ABSTRACT: Conflicting results have been reported about abnormalities of the N30 somatosensory evoked potential (SEP) in movement disorders. In these studies, the N30 amplitude was measured in the frontal scalp region. Our aim was to identify the scalp electrodes recording the genuine activity of the N30 generator. In 18 subjects, we recorded the scalp SEPs from 19 electrodes and found a negative potential around 30 ms reaching its maximal amplitude in the frontal region. However, neither simple visual inspection of the frontal traces nor topographic analysis could distinguish the N24 from the N30 component of the frontal negativity. Brain electrical source analysis of SEPs showed that a four dipolar source model could well explain the scalp SEP distribution. We calculated the scalp field distributions of the source activities as modeled from the scalp recordings and observed that the maximal field distribution reflecting the activity of the N30 source was in the central region, whereas that reflecting the N24 source activity was frontal. We conclude that the negative response recorded around 30 ms in the central traces represents "genuine" N30 source activity, whereas the frontal negativity, which is higher in amplitude, is a mixture of the activities of both the N30 and N24 sources.
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CENTRAL SCALP PROJECTION OF THE N30 SEP SOURCE ACTIVITY AFTER MEDIAN NERVE STIMULATION

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Recording of somatosensory evoked potentials (SEPs) after median nerve stimulation shows a large negative response at 30 ms in the frontal scalp region. Two different components, which have been labeled as N24 and N30,[9] appear to contribute to this negative potential. These components can be hard to separate in the frontal traces, because the first is inconstant and often appears as a shoulder on the rising phase of the N30. The N24 response is believed to originate from a tangential dipolar source whose positive extreme generates a P24 potential in the parietal region.[9] However, the P24/N24 dipolar field distribution cannot always be used to identify the N24 response, because the P24 potential may be indistinguishable from the temporoparietal P27 response.[6] Moreover, this latter component sometimes peaks at the same latency as the frontal N30 SEP, so that the P27/N30 and the P24/N24 scalp distributions may overlap.

The precise analysis of the N24–N30 complex and the distinction of these two negative components is important so as to improve the diagnostic capability of the early cortical SEPs. Indeed, selective abnormalities of the N30 response with preservation of the parietal SEPs (N20–P24) have been described in both frontal lesions[23] and motor system degenerative diseases.[1,5,14,16,17,19,25] These findings have suggested a precentral origin of the N30 SEP. By contrast, others have shown a preserved N30 response with the same pathologies[2,8,11,12] and some authors have concluded that the N30 component has a post-

Abbreviations: ANOVA, analysis of variance; BESA, brain electrical source analysis; CSD, current source density; SEP, somatosensory evoked potential; RV, residual variance; EPs, evoked potentials
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central location.[2] These differing results have been obtained by labeling the N30 component in the frontal traces, which are thought to reflect optimally the N30 source activity. Central recordings have usually been ignored because they seemed to represent only a mixture of frontal and parietal potentials. To our knowledge, only Ozaki et al.[13] raised some doubt about the use of the term "frontal N30" and proposed a central topography of the genuine N30 potential.

The aim of this study was to identify the scalp traces in the 10–20 system of electrode placement which reflect the genuine N30 activity maximally. To achieve this, we recorded the scalp SEPs to right median nerve stimulation in 18 healthy subjects and calculated their topographic distribution by using both linear interpolation and current source density (CSD) maps. Moreover, the SEPs recorded in our subjects underwent dipolar source modeling. This technique has proved helpful to localize the generators of evoked potentials (EPs) reflecting responses from well-identified primary sensory areas[3,7,21,22,26] and to separate clearly the activities of neighboring cerebral structures. We used source modeling to separate the different SEP generators and then projected the field distribution corresponding to their activities on the scalp. This approach permits a spatial analysis of field distributions which, in contrast to direct visual analysis of the raw data, is not dependent on spatial overlapping between multiple source activities.

