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TRAVELING WAVES OF THE HUMAN SCALP-RECORDED SOMATOSENSORY EVOKED RESPONSE:

EFFECTS OF DIFFERENCES IN RECORDING TECHNIQUE AND SLEEP ON SOMATOSENSORY AND SOMATOMOTOR RESPONSES[1]

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Since Dawson (1947) introduced averaging techniques by recording the somatosensory evoked response (SER) from the human scalp, differences in the wave form characteristics of the scalp-recorded SER have been reported by many laboratories. These differences may be attributed to differences in stimulus parameters, variability contributed by background EEG activity, fluctuations in subject arousal level and contamination by potentials of extracranial origin. Differences in recording technique are also thought to be responsible (Broughton 1969; Goff *et al.* 1969; Vaughan 1969).

In this study the effect of differences in recording technique on scalp-recorded somatosensory and somatomotor (Cracco and Bickford 1968) responses was investigated by comparing ear reference and bipolar recordings. Marked differences between the two recording methods were found. With somatosensory responses, this was largely due to differences in the latencies of individual components at any two recording locations. Therefore, anterior–posterior (A–P) and coronal right ear reference recordings were obtained and the latencies of SER components at different recording locations were compared. The effect of sleep on the SER and on scalp-recorded somatomotor responses was also investigated.

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METHODS AND MATERIALS

Observations were made on 18 normal adult volunteers (12 females, 6 males) ranging in age from 17 to 37 years. Subjects relaxed in a comfortable reclining chair in a quiet room. Stimulating electrodes were placed over the right median nerve just proximal to the wrist. These consisted of tin discs (7 mm in diameter) attached with collodion and filled with conductive jelly. The cathode was placed 3 cm proximal to the anode. Stimulation pulses (0.2 msec duration) were generated by a Grass S-8 stimulator at a rate of 1 every 1 or 2 sec. The stimulus intensity was adjusted to produce a twitch of the thumb.

Recordings were made from tin discs (7 mm in diameter) attached to the scalp with collodion and filled with conductive jelly. In all subjects 4 recording electrodes were attached to the left side of the scalp in the A–P plane. Additionally, in 6 subjects 4 recording electrodes were attached in a central coronal plane. Right ear reference recordings were performed and the 4 leads were recorded simultaneously.

In 5 subjects the effect of sleep on the SER was studied in A–P right ear reference recordings taken from the left side of the scalp. Chloral hydrate (0.5 or 1.0 g) was administered orally to 3 subjects 1 h prior to the recording sessions. Two subjects slept without medication. Level of consciousness was monitored electroencephalographically.

In 8 subjects right ear reference and bipolar recordings from a variety of scalp locations

were compared. In 5 of these subjects, reference and bipolar recordings were performed simultaneously. In 3 bipolar and reference recordings were alternated.

Evoked responses associated with active contraction of the scalp musculature were studied in 5 subjects. These responses were elicited by applying tension to the temporalis muscles or neck extensors. In separate experiments, each subject clenched his teeth or held his head erect while tension was applied to the back of the head. Right ear reference and bipolar recordings from a variety of scalp locations were compared. Recordings were performed both with the subject relaxed and during muscle tensing maneuvers. Myogenic potentials were differentiated from cerebral responses on the basis that the former were most prominent in relation to contracting muscle groups and were recorded only with applied local muscle tension or were markedly augmented by it.

Input from the recording electrodes was led to Tektronics 3A9 differential amplifiers with a frequency response of 1–1000 c/sec. The output was summated by a Fabri-Tek 1072 computer and then recorded by an X–Y plotter. The computer was triggered by the stimulator. Routinely 128 responses were summated and analysis times of 40 or 200 msec were used. Recordings were continuously displayed on a cathode ray oscilloscope which was visually monitored for extraneous potentials including myogenic activity. A minimum of 3 summated responses were obtained from each recording location. Response peak latencies at each recording location were measured and compared. Two or three traces were often superimposed to demonstrate the constancy of the observations.

RESULTS

Effect of differences in recording technique

Somatomotor response. Evoked responses which were affected by applied local muscle tension and which were most prominent in relation to contracting muscle groups were recorded in right ear reference recordings in all 5 subjects in whom they were studied. These responses were often relatively symmetrical on the two sides of the scalp. They were attenuated in biparietal recordings and in close bipolar A–P leads.

