Waveform changes due to conduction block and their underlying mechanism in spinal somatosensory evoked potential: a computer simulation

Technical note

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Based on a square-wave solid-angle analysis, a simplified mathematical model was produced for computing a sequence of potential change in a volume conductor generated by an impulse traveling along a nerve fiber. A conduction block was simulated as a phenomenon in which a depolarization wavefront stops traveling when it reaches a certain point, although the following repolarization wavefront continues to travel until it reaches the same point. The spinal somatosensory evoked potential (SSEP) was produced as an algebraic sum of simulated nerve fiber action potentials (NFAPs).

With a conduction block, an NFAP that was normally triphasic showed a positive–negative diphasic wave with reduced negativity at the point of the block, diphasic waves with enhanced negativity at points immediately preceding the block, and initial-positive waves alone or abolition of any wave at points beyond the block. The absence of their terminal-positive phases paradoxically enhanced the negative peak of the spinal SSEPs in a partial block that involved only the constituent fastest fibers, because phase cancellation of the phases between the terminal-positive phases of the fastest fibers and the negative phases of the slower fibers, which normally happens, failed to occur. At the points immediately preceding the block, the identical mechanism sustained the spinal SSEP enhancement even when every fiber was included in the block. The computer model predicted that localization of the precise site of conduction block can be achieved by demonstrating an abrupt reduction in the amplitude of the spinal SSEP, which is accompanied by an increased negative wave caudally and an enhanced monophasic positive wave rostrally.

Key Words • spinal somatosensory evoked potential • amplitude • conduction block • computer simulation • phase cancellation

Intraoperative spinal cord monitoring is now widely used for diagnostic purposes and for surgical monitoring. The placement of recording electrodes in various structures around the spinal cord itself, such as the subdural or epidural space, the ligamentum flavum, or the intervertebral disc, yields a better-defined spinal somatosensory evoked potential (SSEP) with greater electrical stability than is obtained by less invasive recording techniques. It may safely be stated that a conduction block is ranked first among the physiological consequences of spinal cord compression that may be revealed by the test, because this type of abnormality is known to be one of the most important causes of clinical weakness and sensory loss. Hence, a better understanding of the waveform changes caused by a conduction block would maximize the diagnostic value of spinal SSEP monitoring.

Figure 1 shows spinal SSEPs recorded unipolarly from the ligamentum flavum at multiple levels after epidural stimulation of the cauda equina in a patient with cervical spondylotic myelopathy. In our experience, this combination of an abrupt reduction in amplitude of the negative peak (Fig. 1 downward oblique arrow) at one level, augmentation of the negative peak (Fig. 1 upward vertical arrow) in the lead closely caudal to that level, and monophasic positive waves at more rostral levels seems to be a typical pattern of waveform changes, representing the phenomena probably related to focal conduction block. In this study, we analyzed the mechanism underlying such alterations in waveform with the help of a simplified mathematical model. Attention was also directed to the possible paradoxical increase of amplitude in a partial block, which seemed to be another aspect of the same underlying mechanism.

Methodology

Square-Wave Solid-Angle Analysis

Based on quasistatic assumptions, the validity of which is commonly accepted, we treated the electrical potential generated in a volume conductor by a propagat-
ing nerve impulse in the following manner (Fig. 2). A nerve fiber carrying an impulse can be divided into two regions: active and inactive. In the inactive region, the axon membrane can be viewed as dipole layers with positive charges on the outer surface and negative charges on the inner surface, whereas in the active region, the dipole layers have reversed polarity, with negative charges on the outside surface. Approximating the intracellular action potential to the square wave, the transition between the two regions is assumed to occur instantaneously. The assumption of instantaneous repolarization obviously ignores the refractory period of nerve fibers. The refractory characteristics of the excitable membrane must be taken into account in estimating the potential field caused by the second impulse generated at a very short interval after the first, although this is not the case in this study. Supposing that each region has a different but constant surface-dipole-moment density and that a nerve fiber is surrounded by a uniform medium that extends infinitely, the potential at point P, which is caused by an idealized impulse, depends on the solid angles $\Omega_d$ and $\Omega_r$ subtended by the boundaries between the active and inactive regions. The potential is consequently obtained by adding the contribution of each wavefront:

