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Title
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Abstract
Close scrutiny of scalp recorded P9 far-field potentials following stimulation of the **median nerve** often revealed dilobed wave forms. We observed that the P9 became 2 distinct peaks (P9a and P9b) when the arm was flexed 90 degrees forward at the shoulder and that it became a pointed single peak with 90-170 degrees lateral abduction of the arm. A simultaneously recorded stationary negative peak (N9), registered over the stimulated arm with the use of a distant reference, also showed similar changes, a dilobed configuration (N9a and N9b) with forward flexion and a single peak with lateral abduction. The latencies of the scalp recorded P9a and P9b and arm recorded N9a and N9b were close but not exactly the same. Nevertheless, the latencies of the scalp-positive and arm-negative peaks shifted in nearly a parallel fashion by changing the arm positions. These findings suggest that the change of axial orientation of the propagating **nerve** impulse plays an important role for the rise of P9a and that the change of volume geometry surrounding the **nerve** contributes to the P9b generation. Also, the scalp recorded P9 and arm recorded N9 are one and the same, and oriented with dipole fields extending from the arm, body and to the scalp.

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Presence of two subcomponents in P9 far-field potential following stimulation of the median nerve [1]

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Summary Close scrutiny of scalp recorded P9 far-field potentials following stimulation of the median nerve often revealed dilobed wave forms. We observed that the P9 became 2 distinct peaks (P9a and P9b) when the arm was flexed 90° forward at the shoulder and that it became a pointed single peak with 90–170° lateral abduction of the arm. A simultaneously recorded stationary negative peak (N9), registered over the stimulated arm with the use of a distant reference, also showed similar changes, a dilobed configuration (N9a and N9b) with forward flexion and a single peak with lateral abduction. The latencies of the scalp recorded P9a and P9b and arm recorded N9a and N9b were close but not exactly the same. Nevertheless, the latencies of the scalp-positive and arm-negative peaks shifted in nearly a parallel fashion by changing the arm positions.

These findings suggest that the change of axial orientation of the propagating nerve impulse plays an important role for the rise of P9a and that the change of volume geometry surrounding the nerve contributes to the P9b generation. Also, the scalp recorded P9 and arm recorded N9 are one and the same, and oriented with dipole fields extending from the arm, body and to the scalp.

Key words: Somatosensory evoked potential; Far-field potential; Stationary field potential; Volume conduction; Dipole

Scalp recorded far-field evoked potentials following stimulation of the median nerve generally consist of 4 positive peaks: P9, P11, P13 and P14. Although several theories concerning the generator mechanisms of far-field potentials have been proposed, many questions remain unanswered. On the scalp recorded far-field potentials, P9 is thought to arise near the brachial plexus (Cracco and Cracco 1976; Desmedt and Cheron 1980; Yamada et al. 1980; Lueders et al. 1983). The generation of P9 has been attributed to either a change of the volume geometry surrounding the nerve (Kimura et al. 1983, 1986; Yamada et al. 1985; Cunningham et al. 1986) or a change of the axial orientation of the propagating nerve impulse (Desmedt et al. 1983; Nakanishi et al. 1986). We have demonstrated the presence of a stationary negative potential, N9, that has a fixed latency regardless of the recording site over the stimulated arm (Yamada et al. 1985; Kameyama et al. 1988). In contrast to P9 which is recorded distant to the presumed generator site, N9 was best recorded over the acromian process, i.e., near the generator source. We have proposed that N9 is the counter-

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potential for the scalp recorded P9 representing a dipole distribution (Yamada et al. 1985).

In this study we examined the changes of N9 and P9 induced by different arm positions and studied their relationship in further detail. We believe the findings provide additional insight into the physioanatomical mechanisms for the generation of far-field potentials.