MATERIALS AND METHODS

Subjects and SEP Recording. We recorded SEPs to right median nerve stimulation in 18 healthy subject (10 men and 8 women, mean age 36.7 ± 15.9 years). For SEP recording, subjects lay on a couch in a warm and semidarkened room. Stimuli (0.2 ms duration) were delivered by skin electrodes at the wrist; stimulus intensity was adjusted to be slightly above the motor threshold. The stimulation rate was 1.5 Hz. Disk recording electrodes (impedance below 5 KΩ) were placed at 19 locations of the 10–20 system (excluding Fpz and Oz). The reference electrode was at the lobe of the ear ipsilateral to stimulation, as suggested by a previous report,[24] and the ground was at Fpz. The analysis time was 64 ms, including also 5 ms of prestimulus delay, with a bin width of 250 μsec. The amplifier bandpass was 1–3,000 Hz (12-dB rolloff). An automatic artifact-rejection system excluded from the average all runs containing transients exceeding ± 65 μV at any recording channel. To ensure baseline stabilization, SEPs were digitally filtered offline by means of a digital filter with a bandpass of 19–1,900 Hz. Two averages of 1,000 trials each were obtained and printed out by the computer on an ink-jet printer. Maps showing the distribution of the responses over the scalp were obtained by linear interpolation from the four nearest electrodes. We also calculated the CSD traces and maps by means of the Laplacian transformation of the potential values without taking the reference electrode into account, as suggested by Hjorth.[10] CSD mapping allows regions to be identified where current exits (current sources) or enters the head (current sinks) and may be particularly useful for studying the topography of responses peaking close to each other in time and in space.

SEP Analysis. Scalp SEPs were identified independently on the basis of latency, polarity, and scalp distribution. In particular, when there was one frontal negativity with a latency of approximately 30 ms, it was labeled the N30 potential. A well-defined shoulder on the N30 upslope was named the N24 response. When there were two separate frontal negative components, the earlier one was labeled the N24 potential. Amplitudes were measured from baseline on the average of the two runs. In order to assess the distribution of the responses, their amplitudes were submitted to analysis of variance (ANOVA), considering scalp location as source of variability. When statistical significance was reached, a post-hoc analysis using Student's *t*-test with Bonferroni's correction for multiple comparisons was also performed.

Brain Electric Source Analysis. Detailed description of brain electrical source analysis (BESA) is provided elsewhere.[20] Basically, BESA is a program that allows a model of dipolar generators of scalp EPs to be computed and then verifies that the hypothesized model can explain satisfactorily the recorded scalp EP topography. It calculates potential distributions over the scalp from preset voltage dipolar sources within the brain, and then evaluates the agreement between the recorded and calculated field distributions. The percentage of data that cannot be explained by the model is expressed as residual variance (RV). The lower the RV, the better is the dipolar model; in an ideal case, the RV should be due only to the recorded noise. In general, RV values lower than 10% are considered good, especially if obtained from analyses of individual EP recordings. After the model is built, the contribution of each dipolar activity to the potentials recorded by the different scalp electrodes can also be calculated. BESA uses a spherical three-shell model

with an 85 mm radius and assumes that the brain surface is at 70 mm from the center of the sphere. The spatial position of each source is described on the basis of three axes: 1) the line through T3 and T4 (x axis); 2) the line through Fpz and Oz (y axis); 3) the line through Cz (z axis). The three axes have their intersection point at the center of the sphere. The spatial orientation of the sources is described by two angles: 1) phi is the angle in the xy plane measured anticlockwise from the nearest x axis; 2) theta is the vertical angle that is measured from z axis and is positive for the right hemisphere. The model calculated by BESA is a hypothesis which does not exclude other solutions, but it can be validated when applicable to individual data and coherent with the anatomical and physiological knowledge of the identified source areas.

Statistical Analysis of BESA Results. Dipolar source parameters in single individuals were compared using Wilcoxon's test. In order to assess the distribution of the responses generated by a single dipolar generator, their amplitudes measured in different traces were submitted to ANOVA, considering scalp location as source of variability. When statistical significance was reached, a post-hoc analysis using Student's *t*-test with Bonferroni's correction for multiple comparisons was also performed.