$$
\phi = |D\Sigma| \left\{ \frac{\Omega_d + (-\Omega_r)}{4\pi\epsilon} \right\},
$$

where $\epsilon$ represents the permittivity of the surrounding medium and $|D\Sigma|$ indicates the sum of $|DA|$ and $|DI|$, which are absolute values of the surface-dipole-moment density of active and inactive axon membranes, respectively. The minus sign is added so that the plus or minus sign of each solid angle coincides with the sign of each wavefront when it is "seen" from point P. The reference electrode is assumed to be so far distant from the source of the activity to be recorded that its potential is negligible.

**Generation and Propagation of a Nerve Impulse**

In a myelinated fiber, depolarization of the axon membrane at one node, which is caused by an externally applied stimulus with suprathreshold intensity, gives rise to the local current, which depolarizes the adjacent nodes on either side, transmitting the impulse in both directions. However, we assumed that the impulse travels continuously along each axon with its constant conduction velocity (“v”). Deviations from this assumption caused by the real saltatory character of nerve conduction can be neglected when the observation distance is not too small. Supposing the situation in which a nerve fiber is placed on the x-axis of a rectangular coordinate system (X, Y, Z), we have only to work in the x–y plane because the potential produced by a nerve impulse is symmetrical around the x-axis (Fig. 2). The depolarization was assumed to be initiated at the origin of the coordinates. Unless the detecting point P is in the vicinity of the origin, the impulse traveling in the negative direction provides a negligible contribution to the potential at P. In the following discussion, therefore, we will focus on the impulse traveling in the positive direction alone. Once the membrane depolarizes, it takes a certain time (“τ”) to repolarize. During the time τ, the depolarization wavefront of the idealized impulse will travel the distance $\tau v$, which is denoted by “λ.” It is, therefore, not until $vt$ exceeds “λ” that the repolarization wavefront appears, where “t” represents the elapse of time.

Hence, if $0 \leq vt \leq \lambda$, 

$$
\phi = |D\Sigma| \Omega_d / 4\pi\epsilon \tag{1}
$$

If $vt > \lambda$, 

$$
\phi = |D\Sigma| \left\{ \Omega_d + (-\Omega_r) \right\} / 4\pi\epsilon \tag{2}
$$

The locations of a pair of wavefronts traveling in the positive direction are specified by $(vt, 0)$ and $(vt - \lambda, 0)$, respectively, at a given instant, whereas the location of point P is specified by $(Px, Py)$, where $Px, Py > 0$. Using these notations, $\Omega_d$ and $\Omega_r$ are expressed in accordance with the definition of a solid angle (Fig. 3) as:

$$
\Omega_d = A (Px - vt) / \{(Px - vt)^2 + Py^2\}^{1/2}, \quad (3)
$$

$$
\Omega_r = A (Px - vt + \lambda) / \{(Px - vt + \lambda)^2 + Py^2\}^{1/2}, \quad (4)
$$

where “A” is wavefront area.

Equations 1 and 2, therefore, can be expressions for computing a sequence of potential change as a function of
Using the notations of Fig. 2, Equations 1, 2, 3, and 4 hold in time “t” due to an impulse traveling in a positive direction along a nerve fiber, just like an actual situation of an action potential recording with the detecting point fixed.