Materials and methods

We studied 10 healthy volunteers (9 males), aged 28–40 years (mean 36.1 years) after informed consent was obtained. Subjects lay on a bed and were instructed to relax with eyes closed in a semidarkened quiet room, separate from the instrument control room but in view through a window. Stimulus electrodes were flat-surfaced disks, 7 mm in diameter, placed over the right median nerve at the wrist, with the cathode 2 cm proximal to the anode. We also stimulated the ulnar nerve at the wrist in 3 subjects. Stimuli with a pulse duration of 0.1 msec were delivered at a rate of 4.3/sec via a stimulus isolation unit. The intensity was approximately 3–4 times sensory threshold and elicited a modest contraction of the abductor pollicis brevis muscle. The responses were obtained using a filter bandpass of 50–3000 Hz (−6 dB) Eight-channel outputs were simultaneously averaged using a Nicolet Pathfinder I. Each test consisted of averaging a total of 1500–2000 responses. The digitized intersample interval was 0.08 msec with an analysis time of 20 msec. Each test was repeated to confirm reproducibility of the response. Recording electrodes were silver-silver chloride cups filled with ECG gel attached to the skin with collodion. Electrode impedance was less than 5 kΩ. One electrode was placed at C3 of the 10–20 system, over the acromion process bilaterally, Erb's point, and the neck just above the C7 spinous process. Multiple electrodes were placed over the lateral aspect of the middle upper arm. The right knee electrode served as a reference for all recordings. Also multiple bipolar electrodes along the median nerve course in the forearm and arm recorded the nerve action potential. We examined responses with the following 4 different arm positions: (A) arm adduction (natural rest position), (B) 90° arm forward flexion, (C) 90° arm abduction, (D) 160–170° arm abduction (see Fig. 1). In all positions the elbow was extended.

Results

Using a non-cephalic reference (knee), scalp electrodes registered 4 positive far-field potentials (P9, P11, P13 and P14) following stimulation of the median nerve. Close scrutiny of the broadly based peak of P9 revealed a small notch on the descending phase following the onset peak (N6), suggesting the presence of two components (Fig. 1A). The dilobed peak configuration became more distinct when the arm was flexed 90° forward (Fig. 1B). We designated the first peak P9a and the second peak P9b. In the naturally rested arm position (Fig. 1A), P9b was a dominant component but P9a was identified in 7 of 10 subjects. With 90° arm flexion both peaks became distinct in all subjects. In contrast, P9 developed a more pointed configuration losing the P9a component when the arm was abducted 90° (Fig. 1C). Essentially the same change was observed with 160–170° abduction of the arm (Fig. 1D). Digital zero-phase shift filtering using the high low frequency filter was useful to identify the small

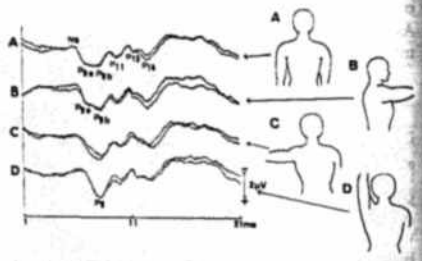

Fig. 1. Wave form changes of P9 with right median nerve stimulation in different arm positions. The SEPs were recorded from C3 referenced to the knee. Note the dilobed peaks (P9a and P9b) with position A or B and single peak with position C or D. The dilobed configuration is more distinct with position B.

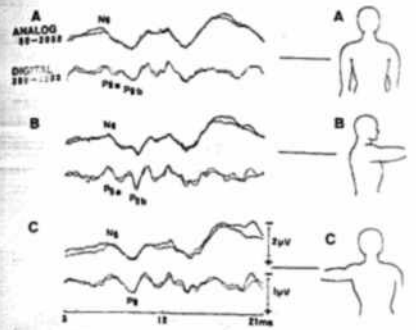

Fig. 2. Original tracings using wide open bandpass filter (top) and digitally filtered tracings (bottom) in different arm positions. Digitally filtered tracings show clear separation of P9a and P9b with positions A and B. P9a was lost with C position.

fast frequency components by eliminating the underlying slower frequency waves (Eisen et al. 1984; Green et al. 1986). Digitally filtered tracings with a bandpass of 300–2500 Hz thus confirmed the presence of two distinct peaks, P9a and P9b, with arm positions A and B but a single peak with position C or D (Fig. 2).

We then compared the scalp N6–P9 potentials with the N6–N9 responses recorded from the stimulated arm with the use of a distant reference (knee). N6–N9 are stationary potentials which have a fixed latency regardless of the recording sites but with the highest amplitude distal to the deltoid muscle and acromion, respectively (Yamada et al. 1985; Kameyama et al. 1988). With the changes of arm position, we observed similar alterations in N6–N9 potentials with those of scalp N6–P9. The latencies of the scalp and