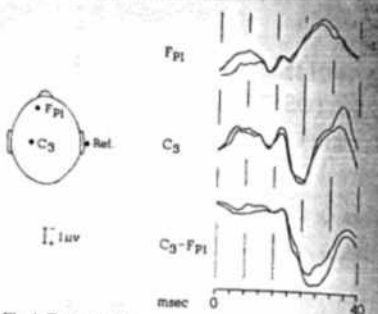

Fig. 1. Two recordings are superimposed in each trace. The initial positive potential (peak latency 14 msec) is similar in amplitude in the reference leads and cancels in the bipolar record. The peak latencies of the subsequent negative, positive and negative potentials are greater at central than at frontal recording locations. These differences are reflected in the bipolar lead where potential peaks do not coincide with those in either reference recording (Subject 12).

Somatosensory response. In right ear reference recordings the initial positive potential (parietal peak latency 13–17 msec) was widespread in its distribution over the scalp. It was consistently attenuated in bipolar recordings (Fig. 1). In reference recordings, the subsequent negative and positive potentials (parietal peak latencies 17–22 and 22–30 msec) were greatest in amplitude at parietal recording locations contralateral to the stimulated median nerve. Their peak latencies were not identical in frontal and central or frontal and parietal reference leads or in bipolar and reference recordings (Fig. 1). In some subjects their amplitudes were greater in central-frontal and parietal-frontal leads and less in parietal-occipital leads than in parietal reference recordings (Fig. 2). In reference recordings, the next negative component (parietal peak latency 26–42 msec) was usually most prominent at frontal and central recording locations contralateral to the stimulated median nerve. In most subjects its peak latency was not identical at frontal and parietal recording locations. Therefore, in parietal-frontal and central-frontal leads it often reversed in direction and changed in latency or vanished (Fig. 2).

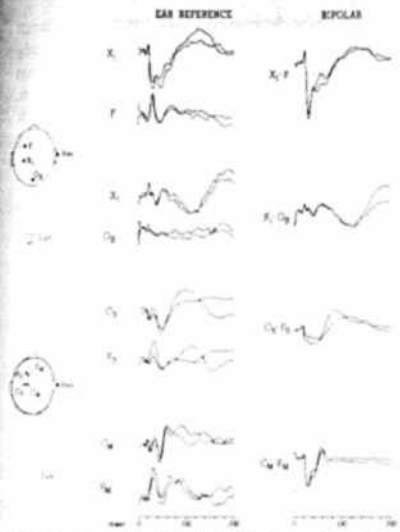

Fig. 2. *Top traces*: Electrode X_1 is placed over the anterior parietal region 7 cm to the left of a point 2 cm behind the vertex. Electrode F is placed 7 cm anterior to X_1. In the parietal-frontal recording the upward and downward deflections peaking at 22 and 29 msec are greater in amplitude than the corresponding negative and positive potentials in the parietal reference lead (Subject 5). *Second set of traces*: Electrode O_Z is placed midway between the O_1 and O_2 placements of the 10–20 system. Potentials in the parietal-occipital recording are similar in configuration but smaller in amplitude than those in the parietal-reference lead. This is due to the occipital potentials of similar latency and polarity (Subject 9). *Third set of traces*: The negative potential peaking at 30 msec in the central reference lead is not apparent in the central-frontal recording. This is due to the frontal potential of similar latency and polarity (Subject 14). *Bottom traces*: Electrode C_m is placed 4 cm to the left of C_z. F_m is 4 cm anterior to C_m. The negative potential peaking at 34 msec in the central reference lead is reflected as a downward deflection in the bipolar record. This is due to the larger frontal negative potential of similar latency. The subsequent positive potential in the central reference recording is not well defined in the bipolar record. This is due to the frontal positive potential of similar latency. Potential peaks in the 3 recordings do not coincide (Subject 10).

Later components also showed marked differences in configuration in bipolar and reference recordings. They were sometimes greater or less in amplitude, altered in peak latency, reversed in direction or vanished in bipolar leads compared with reference recordings (Fig. 3). As with the earlier components, this was due to differences in the wave form characteristics of the response at any two recording sites.