Simulation of a Conduction Block

A conduction block (Fig. 4) is considered to be a failure of local current initiated by depolarization at one node to activate the next node. It can therefore be viewed as the phenomenon in which a depolarization wavefront stops traveling when it reaches a certain point B (Bx, 0), although the following repolarization wavefront keeps on traveling until it reaches the same point. Supposing Bx > λ, a simulation of a conduction block will be attained as follows: if 0 ≤ vt < Bx, Equations 1, 2, 3, and 4 hold in calculating “φ”. If Bx ≤ vt ≤ Bx + λ, Equations 2 and 4 hold on condition that Ωd = A(Px - Bx) / ((Px - Bx)^2 + Py^2)^1/2 because the depolarization wavefront of the impulse is fixed at point B. If vt > Bx + λ, φ = 0 because both of the wavefronts should be extinguished.

Determination of Parameters

We dealt with |ΔΩ|/4πε in Equations 1 and 2 as a constant, because we assumed not only that the volume conductor was uniform, but also that the membrane properties were the same for nerve fibers of different diameter. “Px” and “Py” were determined by the location of point P, whereas “A,” “v,” and “λ” were defined by nerve fiber diameter “d.” The cross-section area “A” of a nerve fiber is given by A [m^2] = π (d/2 [μm])^2 × 10^-12.

On the basis on Hursh’s observation relating conduction velocity to fiber diameter, we have chosen 6.0 as the conversion factor that is the ratio of conduction velocity in meters per second to total fiber diameter in μm: v [m/sec] = d[μm] × 6.0 [m/sec × μm]. (5)

As mentioned previously, “A,” means the spatial extent of depolarization along the nerve fiber, and it can be calculated7, as the product of the nerve action potential duration “r” multiplied by the conduction velocity “v”:

λ[m] = r[sec] × v[m/sec]. (6)

As Paintal29 showed that “r” is inversely proportional to “v,” we computed the regression equation for Paintal’s experimental data obtained in three cats from saphenous fibers at a temperature of 37.1˚C:

r × 10^3 [msec] = 3.05 / v [m/sec] + 0.32 (r = 0.734, p = 0.0001). (7)

Equations 5, 6, and 7 yield:

λ[m] = (1.92 × d[μm] + 3.05) × 10^-3. (8)

Computer Techniques and Supplies

The program for computer simulation was written in Quick Basic Version 4.5 (Microsoft Corp., Redmond, WA) running under MS-DOS Version 3.3C on a PC 9801 (NEC Corp., Tokyo, Japan). In the process of digital-to-analog conversion for displaying calculated values on a CRT screen, a time resolution of 0.05 msec was used. A negative potential calculated was displayed as an upward deflection.

Simulation of the SSEP

Because each charge contributes independently to the potential at a point from the superposition principle,14 the compound nerve action potential (CNAP) can be conceived of as a linear summation of each constituent nerve fiber action potential (NFAP).39 For simplicity, a simulation of the SSEP was conducted on the assumption that it was derived only from the dorsal spinocerebellar tract.15,18,44 The contributing fiber diameter distribution in the model was based on the diameter histogram of the tract in a human.15 The smoothing procedure was applied to the histogram with 17 classes using cubic spline inter-
The effect of graded spinal cord compression was simulated by blocking the conduction in the largest fiber first, followed by a conduction block in progressively smaller fibers at a Px of 119 mm because pressure is known to exert its greatest effect on the largest fiber. As an increasing number of large fibers were involved in conduction block, the negative peak of the spinal SSEP did not decrease, but increased gradually at the point of the block and at any point beyond it. In addition, the same extent of conduction block also increased the negative peak at the points preceding the block. Such augmentation of the spinal SSEP is shown in Fig. 7 right, in which every fiber of 12 μm or more in diameter, classified as Group I, which corresponds to 15% of the whole population, is involved in the conduction block. Subsequent courses of the increased amplitude, however, are quite different, depending on the detecting point (Figs. 7 right and 8). At the point of the block and every point beyond there, the negative peak gradually declined toward the control level and then fell below it as an increasing number of the Group II fibers were included in the conduction block. Although the progressive decline of the negative peak reached a minimum value of 50% of the control at the point of the block, the peak was completely positive, although these changes were less conspicuous with increasing distance from the block.