RESULTS

SEP Traces. In all our subjects, the parietal traces showed the N20 potential following the widespread lemniscal P14 SEP. Later, the P24 potential was identifiable in 11 subjects. At a latency of approximately 30 ms, a large negative response was present in the frontal and central traces. By using the criteria described above (see Materials and Methods), we labeled the N24 and N30 components in this negative potential recorded by the central (C3, Cz) and frontal (F3, Fz, F4) leads. In four subjects, the N24 potential was not identifiable. In 13 out of the remaining 14 subjects, the N24 SEP appeared as a shoulder on the rising phase of N30 (Fig. 1), whereas in 1 subject, two well-separated negative responses were recognizable. The N24 mean amplitude was higher in the Fz traces than in traces from the other scalp locations (Fig. 2A), but there were no significant differences among the frontocentral electrodes (ANOVA: $F = 2.1$, $P = 0.09$). Instead, the interaction across N30 amplitudes and electrode position reached statistical significance (ANOVA: $F = 4.525$, $P < 0.005$). The post-hoc analysis showed that the N30 amplitude was maximal in the F3 and Fz traces ($P < 0.05$; Fig. 2B). The similar scalp distribution of both N24 and N30 amplitudes corresponded to a similar topography of these SEP components in the linear interpolation maps (Fig. 1).

In 9 out of 18 subjects, a further negative response, which probably corresponds to the N33 component,[18] was identifiable in the central and parietal traces contralateral to the stimulated side (C3 and P3). While in 7 subjects, the N33 latency was later than the N30 latency, in 2 subjects, it was earlier. In these last subjects, the N33 potential could be distinguished from the N30 component because it spread to the contralateral parietal region, whereas the N30 diffused to the frontal cortex.

CSD Mapping. Theoretically, CSD traces should differentiate clearly the scalp distributions of two overlapping but different SEPs. However, both the N24 and N30 responses showed their maximal mean amplitudes in the F3 traces (Fig. 1), even if only the interaction between N30 amplitudes and electrode position reached statistical significance (ANOVA: $F = 7.693$, $P < 0.0001$; post-hoc analysis $P < 0.05$).

Brain Electrical Source Analysis. The four-dipolar source model, which we have already tested in other series of healthy subjects,[27–29] was applied to the traces recorded from each individual (Fig. 3) in the time interval from the P14 to the N30 responses. It included a first dipolar source (1) at the base of the skull, which explained the P14 potential, i.e., the entrance of the afferent volley into brain volume, and three perirolandic sources, activated at the latencies of the early cortical SEPs. Dipolar source 1 could not be fitted because of the limited number of electrodes we used; therefore, we chose a low location for it, according to the position of the lemniscal activity source in previous median nerve models.[4,7] Instead, the other three sources were allowed to move freely. Source 2 assumed a tangential orientation in the convexity of the sensorimotor region and was activated twice in all our subjects: initially at the same latency of the N20 response and then in the latency-range of the N24–N30 complex. Dipolar source 3 activity also showed two peaks, but in the 2 subjects in whom the centroparietal N33 potential had an earlier latency than the N30 response, it showed a third activity-peak. Lastly, source 4 was activated once in the N24–N30 complex latency-range. The RV values ranged from 2.3 to 9.7% with the mean at 5.6%. Mean RVs and dipolar coordinates in all our subjects are shown in Table 1. From the dipolar source modeling, two different source activities, namely the last activity of source 2 and the dipolar source 4 activity, contributed to the electric

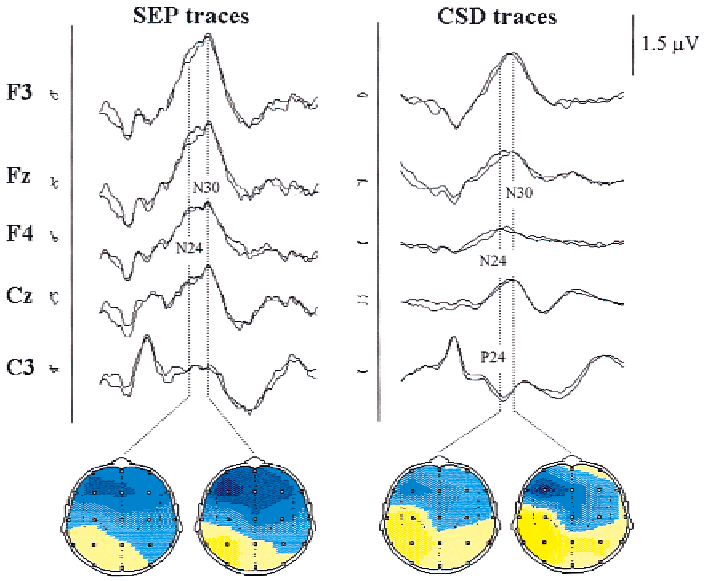

FIGURE 1. SEP traces (left) and CSD traces (right) recorded by the F3, Fz, F4, Cz, and C3 electrodes in one subject. In the lower row, maps obtained by linear interpolation (left) and CSD mapping (right) are shown. Maps are calculated at the N24/P24 and N30 latencies (dotted lines). Note the very similar scalp distribution of both responses.