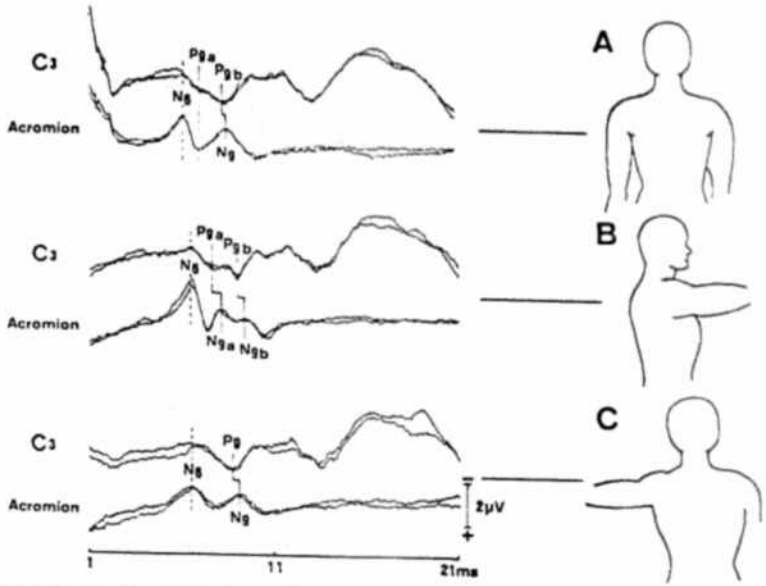

Fig. 3. Comparison of scalp recorded N6-P9 (a and b) and arm recorded N6-N9 (a and b) latencies. The latencies of scalp N6 and arm N6 were close and shifted together by changing arm positions, with longer latencies in position B or C than in A. Although the latencies of scalp P9 and arm N9 were slightly different, they shifted in nearly parallel fashion with different arm positions. Note the distinctly dilobed N9a and N9b together with P9a and P9b in position B.

TABLE I

Peak latencies (mean and S.D.) of scalp recorded potentials. A, B, C and D indicate different arm positions. The latencies of B, C and D were compared with A by the paired t test.

	N6	P9(a)	P9(b)	P11	P13	P14	N19
A	6.29	7.36	8.60	10.77	12.94	13.74	18.79
	0.46	0.55	0.75	0.92	0.94	0.93	1.69
B	6.77 *	7.98 *	9.19 *	11.23 *	12.98	13.95	19.04
	0.54	0.84	0.64	1.02	0.98	0.95	0.96
C	6.63 **	–	8.83 *	11.24 *	13.11	14.35	19.04
	0.61		0.63	0.97	0.99	1.66	1.58
D	6.86 **	–	8.95 *	11.16	13.03	14.04	19.53
	0.44		0.63	0.88	0.90	0.84	0.54

* $P < 0.05$; ** $P < 0.01$.

arm recorded N6 were very close and shifted in a nearly parallel fashion with changing arm positions (Fig. 3). They were shortest with position A (Fig. 3). We also observed similar morphological changes in N9 as seen in P9 with different arm positions. All subjects showed a double-peaked N9, named N9a and N9b, with position B and a single peaked N9 with position C (Fig. 3). With position A, separation of N9 into N9a and N9b was seen in 2 subjects. Although the latencies of P9 (P9a, P9b) and N9 (N9a, N9b) were not the same, they correspondedly shifted with different arm position showing a longer latency with position B or C than in position A (Fig. 3).

Tables I and II respectively show the mean latencies and standard deviations of the N6, P9a, P9b, P11, P13, P14 and N19 peaks recorded from the scalp and the N6, N9a, and N9b peaks recorded from the arm. Using the paired t test, N6 and P9b latencies were significantly longer in positions B, C and D compared to position A. The P9a peak registered in all subjects with position B had a significantly longer latency compared to position A. P11 in positions B or C was significantly longer than in position A. There were no statistically significant latency differences in subsequent peaks, P13, P14 and N19.

We also examined the field distribution of P9a and P9b. In position A, P9b was generally a more dominant component at the scalp electrode and P9a was more prominent at the cervical (Cv7), and

TABLE II

Peak latencies (mean and S.D.) of arm recorded potentials. For explanation of abbreviations see Table I.

	N6	N9(a)	N9(b)
A	6.42	–	8.81
	0.40		0.75
B	6.70 *	8.29	9.19 *
	0.46	0.52	0.65
C	6.73 *	–	9.08 *
	0.45		0.65
D	6.83 *	–	9.21 *
	0.44		0.64

* $P < 0.05$; ** $P < 0.01$.