Spinal Somatosensory Evoked Potential

Basically, configuration of the spinal SSEP inherited a positive-negative-positive triphasic waveform from the constituent NFAP. A simulation of serial recording of spinal SSEP along the length of the spinal cord was conducted with the same setting as Fig. 7 left. Without the conduction block, the result clearly demonstrated physiological temporal dispersion, which means that with increasing distance of impulse propagation, the recorded potentials become smaller in amplitude and longer in duration (Fig. 7 right). The effect of graded spinal cord compression was simulated by blocking the conduction in the largest fiber first, followed by a conduction block in progressively smaller fibers at a Px of 119 mm because pressure is known to exert its greatest effect on the largest fiber. As an increasing number of large fibers were involved in conduction block, the negative peak of the spinal SSEP did not decrease, but increased gradually at the point of the block and at any point beyond it. In addition, the same extent of conduction block also increased the negative peak at the points preceding the block. Such augmentation of the spinal SSEP is shown in Fig. 7 right, in which every fiber of 12 μm or more in diameter, classified as Group I, which corresponds to 15% of the whole population, is involved in the conduction block. Subsequent courses of the increased amplitude, however, are quite different, depending on the detecting point (Figs. 7 right and 8). At the point of the block and every point beyond there, the negative peak gradually declined toward the control level and then fell below it as an increasing number of the Group II fibers were included in the conduction block. Although the progressive decline of the negative peak reached a minimum value of 50% of the control at the point of the block, the peak was completely positive, although these changes were less conspicuous with increasing distance from the block.

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abolished and even replaced by a positive deflection at points beyond the block. In contrast, the increased negative peaks at the points preceding the block still kept increasing and then reached maximal values, which were sustained even when every fiber of Group II was included in the block. Furthermore, at the points preceding the block, the extensive block of Group II fibers produced the configuration change in the spinal SSEPs, characterized by poorly developed or even absent terminal positivity with a prolonged descending phase of the negative deflection. When the conduction in all constituent fibers was blocked, the effects on the simulated spinal SSEP were approximately the same as the block involving both Group I and Group II fibers, except that a trace of late components was completely abolished at all points beyond the block.

**Discussion**

_**Relevant Experimental and Clinical Works**_

The computer model described above predicted that localization of the precise site of the conduction block can be achieved by demonstrating an abrupt reduction in the amplitude of the spinal SSEP, which is accompanied by an increased negative wave caudally and a monophasic positive wave rostrally. In animal studies, the augmentation of the unipolarly recorded spinal SSEP has been observed in the lead immediately caudal to the site of the lesion caused by acute sectioning or subacute compression of the spinal cord. In human studies using unipolar leads, the phenomenon has received little attention, although it is evident in figures showing spinal SSEP recordings for cervical spondylotic myelopathy. In one study using bipolar leads, attention was directed to this phenomenon, but insufficient explanation was given. The potential enlargement to be discussed here is entirely different from that of potentials recorded by the bipolar lead that bridged the spinal cord transection. A potential recorded bipolarly is produced as the difference between two potentials recorded unipolarly. If one of the unipolarly recorded potentials is primarily negative and the other is primarily positive, an enhanced negativity of the bipolarly recorded potential will ensue. It is fairly obvious, therefore, that when G₁ and G₂ are, respectively, at a point preceding and beyond a conduction block, the bipolarily recorded potential will show a greater augmentation than that found in unipolar recording.

With volume recording, the monophasic positive wave, the killed-end potential (KEP) or the evoked injury potential, has been known to be recordable experimentally.
Thus, the negative peak amplitude is maximized with constant-speed sub-.

