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Somatosensory Evoked Potentials in Schizophrenia
A Lateralisation Study

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The SSEPs obtained from 19 schizophrenics defined by RDC, DSM-III and PSE criteria were compared with those from a control group of healthy volunteers. Previous findings of an abnormal lack of lateralising response in schizophrenic patients were not replicated. No significant difference in either amplitude or morphology between the traces obtained from the two groups were recorded. Ipsilateral and contralateral latencies for stimulation of the left and right index finger showed no significant difference in peak latency for any component between patient and control group. When mean peak-to-peak amplitudes were plotted the contralateral component was always greater in amplitude than the ipsilateral one. An objective measure of the degree of lateralisation, the percentage lateralisation quotient, showed no lateralisation differences between the patient and control groups. A case of myogenic contamination of ipsilateral components was observed calling into doubt findings where no temporal region monitoring has been performed.

In 1970, Hardin & Castelucci hypothesised that the latency difference between selected components of the ipsilateral and contralateral somatosensory evoked potential (SSEP) could be interpreted as representing commisural delay. Salamy (1978) performed SSEP recordings over a wide age range, employing a vibrotactile stimulus and recording electrodes over both somatosensory 'hand areas' referenced to the vertex (Cz) electrode, and observed an ipsilateral response of longer latency and lower amplitude than the contralateral response. This difference in latency decreased with age and was said to reflect maturation in somatosensory commisural transmission.

The origin of the ipsilateral somatosensory evoked response remains open to question. Although it may arise from an afferent volley originating from the contralateral cortex mediated via an interhemispheric pathway (the corpus callosum), the potentials could equally result from volume conduction from contralateral cortex, or by direct uncrossed pathways arising in the thalamus. Rosenthal & Biggelow (1972) showed a 20% thickening of the corpus callosum in a post-mortem study of schizophrenic subjects.

Jones & Miller (1981), inspired by the previous studies, set out to investigate the relationship between ipsilateral and contralateral SSEP components in schizophrenics. Using a recording protocol similar to that employed by Salamy, they found that the ipsilateral response in controls was small in amplitude and delayed in onset compared with the contralateral response. In schizophrenics, however, no delay was seen on the ipsilateral hemisphere, and it was suggested that ipsilateral pathways originated in the brainstem, rather than in a non-conducting corpus callosum. It was further proposed that schizophrenia might arise from agenesis of the corpus callosum.

In criticism of this work, Connolly (1982) observed that the technique employed would only assess the small number of myelinated callosal axons. Generalisation from this small group to conclusions of total callalosal block were felt to be ill-advised. However, in view of the potential importance of these findings, Shagass *et al* (1983) carried out a replication study after certain methodological faults had been removed. In Jones & Miller's original study, SSEPs had been recorded with a vertex (Cz) electrode used as reference. This electrode can be active, and a spurious ipsilateral response could therefore be recorded (Desmedt & Brunko, 1980). For this reason Shagass *et al* (1983) employed linked ears as well as a vertex reference site. Their findings showed contralaterally higher amplitude SSEPs in both controls and schizophrenics. They found no difference in the latencies between the ipsilateral and contralateral SSEPs in either group.

These and further conflicting studies (Tress *et al*, 1983; Fenwick *et al*, 1983) encouraged Cooper *et al* (1985) to investigate the matter more. SSEPs arising from a complex vibrotactile stimulus to the forefingers, similar to the stimulus employed by Jones & Miller (1981), were further studied. The stimulus, mainly of movement and to a lesser extent of touch, was modified to give an optimal tracing. Eight patients with affective disorder (three depressive and five manic) all had the expected contralateral hemisphere lateralisation as did 12 out of 15 normals. Three normals and three schizophrenics had a loss

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of lateralisation of the evoked response to stimulation of one hand and normal lateralisation when the other hand was stimulated (unilateral abnormality). Ten out of 21 schizophrenic patients had an abnormal lack of lateralisation response whichever hand was stimulated (bilateral abnormality). The wave forms were characterised by major positive peaks with a latency of approximately 30 ms and 45 ms. In control groups, the response recorded ipsilaterally was invariably too small to identify peaks reliably. Judgements on the degree of lateralisation were made subjectively by visual inspection. Lateralisation characteristics were evident only for the earliest part of the responses and at longer latencies there were often similarities in amplitude between the wave forms recorded over the two hemispheres.

