حرکت ارادی پتانسیل‌های ناشی از قشر حسی پیکری را تعدیل می‌کند

ای - ام - سدویک، وی‌چی ویراسینگه *

چکیده:

یک جزء اولیهٔ پتانسیل ناشی از حس پیکری که از قشر اولیهٔ حسی در انسان منشاء می‌گیرد، در طول حرکت ارادی انگشت تخفیف می‌یابد. عصب (Median) مدین در مچ دست تحریک می‌شد در حالی که شخص مورد آزمایش با همان دست حرکات تفکیک شده انگشت را انجام می‌داد.

اجزاء تحت قشری پتانسیل ناشی از حس پیکری تغییری نکردند ولی جزء قشری P25 تضعیف شده بود. آنالیز دوقطبی هم‌ارز نشان می‌دهد که این جزء از نواحی 1 و 2 قشر حسی سرچشمه می‌گیرد.

بنظر می‌رسد که قشر حرکتی (ناحیه 4) یک مسیر مهاری به سمت نواحی 1 و 2 دارد، که در طول حرکات ارادی فعال است.

کلید واژه‌ها: ۱ـ دریافت حسی ۲ـ حرکت ارادی

۳ـ قشر حسی مخ ۴ـ Evoked Potential

* بخش بالینی علوم اعصاب ، دانشگاه سوتهامتون - سوتهامتون - انگلستان

MODULATION OF SENSORY INPUT FROM THE HAND DURING VOLUNTARY MOVEMENT

E.M Sedgwick*
V. Weerasinghe

ABSTRACT

An early component of the somatosensory evoked potential, arising from the primary sensory cortex in man is attenuated during voluntary finger movement. The median nerve at the wrist was stimulated while the subject performed fractionated finger movements with the same hand.

Subcortical components of the somatosensory evoked potential were not changed but the P25 cortical component was attenuated. Equivalent diapole analysis shows this component originates in areas 1 and 2 of the sensory cortex.

It is proposed that the motor cortex (area 4) has an inhibitory pathway to areas 1 and 2 which is active during voluntary movements.

Key Words: ***1) Sensory Input*** ***2) Voluntary Movement*** ***3) Evoked potential*** ***4) Sensory Cortex***

Processing of somatosensory information

Somatic sensations provide us with an impression about the environment but the physiologic events underlying the process of somatosensory perception are not fully understood. We Know how a receptor transduces the stimulation into aneuralim-pulse and how this impulse is transmittedup to the cortex. But what happens after that is still not clearly Known. Present study attempts to investigate some aspects of human sensory processing.

The human hand is considered as an active sensory organ which explores environment rather than simply receiving stimuli passively. This active touch is

* Clinical Neurological Sciences Faculty of Medicine, University of Southampton, General Hospital, Southampton, UK Department of physiology, University of peradenya, Sri lanka

analogous to active "Looking" compared to mere "seeing". In order to use tactile sensation usefully it is important to analyse and integrate the activity of many neurones connected with receptors in skin, joints and muscle. The spatial properties of the object have to be reconstructed in the brain using temporal sequences of neural events. Nerve impulses from the hands initially reach the primary somatosensory cortex (SI) made up of Brodmann areas 3a, 3b, 1 and 2 posterior to the central sulcus. Sensory association areas are located behind SI in area 5 and 7. Cutaneous input from the hand is transmi -tted through dorsal column lemniscal system and relays in the ventrobasal complex of the thalamus. Thalamocortical tracts start from the ventroposterior lateral nucleus (caudal part) of the thalamus (VPLc) and supply the primary somatosensory cortex (mainly areas 3b and 1). The secondary somatosensory area (SII) lies lateral to SI and is also known to receive afferent projections from the thalamus. It could therefore be assumed that the processing of tactile information proceeds in parallel in SI and SII. Pons et al. (1992) have provided evidence from cortical ablation studies in monkeys that it is more likely that the sensory information processing occurs serially from SI to SII. These results were in contrast to the anatomic and electrophysiological findings in lower species like rabbits, cats and tree shrews in whom SI and SII process somatosensory information in parallel. This indicates that in higher primates there is an evolutionary shift to a new organisation in higher primates in which the processing of tactile information proceeds serially form SI to SII.