The present computer model also predicted that a partial block could paradoxically increase the spinal SSEP amplitude. In one animal study,24 with constant-speed sub-acute compression of the spinal cord, the spinal SSEP was shown to be increased in amplitude initially and turned to a positive wave subsequently in the lead near the compression site. However, the mechanisms underlying the initial enhancement remain to be elucidated. Clinically, the various kinds of augmentation of the spinal SSEP were noticed during spinal cord monitoring,25 including that related to a conduction block. In one series of spinal cord monitoring during operations for scoliosis,19 there was a significant amplitude increase in the final spinal SSEP compared with initial recordings for 22 of 212 limbs stimulated. A simplistic view that a conduction block results in a reduction in the size of CNAP, therefore, does not seem to suffice in analyzing waveform changes related to a conduction block.

**Underlying Mechanisms of Waveform Changes**

The spinal SSEP is the CNAP that represents the summed potentials arising from constituent nerve fibers with different diameters. Thus, understanding of the waveform change in the NFAP due to a conduction block would be a prerequisite for more detailed insight into the phenomena seen in the spinal SSEP.

**Nerve Fiber Action Potentials.** A simulated NFAP normally consisted of an initial positive, subsequent negative, and terminal-positive peaks. With a conduction block, the simulated triphasic NFAP was turned to a positive-negative diphasic sequence or a triphasic sequence with an incompletely developed terminal positivity in the vicinity of the block. This phenomenon can be explained by the following. The terminal-positive phase of the potential is normally produced after the repolarization wavefront has passed directly under the detecting point P because the phase is derived from the positivity of the repolarization wavefront seen at the point. Hence, it follows that the potential lacks terminal positivity at the point of the block and at any point beyond the block. Even at the point preceding the block, unless this point is sufficiently far from the block, the same or a similar result is expected to ensue because $\Omega r$ with a plus sign cannot exceed or fail to exceed fully $\Omega d$ with a minus sign.

A second point deserving attention is that the negative peak of the simulated NFAP was enhanced at the points preceding the block. The longitudinal extent of depolarization, denoted by “$\lambda$” in this model, is normally a constant determined by nerve fiber diameter. With a conduction block, however, the value of “$\lambda$” decreases progressively once the depolarization wavefront reaches the point of the block (Fig. 4). Mathematically, the solid angle subtended by a wavefront is greatest when the horizontal distance between the detecting point and the wavefront is $\text{Py}/2$.1 Thus, the negative peak amplitude is maximized when $\lambda = \text{Py}/2$ (that is, twice as much as $\text{Py}/2$) because the negative peak is produced at the moment that the impulse reaches directly under P. If “$\lambda$” is normally more than $\text{Py}/2$, it approaches and then reaches $\text{Py}/2$ in the course of the progressive reduction in value caused by the conduction block, which results in an enhanced negative peak. The greatest enhancement of the peak can be expected, of course, at the point preceding the block by $\text{Py}/2$.

Another important finding was that monophasic positive waves were not produced at exactly the same point as the conduction block, but at the points beyond the block. It is because the point P directly over the block still “sees” the negativity of the repolarization wavefront, although point P has no chance to “see” the negativity of the depolarization wavefront, resulting in a reduced negative peak. The monophasic positive wave is derived from the positivity of the stationary depolarization wavefront seen at points beyond the block. It starts to be produced at the point beyond the block by $\text{Py}/2$, where $\Omega r$ with a minus sign has just failed to exceed $\Omega d$ with a plus sign. As the
detecting point is further away from the point of the block, the positive wave progressively declines as a result of the progressive reduction of $Vd$.

Spinal Somatosensory Evoked Potentials. It is known that phase cancellation normally dictates the waveform of CNAP, which means that CNAP can be conceived of as a linear summation of the constituent NFAPs. The overlap of the NFAP peaks with opposite polarity, therefore, results in cancellation.