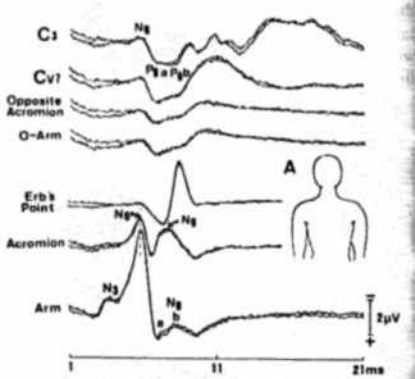

Fig. 4. Field distributions of P9 (a and b) and their relationship with arm recorded N6 and N9 (a and b) in position A (adducted position). P9a was better recorded in cervical than scalp electrodes and extended to the opposite arm (O-Arm), while P9b was better identified at the scalp. Note the small N9a and N9b at the arm (deltoid).

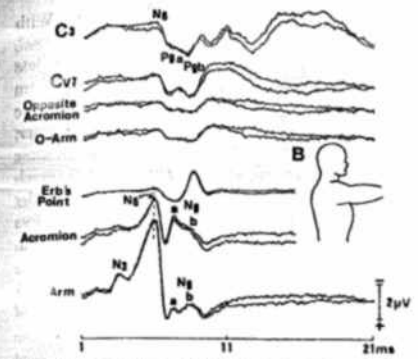

Fig. 5. The same subject and recording parameters as in Fig. 4 but with position B (90° forward abducted position). Both P9a and P9b became distinct at all recording sites, especially at Cv7. Also note the accentuation of N9a at the acromion and arm resulting in dilobed configuration of N9.

the acromion and arm electrodes opposite to the side of stimulation (Fig. 4). With position B, both P9a and P9b became more distinct, especially at the Cv7 electrode (Fig. 5). Also N9a became the dominant component at the ipsilateral acromion. In contrast, position C or D resulted in a single peaked P9 and N9 at all electrodes (Fig. 6). These findings were consistent in all 10 subjects.

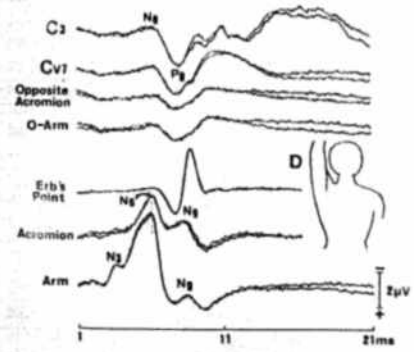

Fig. 6. The same subject and recording parameters as in Fig. 4 but with position D (160–170° lateral abduction). Both P9 and N9 were a single peaked wave form at any recording site.

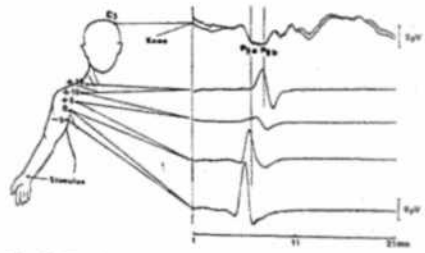

Fig. 7. The latency comparison of P9a and P9b with the traveling impulse recorded with a bipolar derivation. The P9a and P9b latencies were close to the peak latencies of action potentials recorded at the axilla and just distal to the Erb's point, respectively.

In order to estimate the site of origin for P9a and P9b, we compared the P9a and P9b latencies with the action potentials recorded with conventional bipolar electrodes along the course of the nerve in 3 subjects. This revealed P9a latency was close to the action potential at the axilla and P9b was just distal to Erb's point (Fig. 7).

Finally we tested the same maneuvers using ulnar nerve stimulation in 3 subjects and found the same changes as with median nerve stimulation.

Discussion

A number of studies have discussed the origin of the P9 far-field potential (Cracco and Cracco 1976; Jones 1977; Chiappa et al. 1980; Desmedt and Cheron 1980; Yamada et al. 1980). It has generally been agreed that P9 arises just distal to the brachial plexus. Maccabee et al. (1983) mentioned that P9 was biphasic in 45% of recordings, showing an additional earlier peak than the one generally identified as P9; however, they did not further discuss or investigate this feature. Indeed, we found dilobed P9 (P9a–P9b) or an additional small notch (P9a) over the descending phase of P9 (P9b) in 70% of the subjects when their arms were in the usual arm adducted position. Accentuation

of P9a by arm flexion and diminution of the same peak by arm abduction may provide some insight into the mechanism of generation of far-field potentials.