It was claimed that the results could not be compared directly with those elicited by the mainly vibrotactile stimulus of Shagass *et al* (1983), or indeed, with those elicited by the commonly used artificial electrical stimulation of the median nerve (Fenwick *et al*, 1983). A further study that might replicate these findings was urged by the authors.

The following study was carried out using digital electrical stimulation, with the results recorded on the Biologic Brain Atlas III system to facilitate more detailed topographical analysis of the data.

Method

A group of 19 schizophrenics, both in-patients and out-patients, were compared with a group of normal healthy volunteers. Patients were interviewed using a modified form of the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1975) and included in the study if they fulfilled Research Diagnostic Criteria (RDC; Spitzer *et al*, 1975) and DSM-III (American Psychiatric Association, 1980) criteria for schizophrenia. Basic demographic data and clinical details, such as the duration of illness, were collected. A case-note review, using the Present State Examination (PSE) symptom check-list (Wing *et al*, 1974), allowed all patients to be classified as having nuclear schizophrenia.

Twenty electrodes were placed over the scalp according to the international 10–20 system at points Fp1,Fp2,F3,F4, F7,F8,Fz,Cz,Pz,T3,T4,T5,T6,C3,C4,P3,P4,O1,Oz and O2 with linked earlobe electrodes as the reference. The stimulus was produced by ring electrodes placed on the distal phalanx of the index finger, with cathode proximal to the anode. An earth lead held by a velcro strip was attached on the forearm; some light abrasion of the skin with the velcro beforehand produced a low resistance. Subjects were placed in a prone position, made warm and comfortable and asked to remain still with their eyes closed but to remain awake. Subjective threshold of the stimulus was ascertained and a current level three times this threshold used to elicit the SSEP. Stimulus duration was 200 μs and current levels were typically 3–6 mA.

Both right and left index fingers were stimulated in each subject, the order being on a random basis between subjects. Stimulation rate was 2.7 per second and 1024 stimulus runs were averaged. Careful monitoring of the input signal with automatic artefact rejection was maintained to ensure artefact-free recordings.

The Biologic Brain Atlas III system with built-in amplifiers was employed for recording. Gains were set at 50 000 with low-frequency filters at 10 Hz and high-frequency filters at 1500 Hz (-3dB points, roll-offs 12 dB octave). Twenty channels of data were recorded and stored on a 10-megabyte disc cartridge for analysis. The system allows generation of data in the form of isopotential colour contour maps known as brain maps. This facility allows topographical data to be readily compared and although this material is not shown here it was used extensively with the raw waveforms as an aid to analysis.

Results

Recordings were obtained from 13 normal control volunteers (six men and seven women; mean age 29.9 years, range 19–57 years) and 19 schizophrenic patients (12 men and seven women; mean age 35.5 years, range 17–68 years). There was a positive family history of schizophrenia in three patients. The average time since onset of first illness was 10 years (range 2–40 years) and patients had been admitted to hospital for an average of 59 months (range 3 months–24 years). All patients were currently receiving drug treatment.

In the control group, of the 26 possible recordings obtainable (right and left index fingers), 24 were actually achieved. Two recordings were lost from two individuals with right-hand dominance from whom no recognisable potentials to stimulation of the left index finger were discerned, but who subsequently showed normal responses to median-nerve stimulation.