During the course of the blocking, in which the percentage of blocked fibers was gradually increased, the negative peak of the simulated spinal SSEP increased in amplitude initially and decreased subsequently at the point of the block and at all points beyond the block (Figs. 7 right and 8). This phenomenon can be explained as follows. When only Group I fibers are involved in the conduction block, the potentials derived from those fibers are expected to be positive-negative diphasic waves with reduced negativity at the point of the block and monophasic positive waves or extinction of any wave at points beyond the block. It is important that the terminal-positive phase is lacking in either case. Because the terminal-positive phases of the Group I fibers (faster fibers) tend to overlap with the negative phases of the largest Group II fibers (slower fibers), which normally constitute a main negative peak of the simulated spinal SSEP, the absence of the former could contribute to the augmentation of the spinal SSEP, because the phase cancellation that normally happens fails to occur (Fig. 9). In a similar way, absence of initial-positive phases instead of terminal-positive phases of constituent NFAPs could be a cause of augmentation of the simulated spinal SSEP, if the smaller fibers of Group II were selectively involved, because the initial positivity of those fibers are also normally out of phase with the main negativity of the spinal SSEP. Those effects, however, are inevitably more than offset by further involvement of constituent fibers, resulting in reduction of the spinal SSEP amplitude.

At the points immediately preceding the block, the identical mechanism should contribute to the augmentation of the simulated spinal SSEP, because the terminal-positive phases derived from blocked fibers are also lacking or incompletely developed there. With the involvement of Group II fibers, there must be superimposition of the effect of the aforementioned phenomenon that the NFAPs themselves can be augmented at the points preceding the block. With the involvement of the progressively smaller fibers of Group II, the two mechanisms increased the area under the negative phase of the spinal SSEPs in association with the prolonged descending phase of the negative deflection (Fig. 7 right). It is quite understandable that the spinal SSEP at those points remained augmented even when every fiber was included in the conduction block. There should be the same mechanism underlying the equivalent phenomenon that we frequently encounter clinically in cervical spondylotic myelopathy (Fig. 1).

Furthermore, the KEP, the enhanced monophasic positive wave, can also be interpreted using the concept of phase cancellation. When a majority of Group I and Group II fibers are included in the conduction block, the isolated initial positive waves produced at the point immediately beyond the block are now set free from phase cancellation with negative waves, resulting in an enhanced monophasic positive potential. Because the conduction block in 50% of constituent fibers gave rise to the KEP in the present study, it is clear that the KEP in the clinical domain does not necessarily imply that every fiber is affected. In other words, we are testing only relatively fast conducting fibers.

Limitations of the Model

It should be emphasized that many assumptions were made in the construction of the model. The assumption of a uniform and infinite volume conductor allowed us to observe only the propagating potential field. However, with more precise models, disturbances of the uniformity within a restricted volume conductor were shown to be capable of generating the far-field potential.3,40

The square-wave approximation of an intracellular NFAP resulted in the symmetric waveform of the volume-conducted NFAP. However, the actual repolarization process is slower than depolarization, indicating an asymmetric negative phase and indicating that the terminal-positive phase should be smaller in amplitude and longer in duration than the initial-positive phase. The double-peaked NFAP for the fastest fiber shown in Fig. 6 is also an artificial result of the square-wave approximation. From this point of view, more valid would be the “tripole” concept in which depolarization and repolarization phases can have durations that are in accordance with reality.31,32,39 Furthermore, in this study $|D\Sigma|$ which indicates the sum of absolute values of the surface-dipole-moment density of active and inactive axon membranes, was treated as a constant. However, the length of the myelin surrounding the membrane is known to be different for nerve fibers of different diameter,12,45 and the difference can influence $|D\Sigma|$ because myelinated membrane is expected to have a lower surface-dipole-moment density than unmyelinated membrane. The NFAP amplitude for a different diameter can be affected differently thereby.

Nevertheless, this work will at least provide some basis for qualitative predictions. Although compound action potential enhancement in general can be associated with factors such as better synchronization of constituent action potentials,21 hyperpolarization,16 or slowed Na$^+$ inactivation,26 the mechanism of phase cancellation should also be taken into account as the cause of the phenomenon. An awareness of this possibility, which has already been predicted by Kimura, et al.,21 will help to interpret the electrophysiological data properly in both experimental and clinical domains.

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