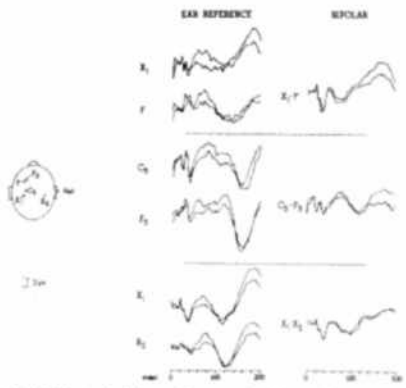

Fig. 3. Electrodes X_1 and X_2 are placed over the anterior parietal regions 7 cm to the left and right of a point 2 cm behind the vertex. *Top traces*: The onset latency of the last upward deflection in the bipolar record corresponds to that of the late frontal positive potential. The peak latency of the late negative potential is different in each of the three leads (Subject 1). *Middle traces*: The large late positive potential recorded in both reference leads cancels in the bipolar recording (Subject 16). *Bottom traces*: The late positive and negative potentials recorded in both parietal reference leads largely cancel in the biparietal recording (Subject 17).

Comparison of SER peak latencies in the A–P plane

In reference recordings, the initial positive potential (peak latency 13–17 msec) showed no consistent A–P latency differences. In all subjects the peak latencies of the subsequent negative and positive potentials (parietal peak latency 17–22 and 22–30 msec) increased (1–3 and 2–9 msec) from front to back. Like most subsequent components which showed A–P latency differences, these were greatest across the central fissure. In most subjects the peak latencies of these potentials increased progressively at each recording location (Fig. 4). One result of these findings was that at parietal regions their peak latencies were often similar but usually not identical to those of subsequent potentials of opposite polarity recorded at frontal regions

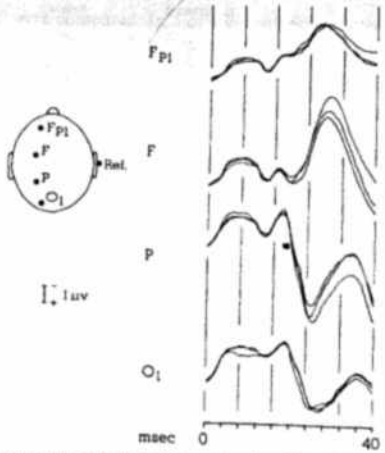

Fig. 4. Electrodes F and P are placed equidistant between FP_1 and O_1. Three recordings are superimposed in each trace. The initial positive potential peaks at 14 msec at all recording locations. The subsequent negative, positive and negative potentials progressively increase in peak latency from front to back. These latency differences are greatest between F and P. The peak latencies of the negative and positive potentials (parietal peak latencies 18 and 25 msec) in posterior leads are similar but not identical to those of the subsequent positive and negative potentials in anterior leads (Subject 15).

(Fig. 4). This would account for some of the differences in wave form configuration between bipolar and reference recordings. These latency differences persisted after the input leads to the amplifiers were reversed which suggests they were not due to equipment malfunction.

A negative component (parietal peak latency 26–42 msec) was recorded in 15 subjects. In 2 consistent latency differences were not apparent. In 10 this component increased (1–8 msec) and in 3 it decreased (4–6 msec) in peak latency from front to back in referential recordings (Fig. 4). In the 3 subjects in whom its peak latency decreased, it was poorly defined in the parietal-occipital region where it appeared as a notch.

A positive component (parietal peak latency 31–54 msec) was recorded in 11 subjects. In 2 consistent latency differences were not apparent. In 4 this component progressively increased in peak latency (2–10 msec) and in 5 its peak latency progressively decreased (2–14 msec) from front to back. In 3 of the subjects in whom its latency decreased, the preceding negative potential was poorly defined in the parietal-occipital region.

In some subjects peak latencies of later components were not identical at the 4 recording locations in the A–P plane but progressive differences were not apparent. In 5 subjects progressive latency differences were evident in some trials. In 2 a negative and a positive potential (parietal peak latencies 40–50 and 60–70 msec) increased (3–15 msec) and decreased (4–10 msec), respectively, in peak latency from front to back. In 2 a positive and a negative potential (parietal peak latencies 85–100 and 128–150 msec) decreased (10–30 msec) and increased (10–34 msec) in peak latency in the A–P plane. In 1 a negative and a positive potential (parietal peak latencies 80 and 100 msec) decreased (16 msec) and increased (10 msec) in peak latency from front to back.