In the schizophrenic group, of the possible 38 recordings, 29 were actually achieved. In four patients, no recognisable components to digital stimulation could be discerned in either hand. Each of these patients subsequently produced clearly recognisable potentials to median-nerve stimulation. One patient had received major injuries to his left hand in a machine accident which had resulted in the loss of his left index finger.

Initial subjective analysis of the waveforms obtained from the parietal area resulted in the recognition of the 'W response' described by Desmedt & Brunko (1980) and Desmedt & Cheron (1980) – the familiar P17-N20-P27-N35-P45 components obtained by median-nerve stimulation. Although mean peak latencies are longer for digital than median-nerve stimulation, we adopted the nomenclature for responses to the latter for ease of communication and comparison.

Table I shows the actual mean peak latencies of components of contralateral parietal hemisphere.

Other consistent components seen in both patient and control groups were frontal N17-P20-N30 and centroparietal N19-P22-N35-P45 potentials.

Utilising the software of the brain-mapping system, group average waveforms were calculated for the control group

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TABLE I

Mean peak latencies of components of contralateral parietal hemisphere in controls and schizophrenics with right and left index-finger stimulation

	Peak latencies: ms									
	P17		*N20*		*P27*		*N35*		*P45*	
	mean	*(s.d.)*	*mean*	*(s.d.)*	*mean*	*(s.d.)*	*mean*	*(s.d.)*	*mean*	*(s.d.)*
Controls										
Right index finger	17.0	(2.0)	22.8	(1.6)	29.4	(2.5)	37.7	(3.4)	46.8	(4.0)
Left index finger	17.1	(1.2)	22.3	(1.4)	28.2	(2.5)	36.1	(3.0)	44.6	(3.9)
Schizophrenics										
Right index finger	17.9	(1.7)	23.7	(1.7)	29.8	(1.8)	36.9	(5.6)	45.7	(4.5)
Left index finger	17.6	(1.0)	22.9	(1.4)	30.2	(2.6)	36.9	(3.6)	44.6	(3.8)

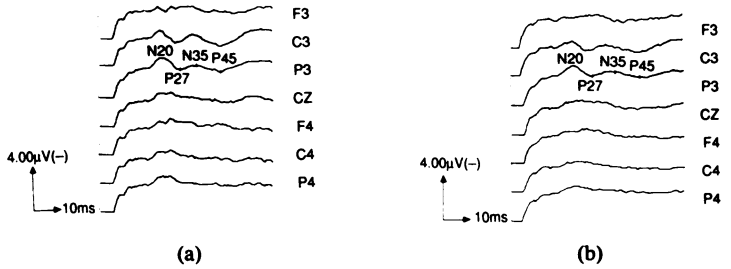


FIG. 1 Stimulation of the right index finger: group mean data for controls (a) ($n = 13$) and patients (b) ($n = 15$).

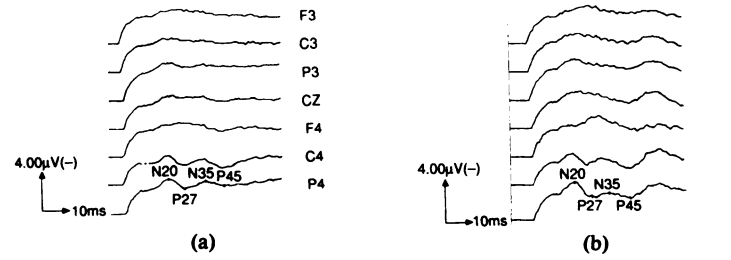


FIG. 2 Stimulation of the left index finger: group mean data for controls (a) ($n = 11$) and patients (b) ($n = 14$).

and schizophrenic patient group. The most relevant waveforms are illustrated in Figs 1 and 2. In order to identify any significant group differences, Student's *t*-test was performed using the brain mapper on every data point of every channel of the two groups. The subsequent results supported the subjective impression from the waveforms that there was no significant difference between the groups ($P > 0.05$).