Far-field potentials from a traveling wave were once thought to reflect an area of positivity moving in front of a nerve impulse as it approached the recording electrode (Woodbury 1965). However, it is not known why a traveling nerve impulse along the first order afferent at a particular anatomical site would give rise to a stationary positive field in the absence of a fixed neural discharge. Nakanishi (1982) proposed that the change of resistance in the surrounding medium along a nerve may result in an abrupt alteration of the extracellular current flow giving rise to a stationary field potential. Using the hand as a model, Kimura et al. (1983, 1986) demonstrated the rise of a stationary field potential when a peripheral nerve impulse passed from the palm to the digits and proposed that an abrupt change of volume geometry surrounding the nerve gives rise to a far-field potential. Desmedt et al. (1983) observed the latency shift of the median P9 far-field potential by raising the shoulder and stressed the importance of the axial orientation of the propagated dipole in determining the features of the far-field potential. Nakanishi et al. (1986) also observed polarity inversion of far-field potentials by changing the limb position in the cat.

In accordance with the above theories proposed by different investigators, Stegeman et al. (1987) developed a computer model which evaluated the potential distribution in a cylinder of infinite length with an impulse propaging along the center line. Varying conditions included: (1) an abrupt change in medium conductivity with uniform geometry, (2) an abrupt change in cylinder volume with uniform conductivity, and (3) a change in direction of impulse propagation without a change in geometry or conductivity. All these conditions produced stationary potential peaks in the cylinder between points on opposite sides of, and distant from, the site of change. In our present study changes in wave form and latency of P9 occurred with different arm positions. Accentuation of P9a with arm flexion forward may by attributable to the near 90° angle change of the nerve axis in the vertical plane as well as the horizontal plane. With the arm resting position, neural axis is angled 90° in the horizontal plane only, resulting in a less conspicuous P9a than that with arm flexion. Arm abduction of 90–170° appears to cause relative straightening of the neural axis at the shoulder, which may explain the loss of P9a with these positions. Despite the straight entrance of a traveling nerve impulse from the arm to the shoulder with arm abduction, P9b remained. Therefore, P9a and P9b could be generated by different mechanisms: P9a by changes of axial orientation of the nerve and P9b by change of volume geometry surrounding the nerve. The field distributions of P9a and P9b also differed. P9a was better recorded from the neck to the opposite arm than at the scalp and its field appeared to be oriented more horizontally than vertically. In contrast, the P9b field appeared to be oriented vertically and was best recorded at the scalp.

We previously reported the stationary field potentials of negative polarity, N3, N6 and N9, recorded from the stimulated arm with the use of a distant (knee) reference (Yamada et al. 1985). Unlike traveling impulses their latencies were fixed regardless of the recording sites. However, their amplitude was highest nearby the generator sites: N3 at the forearm, N6 at the deltoid, and N9 at the acromion. Because of the close latency relationship between the scalp recorded P9 and arm recorded N9, we have suggested that they arise from the same generator site with a dipole representation. However, detailed examination of N9 and P9 in this study revealed slight latency differences between P9a and N9a or P9b and N9b with a consistently earlier peak of the P9a and P9b than the N9a and N9b, respectively. Nevertheless, the changes in their wave form and latency with different arm positions corresponded to each other, suggesting that scalp positivity and arm negativity are one and the same. Since the latency of a given peak within successive waves may shift depending upon the effects of phase addition and cancellation imparted by neighboring waves, the slight latency difference between scalp positivity and arm negativity does not necessarily preclude the notion that they arise from the same generator source and are distributed with a dipole relationship. The longer latencies of these negative and positive peaks with the arm abduction could possibly be explained by stretching the nerve near the axilla, but the latency shift was not sufficient to cause a corresponding delay in the scalp P14 and N19 latencies. Alternatively, a change of the anatomical orientation of tissues surrounding the nerve might shift the generator sites without causing a delay in the nerve conduction velocity.

The latencies of scalp N6 and arm N6 were also very close and shifted in a nearly parallel fashion with different arm positions, suggesting that they arise from the same source. If, however, the arm N9 and scalp P9 are oriented in a dipole with the negative field near the generator source and the positive field at a distance, it is puzzling why the N6 had the same polarity at the arm and at the scalp. Previously we have demonstrated the positivity (P6) at the knee relative to the toe suggesting the presence of counter-positivity against N6 in the lower half of the body (Yamada et al. 1985). Thus the N6 from the scalp may be attributable to the positivity from the reference electrode. Alternatively, scalp N6 may be negative relative to the reference electrode. As the field distribution of P9a and P9b is different, each far-field potential likely has its own characteristic field depending upon the site of origin and/or generator mechanism.