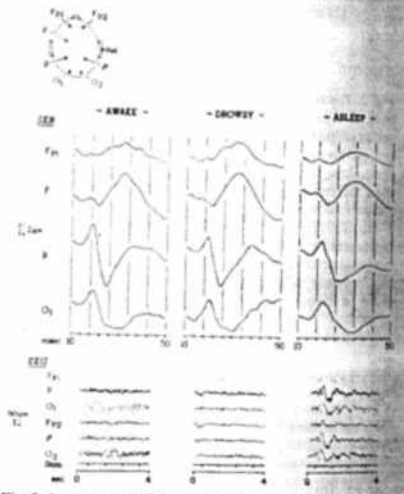

Fig. 5. A segment of EEG recorded during each evoked response trial is shown in the lower portion of the figure. There is a delay of 10 msec between the stimulus and the sweep onset. With the subject awake, drowsy and asleep, the negative, positive and negative potentials (awake parietal peak latencies 19, 25 and 35 msec) progressively increase in peak latency from front to back. With sleep, these peak latencies increase at each recording location. These latency increases with sleep are greater in posterior than in anterior leads (Subject 18).

Effects of sleep on SER peak latencies and on somatomotor responses

Although random myogenic potentials were occasionally observed in sleeping subjects both in the EEG and on the oscilloscope monitor, reproducible, time-locked evoked myogenic responses were not recorded during sleep.

SER A–P latency differences recorded when subjects were awake persisted during sleep in all 5 subjects on whom sleep recordings were performed. The latency of the initial positive potential did not change with sleep. Peak latencies of subsequent components often increased. In 3 subjects peak latencies of certain potentials recorded over the parietal-occipital regions increased more than those of corresponding potentials recorded over frontal regions resulting in greater A–P latency differences during sleep (Fig. 5). In 2 awake subjects a negative potential peaking at 28–38 msec diminished in amplitude and then vanished during sleep, the subsequent positive potential decreased in peak latency and it was followed by a negative potential which was not clearly

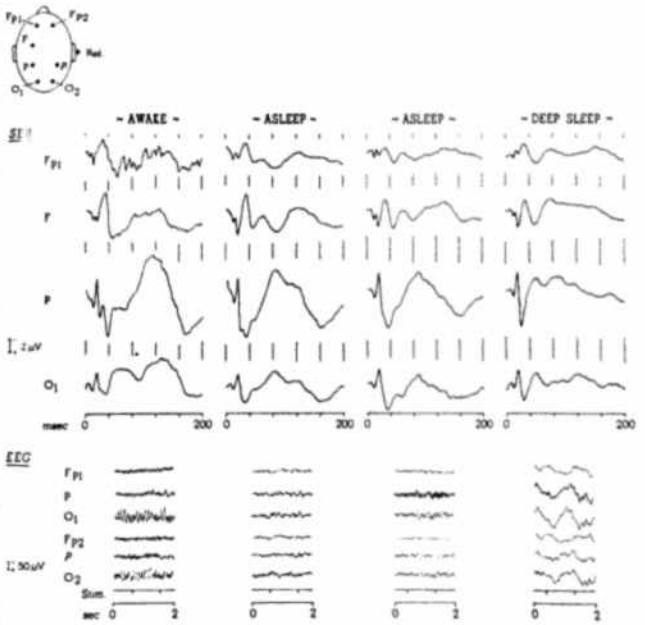

Fig. 6. With the subject awake (*left column*), a "W" shaped response consisting of a positive–negative–positive potential (parietal peak latencies 24, 28 and 38 msec) is recorded in the parietal lead. Random myogenic potentials are recorded in the frontal lead. With sleep, the negative potential becomes smaller (*column* 2) and disappears (*column* 3) changing the W to a V, the positive limb of which decreases in peak latency and is followed by a negative potential (parietal peak latency 50 msec) (*right column*) which was not well defined in the awake subject. These potentials progressively increase in peak latency from front to back. The large late negative potential (awake parietal peak latency 110 msec) decreases in peak latency during spindle sleep (*columns* 2 *and* 3) and is not clearly defined during slow wave sleep (*right column*). During spindle sleep peak latencies of later components are different at anterior and posterior recording locations (Subject 7).