Peak latency (Table I) and peak-to-peak amplitude data (Fig. 3) were recorded from the waveforms obtained at electrode sites P3 and P4 for closer comparison with previous studies (Jones & Miller, 1981; Cooper *et al*, 1985). Statistical analysis using the *t*-test revealed no significant differences in any peak latency between patient and control group ($P > 0.05$). Additionally, paired *t*-testing of interhemisphere peak latency revealed no significant differences of any parietal component ($P > 0.05$). In both patient and control groups, the post-rolandic contralateral components were always of greater amplitude than their ipsilateral counterparts, with contralateral N20-P27 consistently the highest amplitude component for both groups. Comparison of the two groups was made by *t*-test of the group means of peak amplitude; for both right and left index-finger stimulation there was no significant difference in peak amplitudes between groups ($P > 0.05$).

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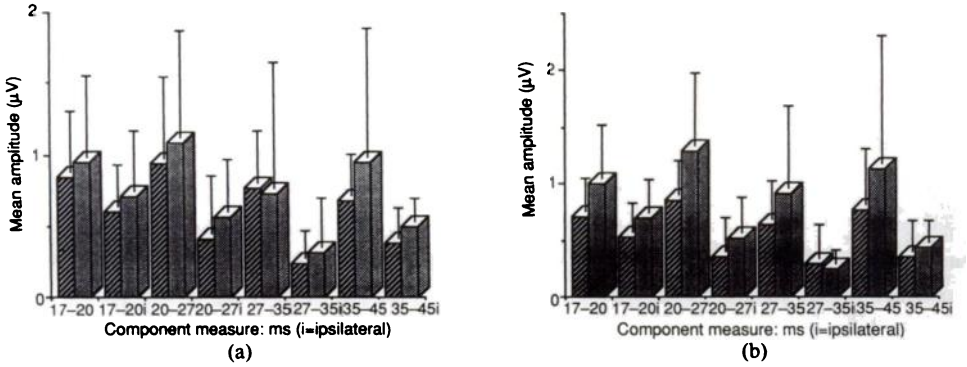


FIG. 3 Mean amplitude of parietal components in controls (▨) and patients (▩); (a) right index-finger stimulation, and (b) left index-finger stimulation (error bars = 1 s.d.).

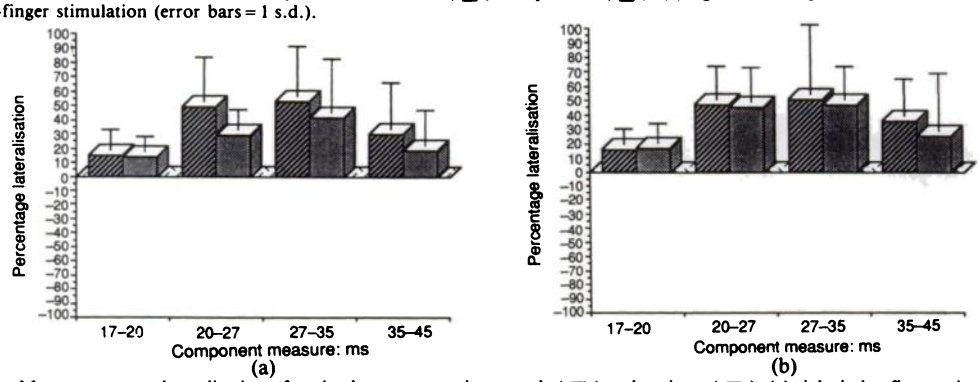


FIG. 4 Mean percentage lateralisation of parietal components in controls (▨) and patients (▩); (a) right index-finger stimulation and (b) left index-finger stimulation.

In order to provide some objective assessment of the degree of lateralisation for each component, the following measure was used:

$$\frac{(\text{Contralateral amplitude} - \text{Ipsilateral amplitude})}{(\text{Contralateral amplitude} + \text{Ipsilateral amplitude})} \times 100\%$$

This formula provided a score which if positive (>0) indicated a degree of contralateral amplitude lateralisation and if negative (<0) indicated ipsilateral amplitude lateralisation. The mean percentage lateralisation for each component is shown in Fig. 4. There was no significant difference in lateralisation between the patient and control groups ($P>0.05$). The mean lateralisation percentages were positive, indicating contralateral lateralisation for each component in both patient and controls.