Emerson et al. (1984) described stationary negative potentials, N10 and N12, that have fixed latencies from the low cervical spine to the scalp after stimulation of the median nerve. The cervical recorded N13 also shows little or no latency shift from the low to high cervical spine (Emerson et al. 1984; Yamada 1988). Our N6 and N9 may share the similar physioanatomical mechanisms with these stationary negative peaks maximally recorded nearby the generator sources. Similar to the N9–P9 relationship, cervical N13 has a positive counter-field at the scalp (Yamada et al. (1980; Kaji et al. 1986) or at the anterior neck (Desmedt and Cheron 1980; Emerson et al. 1984). Lumbar N24, also a stationary potential at lumbar spine after tibial nerve stimulation, has a corresponding positive potential at the scalp (Yamada et al. 1985) or at the abdomen (Desmedt et al. 1983; Seyal and Gabor 1985). These findings suggest that the negative stationary field, generated when a traveling impulse passes a certain anatomic structure, is a prerequisite for the rise of a positive far-field potential at a distance. Our study suggests that the stationary or far-field potentials are generated and also altered by the complex and multiple anatomical substrates surrounding the nerve. In the clinical domain, abnormal far-field potentials may not always represent a conduction disturbance of nerve impulses but could reflect the change of anatomical structures surrounding the nerve.

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Somatosensory evoked potentials and magnetic resonance imaging in intraspinal neoplasms [1]

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Summary Median and posterior tibial somatosensory evoked potentials (SEPs) were studied on 25 patients with pathologically proven intraspinal neoplasms, and the results were compared and correlated with the details of clinical examination and the information derived from magnetic resonance imaging (MR). MR was abnormal in all cases and in 23 of 25 (92%) demonstrated an intraspinal expansile lesion. SEP was abnormal in 19 of 25 patients (76%). Abnormal SEPs were found in 18 of 19 patients (94%) with cervical or thoracic neoplasms but only in 1 of 6 patients (16%) with the tumor in the thoracolumbar or lumbar region. SEP-MR correlation was significant ($P < 0.05$) for thoracic intraspinal neoplasms where all 9 had an abnormal SEP showing a similar pattern of normal median and abnormal posterior tibial study. Clinically, all 7 patients with posterior column sensory deficits had abnormal SEP (100%). Abnormal SEPs were seen in 7 of 11 (63%) patients with spinothalamic deficits and in 4 of 8 (50%) of those with normal sensory examinations. Four of 9 patients (44%) with a normal neurological examination or an examination disclosing ambiguous results indistinguishable from a peripheral pathology had an abnormal SEP strongly suggesting a central sensory disorder. Comparison of preoperative and postoperative SEPs did not disclose useful prognostic information pertaining to the functional recovery.

Key words: Somatosensory evoked potentials; Magnetic resonance imaging; Intraspinal neoplasm

Spinal cord tumors account for 15% of central nervous system neoplasms (Sloof et al. 1984). An incidence of 1.3/100,000 of population has been estimated for primary spinal cord tumors compared to 6–7/100,000 for brain tumors (Percy et al. 1972). Approximately 90% of spinal cord tumors occur in adults. The symptoms of spinal cord tumors are variable but the initial complaints may be vague, sometimes limited to non-localizing pain (Schliack and Stille 1975). In such cases with no clear indication for invasive procedures, non-invasive tests may provide invaluable information as to the altered spinal cord anatomy or physiology.

Somatosensory evoked potentials (SEPs) have been shown to be sensitive to a variety of intraspinal lesions (Giblin, 1960; Halliday and Wakefield 1964; Noël and Desmedt 1980; Chiappa 1983; Lueders et al. 1983; Yu and Jones 1985; Eisen 1986; Seyal and Gabor 1987). SEP abnormalities were previously described in small groups of patients with intraspinal tumors without correlation with magnetic resonance imaging (MR) (Noël and Desmedt 1980; Riffel et al. 1984; Maguière et al. 1985). We now report SEP abnormalities in 25

[1] The opinions expressed in this paper are the private views of the authors and should not be construed as the views of the Department of Defense of the U.S. Army, or the Uniformed Services University of the Health Sciences.

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