Manipulation is a feature acquired in the higher primates in the process of evolution of skilled manual control. It depends upon a truly opposable thumb, the development of glabrous skin as a sensory organ, a motor control system that allows fractionated finger movements and the precision grip of the index finger, which is differentiated from the power grip of the whole hand. Manipula -tion is an interesting situation because both sensory and motor cortical areas are involved in action at the same time. Therefore it is pertinent to ask the question - how does the sensory cortex respond to an afferent volley from a peripheral nerve when both sensory and motor cortical areas are engaged in manipulation?

METHODS

Evoked potentials are the signals recordable from the scalp following a stimulus; Dawson (1947) was the first to use superimposition technique to study evoked potentials.

Nowadays, some form of signal averaging technique is used to extract this evoked response. The stimulus is presented many times and the EEG signals for the duration of interest immediately following are summed and then diveded by the number of

presentations to obtain the average evoked number of presentations to obtain the average evoked potential. This technique makes significant improvement of the signal - to - noise ratio and permits the recording of very small evoked potentials. When a large number of trials are averaged, time locked signal is enhanced whereas random noise is reduced. Generally for somatosensory evoked response averaging, 500-1000 sweeps are necessary. When a large number of sweeps are collected noise is reduced according to the following relationship:

Increase in SNR (or reduction in noise) α number of sweeps.

Modification of Sensory input during movement

Movement is known to attenuate cutaneous perception from the moved limb. This inhibition is known as "gating" Schmidt et al. (1990) have shown that the tactile sensations evoked during intraneural microstimulation within the median nerve is reduced during movement. This confirms the earlier observation [10] that the perception of an electrical stimulus applied to the finger tip is reduced 100 msec before and during an active flexion of the fingers. Several other studies[6] have also shown that the sensory threshold is increased during movement. This gating mechanism can either takes place at lower levels or at the cortical level. It has been demonstrated in animals that the transmission of afferent information along the somatosensory pathway is gated during movement of the corresponding body part[10,11,15].

In man, somatosensory evoked potentials (SEPs) have been used to study the movement gating of sensory input[7,8,23]. When the median nerve is stimulated at the wrist, the short latency potentials can be recorded along the somatosensory pathway which are known to be generated in the subcortical areas. Since anatomical studies show that corticospinal tract terminals are found in the subcortical sensory relay nuclei, it could be argued that the attenuation of cortical potentials may be the result of a subcortical gating effect. In animals, it has been shown that the ascending sensory volley is gated at the dorsal horn of the spinal cord [5], dorsal column nuclei[10,11,15], and thalamus (Tsumoto et al. 1975). Cole and Gordon (1983, 1992) have shown that the stimulation of the sensorimotor cortex produces inhibtion, facilitation or mixed effects of the cuneate nucleus of the cat. However in man the effect of voluntary movement on the Subcortical SEPs is not very clear. Some studies have shown that there is a modification of subcortical SEPs in humans [1,7,18,27] whereas others have shown that there is not [7,8,18,28].

Basis of evoked potentials

Physiological activity of the human sensory cortex can be studied using scalp recorded averaged evoked potentials. The

physiological activity of neurones and other biological structures is accompanied by electrical changes which can cause electric currents to flow in the cytoplasm of the cell and also in the surrounding conducting fluids. Cellular activity may thus be investigated by recording the potentials appearing across the membranes, or in extracellular fluids. Single neuron recording techniques have been used to study the biophysical properties of nerve cells in experimental animals. But in intact human brain a noninvasive method of recording from the surface of the scalp has a much wider practical use. Scalp recorded EEG (electroencephalogram) is a good example for this. Spontaneous electrical activity of the brain was first observed by caton in 1875. He investigated the activity of the brains of cats, monkeys and rabbits using nonpolarisable cortical electrodes connected to a galvanometer with optical magnifica -tion. These early studies were done in animals and it was in 1929 that Hans berger published the first report of the EEG of man. Adrian and Matthews repeated and confirmed these experiments in 1934. Scalp recordings represent synchronised activity of a large number of neurons or nerve fibres. Most of the evidence available at present suggests that the scalp recorded electrical potentials are due to excitatory or inhibitory postsynaptic potentials developed by the cell body and large dendrites of pyramidal neurons. Non-synchronous activity, eg. axonal impulses, stellate cell activity does not appear in EEG.