At other scalp locations, centroparietal components (N19-P22-N35-P45) showed similar lateralisation features to those seen parietally in both patient and control groups. At frontal locations, components (N17-P20-N30) were bilaterally distributed in both groups. P17, although not clearly apparent on the group mean waveforms, was a consistently clear component in both patient and control groups. This component was recognisable in most recordings at every scalp location. These findings are in agreement with previous authors (Goff *et al*, 1977; Kakigi & Shibasaki, 1984; Desmedt & Cheron, 1980; Desmedt & Brunko, 1980; Duff, 1980).

One problem of comparing group mean data is that if an individual patient differed significantly from the control group, this fact may be lost when incorporated into a non-significant larger mean. To monitor for this, data from each schizophrenic subject were compared with the group mean of the control population using a Z-score technique utilising the Brain Atlas software in the manner of Duffy *et al* (1981), where:

$$Z = \frac{(x - m)}{s}$$

(x = individual subject data, m = control group mean data and s = control group standard deviation. Z is given in standard deviations from the control group.)

Each schizophrenic subject produced Z scores of less than two standard deviations across the entire waveform and at every scalp location with the exception of one subject. Patient SXI produced a waveform which on subjective inspection appeared to be quite bilaterally symmetrical across the entire waveform particularly at parietal locations (Fig. 5a). The subsequent Z score indicated a statistically significant deviation over the ipsilateral centroparietal and temporal areas. The highest area of deviation however appeared to be the temporal derivations. Re-inspection of the individual waveforms revealed a higher level of electromyelographic (EMG) artefact in the ipsilateral than the contralateral temporal region (Fig. 5b).

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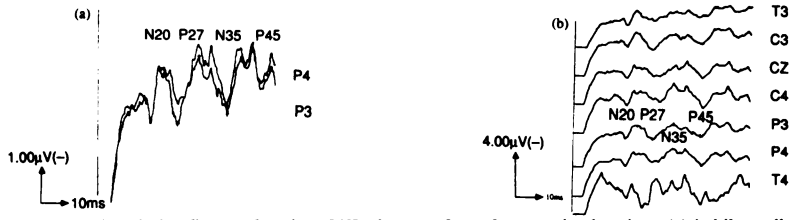


FIG. 5 Stimulation of the right index-finger of patient SXI: the waveform from parietal regions (a) is bilaterally symmetrical, while the waveform from the temporal region is asymmetrical as the result of EMG artefact.

Interpretation of the parietal component amplitudes without the benefit of knowledge of the extent of this asymmetric artefact could produce spurious conclusions. The effect of myogenic enhancement of the components on the ipsilateral hemisphere cannot be excluded (Bickford *et al*, 1964; Calmes & Cracco, 1971).

Discussion

Cooper *et al* (1985) felt that the nature of the stimulus employed in their study of schizophrenia was of major importance. Such a stimulus would achieve consistency with previous authors in this field who had employed either vibrotactile or similar mechanical stimulation (Jones & Miller, 1981; Tress *et al*, 1983). It was also felt that such a stimulus would be less distressing and more easily tolerated by schizophrenics than conventional electrical stimulation. However, there are a number of inherent problems in the design of vibrotactile speaker systems (Jones & Miller, 1981; Shagass *et al*, 1983; Cooper *et al*, 1985). A considerable audiostimulus is delivered to the patient which needs to be masked. The speaker cone is a low inertia system and subjective sensation is modified by pressure exerted from the stimulated digit. The duration of the stimulus is relatively long compared with constant current stimulation of electrical square-waves (10 ms v. 100 μs) and this may result in poorer formed potentials.