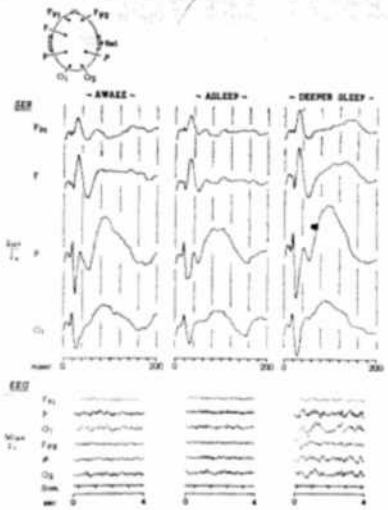

Fig. 7. In the awake subject, the late negative potential (parietal peak latency 85 msec) is confined to the parietal and occipital leads. Its peak latency increases with sleep. During slow wave sleep (*right column*) a similar potential is recorded in frontal leads which progressively decreases in peak latency from front to back (Subject 13).

defined when the subjects were awake. Both these potentials progressively increased in peak latency from front to back (Fig. 6). In 2 subjects a late negative potential (awake parietal peak latency 110–120 msec) decreased in peak latency during spindle sleep and was not clearly defined during slow wave sleep (Fig. 6). In 2 awake subjects a late negative potential (parietal peak latency 85–100 msec) was limited in its distribution to posterior leads. During sleep its peak latency increased (5–8 msec) and a similar potential was recorded in frontal leads whose peak latency progressively decreased from front to back (Fig. 7).

Comparison of SER peak latencies in the coronal plane

Consistent differences in the latency of the initial positive potential on the two sides of the scalp were not apparent in coronal referential recordings in the 6 subjects studied. In 4 subjects the peak latency of the next negative potential was 2–4 msec greater over the hemisphere ipsilateral to the side of stimulation. Similarly, the subsequent positive, negative, positive and negative potentials (left parietal peak latencies 23–27, 29–34, 38–45, 54–56 msec with right median nerve stimulation) were 1–5, 2–3, 1–8 and 2–6 msec greater in peak latency ipsilateral to the side of stimulation in 3, 2, 4 and 3 subjects, respectively (Fig. 8). In 1 subject a negative potential (right parietal peak latency 37 msec with left median nerve stimulation) was greater in peak latency contralateral to the side of stimulation (Fig. 8). These latency differences over the two hemispheres shifted sides when the side of nerve stimulation was changed in the 2 subjects in whom responses to both left and right median nerve stimulation were recorded. In 4 subjects peak latencies of certain components were greater at lateral than at medial locations in coronal recordings taken from the same side of the scalp (Fig. 8).

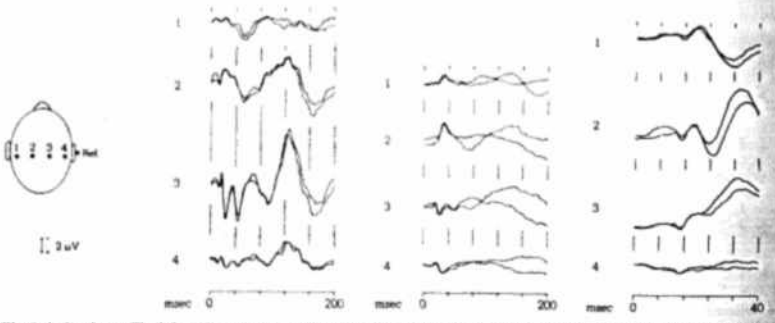

Fig. 8. *Left column*: The left median nerve was stimulated. Electrodes are placed 2 cm behind T_3 and T_4 and at two locations equidistant between these. Peak latencies of several components (at electrode 3: negative at 18 msec, positive at 24 and 44 msec) are greater over the left hemisphere than over the right (Subject 4). *Middle column*: The left median nerve was stimulated. The peak latency of the negative potential (37 msec at electrode 3) is greater over the right hemisphere than over the left (Subject 11). *Right column*: The right median nerve was stimulated. The peak latencies of the negative and positive potentials (19 and 25 msec at electrode 2) are greater at electrode 1 than at electrode 2. The peak latency of the subsequent negative potential progressively increases from electrode 3 to electrode 1 (Subject 11).

DISCUSSION

Scalp-recorded evoked myogenic responses were attenuated in close bipolar A–P and biparietal recordings. They were not recorded in sleeping subjects. This suggests that myogenic contamination of the scalp-recorded SER can be reduced by using close bipolar recording methods or by recording during sleep. This is not unexpected since these myogenic responses are bilateral, often symmetric, and maximal at recording locations which overlie contracting muscles (Cracco and Bickford 1968). Since myogenic responses may be greater in amplitude and similar in latency to cerebral responses, they may potentially cause very serious problems in the interpretation of the scalp response. However, they do not seem to pose a serious problem in normal, cooperative subjects if care is taken to ensure relaxation of the scalp musculature (Calmes and Cracco 1971). Since the SER is also markedly affected by differences in recording technique, it may be preferable to reduce the risk of myogenic contamination by ensuring relaxation of the scalp musculature, monitoring the evoked response for myogenic activity or recording during sleep.