Modification of sensory input during movement

What are the cortical changes in humans? Results of previous studies are not clear. When the median nerve is stimulated at the wrist, after the initial spinal and subcortical events the first cortical activity is recorded best over the contralateral parietal scalp area as a negative wave around 20 msec (N20). Most of the previous gating studies[7,8,26,30] have shown that the N20 is unchanged during movement though there are a few contradictory reports from other studies [1,27]. N20 is known to be generated by a tangential dipole in area 3b in the posterior bank of the Rolandic sulcus in the primary somatosensory cortex [4]. Corresponding positivity is recorded over the frontal areas as P20. Subsequent to these potentials, a localised positivity can be recorded over the central sulcus which Allison et al. (1991) described as P25. There is a controversy about the generators of these positivities. Allison et al. (1991) have provided evidence from scalp as well as cortical recordings to suggest that P25 is generated by a radial dipole in area 1 in the primary somatosensory cortex. On the other hand, Desmedt et al. (1987) have described a prerolandic P22 positivity generated by a radial dipole in area 4 of the primary motor cortex after a delay of 1-2 msec after N20. Although previous

gating studies show evidence that the N20 is not changed during movement, the effect on P20 is not very clear. If it is accepted that N20 and P20 are both generated by the same source, P20 should not change during movement. Several studies on gating [7,8] have shown that frontally recorded P22 is attenuated but their results do not show any P20. They also show a parietally recorded P27 which is attenuated during movement. Is P22 the same as P20? If so, how could it change when there is no change in N20 provided the theory that they are both due to the same generator is valid? If p22 is similar to p25 (generated by a radial dipole) how could one explain P27? These questions remain to be answered in order to clearly understand the generators of early cortical SEPS. One way to address these issues is to study the spatial distribution of SEPs attenuated during movement which is attempted in this study.

In previous studies on gating various types of movements have been used, viz. adduction of the thumb, abduction of the digit 5, flexion and extension of the thumb or all of the fingers and isometric contraction. In the present study we used fractionated finger movements and manipulatory movements of the hand.

Source analysis

When a nerve is stimulated peripherally, the potentials generated inside the brain are recorded over the scalp using electrodes placed at suitable locations. Such scalp potentials are characterised by their temporal and spatial properties. Methodologically EEG and evoked potential waveforms have a very high degree of temporal accuracy although spatial accuracy depends on the number of electrodes used and their locations. It is not always reasonable to assume that sources are generated underneath electrode locations. In order to improve the spatial accuracy, mapping of potential distribution over the scalp surface can be performed. this method interpolates potential distribution in between electrode locations. Maps can be drawn at any latency and the selection of a latency to map appropriately to model the underlying source activity is difficult to decide.

Temporal variation of sources may not be directly equivalent to the potential peaks and troughs. Therefore next logical step in topographical analysis is to calculate underlying source activity using an appropriate source analysis method. This "inverse solution" could be performed using physical laws governing dipole models. For the same scalp potential distribution there may be more than one solutions with sources different in location and orientation. This is the principle of non-uniqueness of the inverse solution. But by applying spatial and other constraints based on underlying physiology one can improve on the solution so as to arrive at a unique solution. In this respect, temporal evolution of the source

activity is of paramount importance.