signal evoked in the N24–N30 complex latency-range. Therefore, we evaluated the singular contribution of each of these dipolar activities to the potentials recorded by the centrofrontal (F3, Fz, F4, Cz, and C3) scalp electrodes. There was a significant interaction across amplitudes and electrode position for both the last activity of source 2 (ANOVA: F =12.895, $P < 0.0001$) and dipolar source 4 (ANOVA: F = 13.39, $P < 0.0001$). The post-hoc analysis showed that the last activity of source 2 generated significantly higher potentials in the frontal than central traces ($P < 0.001$; Fig. 4A), whereas the potentials that originated from dipolar source 4 had a significantly higher amplitude in the central than frontal recordings ($P < 0.001$; Fig. 4B). Figure 5 shows the contribution of both the last activity of dipolar source 2 and source 4 activity to the centro-frontal traces in 1 healthy subject.

DISCUSSION

Our study confirms the difficulty in labeling the N24 and N30 potentials in the traces recorded by frontal electrodes. Indeed, in 4 subjects, the N24 potential was not identifiable and only in 1 case did it appear as a response well-separated from the following N30 component. Since the amplitude distribution of both the N24 and N30 responses was very similar, the scalp topography obtained by both linear interpolation and CSD mapping did not help us to distinguish the two components. The BESA revealed two different source activities (the last activity of dipolar

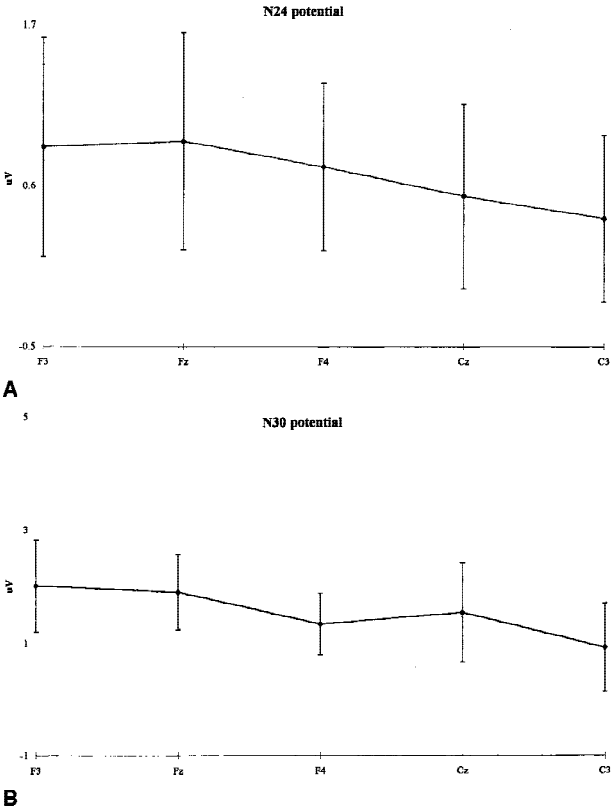

FIGURE 2. (A) Mean N24 amplitudes calculated in the F3, Fz, F4, Cz, and C3 SEP traces across all our subjects. The vertical lines represent SDs. **(B)** Mean N30 amplitudes calculated in the F3, Fz, F4, Cz, and C3 SEP traces across all our subjects. The vertical lines represent SDs. The maximal amplitudes of both the N24 and N30 components are recorded by the frontal electrodes.