Investigators have used different scalp locations to record the SER. The central or parietal region contralateral to the stimulated nerve has usually been used as the "active" electrode site and the anterior-frontal region, the posterior-frontal region, the nose or the ear have been used as the "reference" site (Dawson 1954; Shagass and Schwartz 1961, Goff *et al.* 1962; Debecker and Desmedt 1964; Giblin 1964; Broughton 1969). The results of this study demonstrate the marked differences in SER wave form characteristics between bipolar and reference recordings. The ear was selected as the reference electrode site because it is relatively inactive when compared with other off-scalp cephalic and non-cephalic recording locations (Goff *et al.* 1969; Lehtonen and Koivikko 1971). Components were increased or reduced in amplitude, altered in latency, reversed in direction or vanished in bipolar leads compared with reference recordings. This was due to differences in the wave form characteristics of the response at any two scalp recording sites. These findings demonstrate the difficulties which arise when comparing bipolar and reference recordings and emphasize the importance of designating electrode placement as well as polarity and latency when identifying components of the SER.

An important cause of these differences between bipolar and reference recordings was due to differences in the latency of individual components at any two scalp recording sites. The latency of the initial positive potential was the same at all recording locations. The subsequent negative and positive potentials progressively increased in peak latency from anterior to posterior recording locations in all subjects. In some subjects subsequent components were recorded with progressively increasing or decreasing peak latencies in the A–P plane.

There is general agreement that the SER initial positive potential is subcortical in origin (Broughton 1969; Goff *et al.* 1969; Cracco 1972). Some workers believe that this potential, the subsequent negative potential and an inconstantly appearing notch on the next positive component are all generated in thalamo-cortical axons (Allison 1962; Goff *et al.* 1962; Abrahamian *et al.* 1963; Allison *et al.* 1963; Rosner *et al.* 1963a, b). However, since the distribution of the initial positive potential is different from that of the subsequent potentials (Cracco 1972) and since the latter and not the former are characterized by progressive latency differences over the scalp and increased latencies during sleep, it seems probable that they are generated in different structures.

Differences in the latencies of certain SER components on the two sides of the scalp have been described in both adults and infants (Cernacek and Podivinsky 1966, 1971). Transcolossal transmission was suggested as the most likely mechanism. In coronal recordings performed on most subjects of this study, peak latencies of some components were greater over the hemisphere ipsilateral to the stimulated median nerve. In some subjects component peak latencies were different in coronal recordings taken from the same side of the scalp.

An increase in SER response latency with sleep has been reported (Goff *et al.* 1966). In this study progressive latency differences recorded in the A–P plane when subjects were awake persisted during sleep. With sleep, peak latencies of some components increased more at post-central than at pre-central recording locations in some subjects. Certain components vanished and others appeared. In some subjects, later components became more widely distributed in the A–P plane and showed progressive latency differences which were not apparent when the subjects were awake. These findings demonstrate the marked effect changes in level of consciousness can have on the SER and suggest that this variable must be excluded before differences in its wave form characteristics can be attributed to other causes.

Since some SER components are characterized by progressive latency shifts over the scalp in both the A–P and coronal planes, this gives the appearance of migratory activity or "traveling waves". The mechanism underlying the traveling of human alpha waves is not known (Rémond *et al.* 1969) and transcortical spread of electrical phenomena has been demonstrated only in rabbits with induced seizure discharges (Petsche and Rappelsberger 1970; Petsche *et al.* 1970). Traveling waves have been considered to be the result of either the algebraic summation of the activities of two or more discrete generators with slightly different phases (Rémond 1968) or to cortical potential fields becoming electrically active with slight time differences in a preferred direction (Petsche and Marko 1955). The rate of spread of some SER components over the scalp (several milliseconds from FP_1 to O_1) is greater than would be expected with transcortical spread. The fact that certain SER components were characterized by consistent A–P latency differences in some subjects but not in others and that rate of travel was affected by sleep favors subcortical mediation of these latency differences. Therefore, it seems that traveling waves of the SER may be the result of the algebraic summation of the activities of multiple cerebral generators activated non-simultaneously by thalamic, colossal or other afferent systems. SERs of similar latency have been recorded from separate, independent cortical areas in animals (Rose and Mountcastle 1959) and multiple somatic sensory cortical areas have been described in man (Penfield and Jasper 1954). These findings emphasize the complex nature of the neuronal substrate which underlies the SER in man.