The dipole is not a real entity but an Equivalent Dipole representing the electrical activity of a population of neurons. Instead of recording merely a waveform, an equivalent dipole has 7 measureable parameters: XYZ spatial location, amplitude, orientation, time (and evolution in time) and finally, variance. This latter is a statistical property expressing how well the Equivalent Dipole models the recorded activity(**24**).

Resting subcortical sep

The first activity recorded after stimula -tion of the median nerve was P9 with a latency of 9.7±0.7 msec. This corresponds the compound action potential in the brachial plexus. In the cervical electrodes this activity was followed by two negativities, N11 and N13. N13 peak was more promi -nent and had a latency of 12.6±0.8 msec. Scalp electrodes recorded these peripheral nerve and spinal cord activites as positive far field waves, P9, P11. The wave seen at 13 msec has 2 components. One is due to synaptic activity in the dorsal horn of the spinal cord which acts as a horizontal dipole with negativity posteriorly and positivity anteriorly [20]. The other is the ascending volley in the dorsal column. This is seen as a far field positivity by scalp electrodes. These were followed by P14 wave with a latency of 13.7±0.8 msec, believed to be generated in the dorsal column nuclei and medial lemniscus. In some subjects P13 and P14 were merged together.

These sub cortical waveforms are followed by a large negative wave, the characteristic N20 peak which had a mean latency of 18.4±0.8 msec.

Effect of voluntary movement and manipulation

Compound nerve action potential following stimulation of the fingers recorded over the median nerve at the elbow did not change during voluntary finger movement.

The amplitudes of left median nerve SEPs during voluntary finger movement (FM) and manipulation (MN) were not different from those during rest up to and including N20. ($p>0.1$; wilcoxon ´s paired rank sum test, $n=9$).

It is concluded that voluntary finger movement does not ¨gate¨ or modify sensory transmission in the subcortical Synaptic relay nuclei.

Resting cortical sep

Since in the previous experiment it was shown that the subcortical activity is not modified during movement next logical step was to study the cortical potentials. Our working hypothesis is that in the median nerve SEP waveform, potential peaks up to about 25 msec are generated only in the primary somatosensory cortex. The contrala -teral parietal cortical electrodes records a

conspicuous negativity (N20) which marks the arrival of the efferent volley in the primary somatosensory cortex. Next activity recorded was a parietal P25 with a mean latency of 25.6 ± 0.8 msec. This was mainly a contralateral parietal cortical activity. In some individuals this was recorded parietally but close to the central sulcus.

A frontal N30 with a mean latency of 30.3 ± 1.1 msec and parietal P30 with a latency of 31.8±1.3 msec were the other two componets before all the electrodes showed P45 with a mean latency of 43.1±0.8 msec.N30 was recorded from the frontal scalp both contralaterally and ipsilaterally. Maximum amplitude of N30 was recorded from Fz electrode. The next experiment looked at the modification of these components during voluntary movement and manipulation.

Modification of cortical potentials during manipulation

Up to 23 msec after the stimulus there is no change in the SEP waveform induced by volutary movement. Thus P15, N18, N20 and P20 peaks do not show any change. After 23 msec two waveforms start to deviate. P25 and N30 peaks show amplitude changes without any change in latency the P25 shows up to 58% attenuation during movement. It could be concluded that the earliest cortical peak subjected to any modification during voluntary movement is P25. Manipulation seems to behave similar to movement in that it does not show any modification up to 23 msec and P25 shows a marked attenuation (amplitude reduction of 65%). Interestingly, figure writing does not seem to modify SEP waveform until about 40 msec.

Evidence for an inhibitory Corticocortical pathway from area 4 (motor) to area 1 (sensory)

The most consistent potential we recorded was P25 as a localised potential in the contralateral parietal region. During movement this potential showed a statistically significant attenuation. Difference waveforms showed that there is a significant difference between resting and movement waveforms around 25 msec. In all the subjects P25 was attenuated though the degree of attenuation varied among different subjects. P25 potential could be equivalent to P22 reported by Desmedt and Cheron (1981b). Both these potentials showed attenuation during movement although there generators are not yet resolved. P22 described by Desmedt is claimed to be generated in area 4 whereas P25 whown by allison et al (1991) is claimed to be in area 1. Desmedt and Cheron (1981b), Cheron and Borenstein (1987); Cohen and Starr (1987) showed another positivity in the parietal region which they

called P27. Their results indicate P27 is significantly attenuated during movement. From the size of amplitudes and consistency it appears that our P25 potential is more likely to be similar to P27.

