation of ulnar axons that are present in the median nerve at elbow and in the ulnar nerve at wrist.



S: stimulation **; R:** recording

Figure 5. The pattern of the ulnar nerve conduction when there is a Martin-Gruber median-ulnar anastomosis. It looks like a conduction block located in the forearm below the elbow. The CMAP recovers a normal amplitude when a proximal stimulation is performed as both median and ulnar nerves are stimulated, which recruits all axons.



S: stimulation**; R:** recording

that supply the 1st dorsal hypothenar muscles (<5%). Without using double shock techniques, the diagnosis is easily confirmed by the recovery of the normal CMAP amplitude of the ulnar nerve after stimulation at the arm or axilla that recruits all median and ulnar axons.

Insufficient stimulation. This is an almost daily problem, and it is always necessary to think first about dry felts of the stimulation electrodes that

deliver only mild stimulus or no stimulus. This can lead to diagnostic errors such as false conduction block or axonal sensory neuropathy. When the electrodes are dry, insufficient stimulation is a source of diagnostic errors, especially when stimulation is done at Erb's point for the inferior brachial plexus or at the inguinal and superior gluteal folds for the femoral nerve and the sciatic trunk, respectively. In these cases where the nerve is deep and difficult to stimulate, a monopolar stimulation must be used systematically before concluding the nerve conduction is abnormal.
Excessive stimulation. It is the main cause of co-stimulation.

Sensory Conduction

Insufficient stimulation. Finding a low amplitude SNAP may be due to a device or patient problem. Apart from exceptional machine or cable failure, the most common cause is the drying of electrode felts. When the problem is related to the patient, the delivered impulse is correct, but the nerve fibres are not stimulated because of lower limb oedema, overweight, skin trophic disorders, or an abnormal skin impedance related to the application of a fatty cream. In the lower limbs, before diagnosing an axonal sensory neuropathy, it is essential to perform a SNAP recording with a pair of subcutaneous needle electrodes. In the upper limbs, where orthodromic conduction is frequently used, it is interesting to perform these same tests with the antidromic method, as it is not rare to find normal data.

Excessive stimulation. Too much stimulation can modify the latency of the recorded potential and can therefore change its value from pathological to normal. This problem exists only for short latencies (< 4 ms) and is crucial for the latency differences used in the hand, whose limits of normal are 0.30 to 0.40 ms. This problem exists only for tests performed in antidromic (4^{th} digit, palm-wrist, thumb-wrist, and inching tests). Indeed, the latency of a SNAP obtained by just submaximal stimulation and just supramaximal stimulation can vary by 0.30 ms (see chapter median nerve, Figure 8). This phenomenon of "electrical jump" is linked to the fact that the depolarisation of the nerve takes place 1 or 2 cm from the cathode; in fact, the electrical impulse, which is too strong, is transmitted by the tissues more rapidly than by the axon itself. With the orthodromic method, the stimulation intensity is low and is the same for all fingers as well as for all collateral nerves of the median, ulnar or radial nerves studied, which allows a more precise comparison of latency differences.

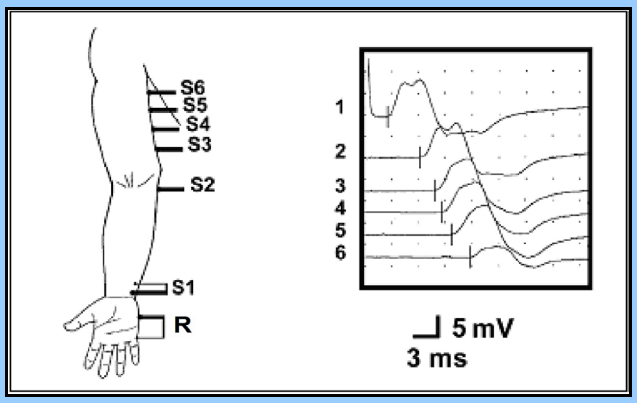
Sometimes, the specificity of the recorded SNAP is problematic. The study of the conduction of the palmar cutaneous branch at the wrist (median nerve) and the study of the calcaneal nerve (posterior tibial nerve) are the best examples. In both cases a specific recording is impossible in the normal subject. In the first case, it is impossible to stimulate the palmar branch without stimulating the median nerve at the same time; thus the SNAP recorded is the sum of both SNAP (palmar nerve + collateral nerve of the thumb). Consequently, this palmar branch can only be specifically studied in severe CTS patients, with no recordable thumb collateral nerve SNAP, as it is not entrapped in carpal tunnel . In the second case, the potential of the "calcaneal nerve" is in fact only the potential of the posterior tibial nerve recorded far (4 cm) from the optimal recording position; in fact its amplitude is low, and a selective anaesthesia of the calcaneal nerve does not make it disappear (cf. chapter posterior tibial nerve) (Seror 1995).

Finally, on a more daily basis, it is important to know that the orthodromic SNAP of the 5th digit will be recorded without difficulty by a bar electrode positioned on the median nerve; the same will be true for the SNAP of the 3rd digit by a bar electrode positioned on the ulnar nerve. In both cases, the potential will have a normal latency and a low amplitude (about 30% of the normal SNAP). This explains why it is necessary to use a low intensity of stimulation to study separately the 2 collateral nerves of the 4th finger. In the same way, the precise study of the lesions of the collateral nerves of the fingers (digital wound) can be carried out exclusively by the orthodromic method and stimulations of very low intensity (4 to 8 mAmp).