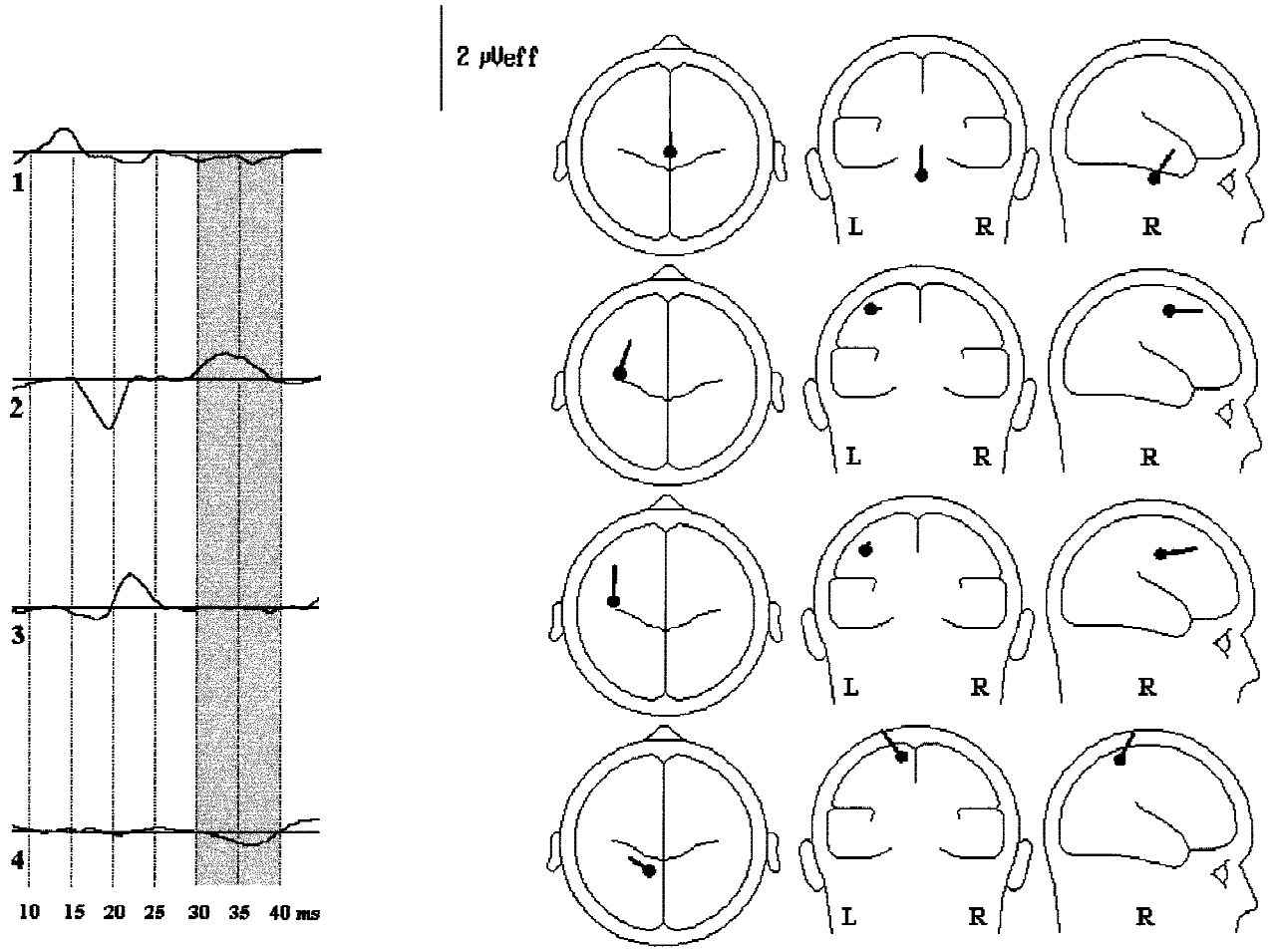

FIGURE 3. Four dipolar source spatiotemporal solutions for the SEPs shown in Figure 1. The residual variance is 3.9%. On the left, the source potentials are shown. On the right, three head views illustrate the location and orientation of the sources. The top row shows source potentials and location of the dipolar source at base of the skull (source 1). The source potential and location of the tangential perirolandic dipolar source (2) are shown in the second row. The third and fourth rows show source potentials and locations of the other perirolandic sources (3 and 4, respectively). Note that only dipolar sources 2 and 4 are activated in the N24–N30 complex latency range (gray area).