Spatial distribution of P25 suggests a generator in the contralateral parietal region. Spatial maps drawn using detailed montages show that a deep positivity located in the C4 area in all subjects. Transcortical recordings have strongly suggest that P25 is produced by a radially oriented generatory located in the anterior crown of the postcentral gyrus in area 1 of the somatosensory cortex, in a region 1 cm medial to the region of largest area 3b potentials.

Anatomical studies have shown that the main output connections of area 4 to the sensory cortex are to areas 3a, 1 and 5 but not 3b. Thus during movement when area 4 is active it could modify area 1 activity but not area 3b activity.

Nelson (1985) has studied sensorimotor cortical responses to vibrotactile stimuli during initiation and execution of hand movements in monkeys performing goal-oriented motor tasks. Of the 111 neurons that showed an increase in firing rate withing 30 msec of stimulus onset, 48% maintained a discharge rate greater than that observed during the hold phase of the task until movement onset. This unit was recorded deep in the penetration and was adjacent to cells receiving cutaneous input and was located in area 3b. These neurons did not significantly decrease firing rate prior to movement onset. In contrast to area 3b neurons, most neurons in areas 1 and 2 that had short-latency excitatory responses to the vibrotactile stimulus also showed a significant decrease in firing rate 60-80 msec prior to movement onset. He further has recorded from 24 units in area 4 during the performance of the task to compare the timing of responses in the somatosensory cortex with the timing of activity in area 4. There was a temporal correlation between the increased activity in area 4 neuron and the decrease in firing rate in area 1 neurons. This is further physiological evidence for the existence of an inhibitory pathway from area 4 to area 1 (and possibly to 2) but not to area 3b. If it is assumed that N20/P20 activity is due to a dipole placed in area 3b, during movement there should not be any modification in this activity because of lack of connection from area 4 (motor) to area 3b. However, if P25 is due to a dipole placed in area 1 it could be attenuated during movement because of the activation of the inhibitory pathway from area 4 to area 1.

Another line of evidences is reported from neuromagnetic measurements carried out by Rossini et al. (1989) during median nerve stimulation at the wrist in complete relaxation and during active contraction of the hand muscles. Their results showed a slightly posterior and shallower localisation of the substraction map compared to the

one in the resting state indicating that the gating of sensory information during voluntary movement presumably took place at the cortical level (SI cortex) at a depth compatible with the crown of the post-central gyrus and the parietal convexity (areas 1 and 2). The effect of transcranial magnetic stimulation on median nerve somatosensory evoked potentials [19] was also to change the excitability of the somatosensory cortical neurons involved in producing the P25 but not those generating N20. SEP to median nerve stimulation in patients with focal lesions of the prefrontal cortex also showed increased P26 amplitude without any change in N20 amplitude[31].

In summing up the results of the experiments of SEP during movement in the light of the above discussion on previous studies, it could be concluded that up to 25 msec after stimulation of the medinan merve, the cortically generated activity is processed in the contralateral primary somatosensory cortex which is selectively attenuated by corticocortical pathway from motor (area 4) to sensory cortex (area 1) when there is simultaneous movement. Normally a large number of afferent impulses ascends in the afferent systems and the somatonensory cortex is continually bombarded with a load of information. When a precise movement is occurring, most of the irrelevant information should be interrupted in order to improve the signal-to-noise ratio so that only the relevant information will be processed by the sensorimotor cortex. It is difficult to account for the mechanism of this screening process. But the selective attenuation of P25 without changing N20 supports the theory that the afferent volley reaches the primary somatosensory cortex and the selective screening of information occurs at the cortical level.

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