Conduction Block (Figure 6)

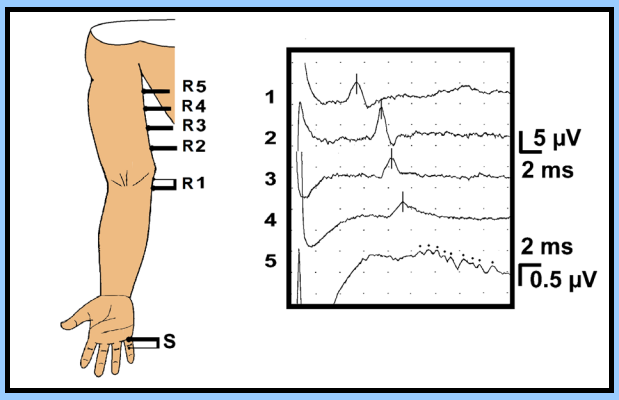
It is defined as the loss of the nerve impulse transmission function of large-diameter myelinated fibres while the axon is not altered. Neurophysiologically, conduction block is expressed by a significant loss of amplitude and/or area of CMAP or SNAP between 2 closely spaced stimulation or recording points. Stimulation below the conduction block recruits all available axons, while only a small part of the axons stimulated above the block transmits nerve impulses until the recording point.

Figure 6. Motor conduction block of the ulnar nerve in the arm during a Lewis and Sumner syndrome. Thanks to multiple stimulations every 5cm we can specify that the block is located between 15 and 20cm above the medial epicondyle.



S: stimulation**; R:** recording

Figure 7. Sensory conduction block in the same patient results in SNAP desynchronization.



S: stimulation**; R:** Recording.

According to the AAEM consensus, a block is "defined" if the loss of amplitude is greater than 50% for the median nerve, ulnar nerve, and SPE nerve at the fibula head. For the SPE and SPI at the leg, amplitude loss must be greater than 60%, and the duration of the decreased-amplitude

CMAP must not be increased by more than 30% than the normal potential. Block is "probable" if these percentages are 40% and 50% respectively.

Finally, sometimes conduction block cannot be demonstrated by studying nerve conduction because it is impossible to stimulate the nerve above the injured area. In these cases, when stimulation is possible only below the injured area, indirect signs of motor conduction block must be known. These are: a normal amplitude of the CMAP/MAP that contrasts with an important clinical motor weakness, without amyotrophy and with a very reduced interference pattern or single potential, with a high firing rate (Seror 2002).

Desynchronization

Desynchronization (or temporal dispersion) of a potential is a degraded kind of conduction block. The damage, in terms of slowing of nerve conduction velocity and block, instead of being identical on all axons, is heterogeneous. Thus, the recorded potentials arrive shifted with respect to each other, and the CMAP or SNAP takes on a desynchronized appearance with an important increase in potential duration (Figure 7). Conduction block and desynchronization have the same meaning of focal myelin lesion, whether the cause is mechanical (pure compression or with pressure hypersensitivity) or related to an inflammatory acquired demyelinating neuropathy: acute Guillain-Barré syndrome (GBS) or chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), multifocal motor neuropathy (MMN), or Lewis Sumner syndrome. A conduction block, which by definition respects the continuity of the axons, allows a rapid functional recovery if the cause of the block is reversible (acute compression, xylocaine block...). On the contrary, chronic blocks (chronic compression and demyelinating disease with persistent blocks) are always at the origin of a progressive axonal loss with the evolution of the disease.

Sensory conduction blocks. If the vast majority of cases of conduction blocks are demonstrated by the study of motor conduction, there are also always blocks on myelinated sensory axons, but they are rarely looked for. Sensory blocks exist in CIDP and Guillain-Barré syndromes but are never looked for because motor blocks are sufficient to make the diagnosis. In the rare cases of purely sensory chronic inflammatory demyelinating polyneuropathy (CIDP), the study of SEP (somatosensory evoked potentials) allows us to evoke their existence in an indirect way (Ayrignac 2013). In some CTS, the proportion of conduction blocks on the sensory

fibres is major, and their demonstration (see chapter on median nerve, p. 36) makes it possible to consider as moderate a CTS judged to be severe and then to avoid surgery in favour of a simple infiltration. The most demonstrative cases (80 to 90% block) are encountered during CTS related to pregnancy and postpartum (Seror 1998) or during intensive DIY sessions in an untrained subject.

Demyelination

It has a precise neurophysiological definition. Excluding Guillain-Barré syndrome, the "ad hoc committee" considers that a chronic or subacute neuropathy is (inflammatory and) demyelinating if at **least one of the 7 modified EFNS/PNS criteria** (Joint Task Force 2010) is fulfilled.

Criteria of EFNS / PNS (Joint Task Force 2010) for CIDP
Diagnosis is positive if at least one criteria is present

Criteria	Threshold	Special conditions
1- DML	> 150% ULN	in 2 nerves CTS excluded
2- MCV	< 70% LLN	in 2 nerves
3 & 4- F wave latency	> 130% ULN > 150% ULN or absent in 2 nerves	in 2 nerves if CMAP amplitude <80% LLN if CMAP amplitude >20% LLN
5- Conduction block	Amplitude drop > 50%	in 2 nerves or 1 nerve + another criteria if CMAP amplitude >20% LLN
6- Temporal dispersion	Duration negative peak >30%	> 2 nerves
7- Distal CMAP duration	> 6.7ms median/ulnar > 7,6ms peroneal > 8.8ms tibial	≥ 1 nerve + another criteria

Conduction block had to be located outside the classical sites of nerve entrapment for at least one nerve. **CTS**: carpal tunnel syndrome
LLN: lower limit of normal; **ULN**: upper limit of normal

The value of the lower (LLN) and upper limits of normal (ULN) data are summarised in one Table for each nerve.