source 2 and the source 4 activity) contributing independently to the electric signal evoked in the N24–N30 latency range. Source 2, tangentially oriented in the sensorimotor region, shows two activity peaks, the earlier of which generates the N20/P20 response, whereas the other, opposite in polarity, probably corresponds to the P24/N24 generator, as we have discussed in detail previously.[27,28] The biphasic time course of dipolar source 2 is very similar to that expected from a "primary response," where an initial excitation precedes an inhibition phase.[2] However, the possibility that the opposite activity peaks of source 2 originate from very close, but different, clusters of neurons cannot be discarded.[28] In our dipolar source model, source 4, which was activated once in the N24–N30 complex latency-range, is likely to represent the generator of the N30 potential.[15,27,28]

Table 1. Coordinates of the dipoles.*

		Dipole 1					Dipole 2					Dipole 3					Dipole 4				
	RV (%)	x	y	z	Theta	Phi	x	y	z	Theta	Phi	x	y	z	Theta	Phi	x	y	z	Theta	Phi
Mean	5.6	0	0	−30	−32	−90	−46	−2.1	47.4	−103	66.6	−44	0.95	41.4	−16.5	3.38	−29	−30	40.6	−24.2	3.05
SD	2.14	0	0	0	0	0	2.71	4.82	2.98	11.5	11.9	7.24	4.4	7.03	74.6	71.6	13.9	6.5	9.76	60.9	39.8

SD, standard deviation.

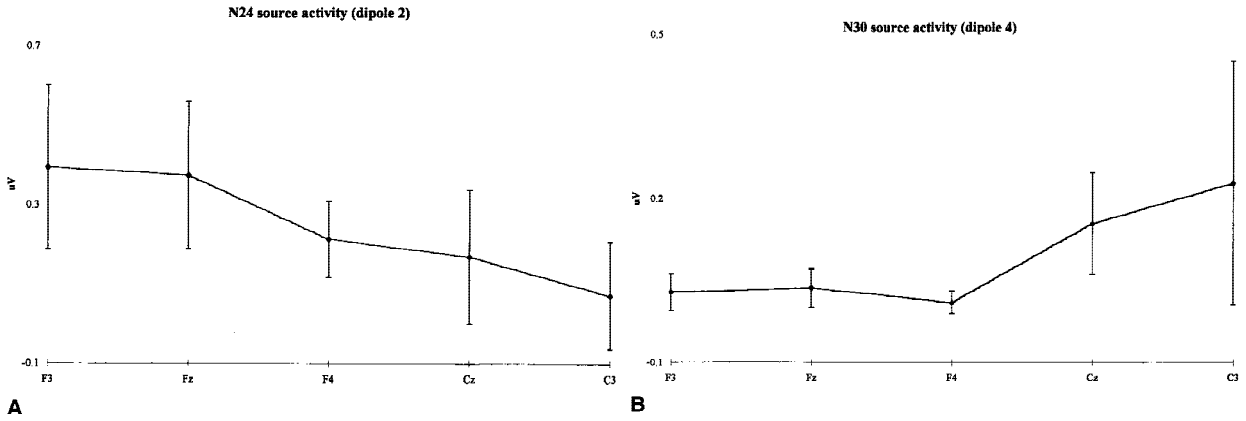

FIGURE 4. (A) Mean amplitudes of the potentials due to the last activity of source 2 in the F3, Fz, F4, Cz, and C3 traces across all our subjects. The vertical lines represent SDs. **(B)** Mean amplitudes of the potentials due to the dipolar source 4 activity in the F3, Fz, F4, Cz, and C3 traces across all our subjects. The vertical lines represent SDs. While the contribution of the last activity of source 2 is maximal on the frontal traces, the highest strength of the source 4 activity is picked up by the central leads.