SUMMARY

The effect of differences in techniques of recording somatomotor and somatosensory evoked responses (SERs) from the human scalp was studied by comparing bipolar and right ear reference recordings. Somatomotor responses were attenuated in close bipolar anterior–posterior (A–P) and biparietal recordings and were not recorded in sleeping subjects. SER components were often increased or reduced in amplitude, altered in peak latency, reversed in direction or vanished in bipolar leads compared with reference recordings. This was due to differences in the wave form characteristics of the SER at any two scalp recording sites. An important cause of these differences was due to differences in the latencies of individual components. Some components progressively increased or decreased in peak latency from anterior to posterior recording locations. In coronal recordings peak latencies of certain components were different at recording locations on the same side of the scalp and over the two sides. These progressive latency shifts in both the A–P and coronal planes gave the appearance of migratory activity. The mechanism which underlies this "traveling" is uncertain.

Progressive SER A–P latency differences recorded when subjects were awake persisted during sleep. In some subjects response latencies of certain components increased with sleep and they sometimes increased more at post-central than at pre-central recording locations. In some sleeping subjects certain components vanished, others appeared and later components sometimes became more widely distributed and showed latency differences which were not apparent when the subjects were awake.

These findings demonstrate the difficulties which arise when comparing bipolar and reference recordings and emphasize the complex nature of the neuronal substrate which underlies the SER in man.

RÉSUMÉ

ONDES PROPAGÉES DE LA RÉPONSE ÉVOQUÉE SOMATOSENSITIVE (SER) ENREGISTRÉE SUR LE SCALP CHEZ L'HOMME. EFFET DES DIFFÉRENCES DE TECHNIQUE D'ENREGISTREMENT ET EFFET DU SOMMEIL SUR LES RÉPONSES SOMATO-SENSITIVES ET SOMATO-MOTRICES

Chez l'homme, l'effet des différences de technique d'enregistrement sur le scalp des réponses évoquées somatomotrices et somatosensitives a été étudié en comparant des enregistrements bi-polaires et des enregistrements avec électrode de références à l'oreille droite.

Les réponses somatomotrices sont atténuées dans les enregistrements bi-polaires antéro–postérieurs rapprochés et bi-pariétaux et ne sont pas enregistrées chez les sujets endormis. Les composantes de la SER sont souvent d'amplitude accrue ou réduite, avec altération de la latence de pic, de direction inversée ou bien sont abolies sur les dérivations bi-polaires comparées aux enregistrements avec référence. Ceci est dû à des différences de caractéristiques morphologiques des SER entre deux points d'enregistrement sur le scalp. Une cause importante de ces différences est due aux différences de latence des composantes individuelles. D'avant en arrière, la latence de pic de certaines composantes augmente progressivement ou diminue. Dans les enregistrements coronaux, les latences de pic de certaines composantes sont différentes entre deux points d'enregistrement situés du même côté du scalp ou d'un côté à l'autre. Ces variations progressives de latence, à la fois dans le plan antéro–postérieur et le plan coronal, donnent l'apparence d'une activité migratoire. Les mécanismes qui soustendent cette "propagation" sont méconnus.

Des différences antéro–postérieures progressives de latence des SER, enregistrées quand les sujets sont éveillés persistent au cours du sommeil. Chez certains sujets, les latences de certaines composantes augmentent avec le sommeil et parfois elles augmentent davantage au niveau de points d'enregistrement post-centraux qu'à des niveaux précentraux. Chez certains sujets endormis, certaines composantes disparaissent, d'autres apparaissent et des composantes tardives deviennent parfois plus largement distribuées et montrent des différences de latence qui n'étaient pas apparentes quand les sujets étaient éveillés.

Ces données démontrent les difficultés qui surgissent lorsqu'on compare des enregistrements bi-polaires et des enregistrements de référence, et soulignent la nature complexe du substrat neuronique qui soustend les SER chez l'homme.

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