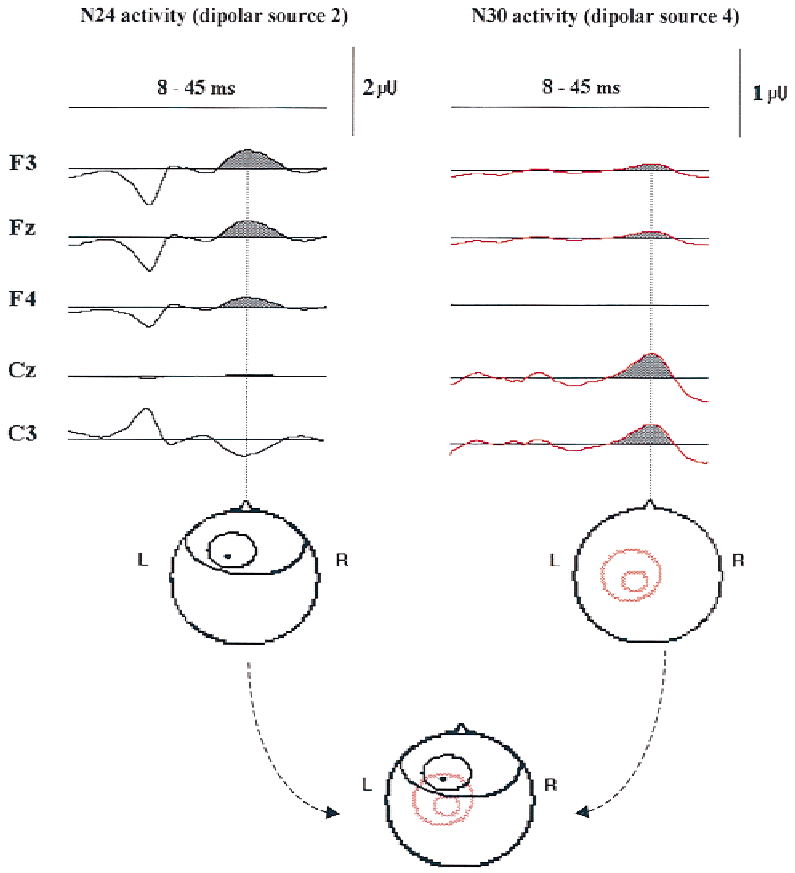

FIGURE 5. Scalp field distribution of the N24 (source 2) and N30 (source 4) source activities, as modeled from the scalp recordings shown in Figure 1. The potentials due to the N24 source activity are shown in black, while those generated by the N30 source activity are shown in red. Note that the maximal amplitudes of the potentials due to the N24 activity (gray areas on the left) are recorded by the frontal electrodes, while the N30 activity contributes mostly to the central responses (gray areas on the right). In the lower row, the negative fields of the dipolar maps corresponding to the N24 activity (left) and to the N30 activity (right) are shown. While the negative field of the N24 activity is distributed in the frontal scalp region, the N30 activity projects to the central fields. The lowest map is derived from the superposition of the previous two dipolar maps.

The analysis of the different contribution of sources 2 and 4 to the centrofrontal traces recording the N24–N30 complex showed that the dipolar source 2 activity projects mostly to the frontal region, whereas the source 4 activity is recorded especially by the central leads. Therefore, we suggest that in raw data, the N30 amplitude should be measured in the traces recorded by the vertex electrode (Cz) and the central electrode contralateral to the stimulation (C3 in this instance), where the N30 generator activity (source 4) is maximal. Instead, the negative potential recorded by the frontal leads around 30 ms receives its major contribution from the N24 source activity (dipolar source 2). Our findings were derived from dipolar source modeling of SEPs. In their interpretation, it should be considered that every dipolar source model represents a hypothesis, which does not exclude other solutions. Dipolar source modeling is an attempt to distinguish the different sources contributing to the evoked electric signal and two main criteria may be useful to judge its results: 1) a low RV, which indicates that the scalp EP topography is almost entirely explained by the model; and 2) the agreement between position and activity of the different sources and anatomophysiological knowledge. Beyond the low RV values obtained from individual traces, the proposed model has the advantage that the cortical sources show noninterfering, thus physiological, activities and are apparently located in the rolandic region, corresponding with the arrival of somatosensory inputs to the cortex.

In conclusion, the many conflicting descriptions of the N24 and N30 responses in pathological situations may be due to the fact that their amplitudes were measured only in frontal traces, with the central recordings being neglected. Instead, our results give substance to the hypothesis of a central scalp distribution of the genuine N30 response after median nerve stimulation,[13] and suggest that the central electrodes are preferable for recording the maximal N30 source activity.

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