Most studies in the literature other than those investigating schizophrenics employ electrical stimulation to nerve trunk or digit, and it is therefore difficult to generalise from findings using the vibrating stimulus and to compare them with wider studies usually of larger groups of patients and controls. Cooper *et al* (1985) felt that their findings were probably stimulus-specific. However, since whatever mediated the ipsilateral cortical response of short latency (other than simple volume conduction) has to be the result of 'hardwired' thalamic and/or cortico–cortical tracts, the nature of the stimulation should be relatively inconsequential, unless we challenge existing anatomical, physiological and electrophysiological knowledge.

The correlation of SSEP abnormalities with proprioceptive deficits suggests that the SSEPs are mediated via dorsal column/medial lemniscal tracts (Halliday & Wakefield, 1963), with the small signals detected in lateral spinothalamic tracts appearing to contribute nothing to the scalp potential (Suzuki & Mayanagi, 1984). These facts coupled with existing knowledge of the cytoarchitecture of the primary somatosensory cortex lead us to conclude that cerebral locations of the SSEP are unlikely to differ widely, whether the stimulus is electrical or vibrotactile, if only digital nerves are stimulated (Hyvärinen, 1982; Kakigi & Shibasaki, 1984); the comparative morphology of tactile and electrical evoked responses would appear also to differ little (Pratt & Starr, 1981).

Given these factors and the relatively poor responses obtained on the vibrotactile stimulus, it was decided to standardise on electrical digital stimulation. It should be noted that the dominant components of Cooper's waveforms were positivities at approximately 30 ms and 45 ms which would equate well with the two dominant components obtained in this study, namely P27 and P45.

The final outstanding consideration was the tolerance of the electrical stimulus by the schizophrenic patients. During a pilot study, patients were able to tolerate both median and digital electrical stimulation at adequate levels (three times subjective current for digital stimulation and motor twitch of the thenar muscle for median nerve) for sufficient periods of time to allow adequate tracings to be obtained. Additionally, there were no topographical changes observed between stimulation rates of 0.7 Hz, 2.7 Hz and 4.7 Hz. These findings are broadly in agreement with those of Pratt *et al* (1980) who demonstrated that for both mechanically and electrically evoked SSEPs, stimulus rates of up to 4 Hz may be used without compromising the short-latency cortical components.

The results of the study are summarised as follows:

(a) There was no significant difference in either amplitude or morphology between the traces obtained from the control and patient group recorded from 20 scalp locations.

(b) There was no significant difference in peak latency comparing patient with control, and ipsilateral and contralateral latencies for right and left index-finger stimulation.

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(c) The mean peak-to-peak amplitudes for parietal components were always greater for contralateral components compared with the ipsilateral counterpart.
(d) The percentage lateralisation quotient showed no lateralisation differences between the subject and control groups.
(e) When the data for each schizophrenic patient were compared with the control group data, no differences were found except in one case of asymmetric temporal EMG artefact.

The present study obtained adequate recordings of the SSEPs using a conventional digital nerve stimulus, and failed to replicate Cooper *et al*'s (1985) finding of a loss of lateralisation in a subgroup of schizophrenics. They tentatively hypothesised that their findings arose from abnormality in the corpus callosum. It has been established, however, that there are no direct callosal connections between the primary somatosensory hand areas in monkeys (Jones & Powell, 1969). Supporting evidence for this in man is provided by magnetic recordings to transcutaneous stimulation of the median nerve (Okada *et al*, 1984), which have revealed no ipsilateral components. Studies of regional cerebral blood flow (Foit *et al*, 1980) also have failed to reveal any ipsilateral components. This suggests that ipsilateral electrical components are volume conducted from the contralateral hemisphere. This is further supported by our lack of significant latency difference between ipsilateral and contralateral components.

An abnormality of the corpus callosum would not then allow one to predict theoretically a loss of lateralisation in the SSEP and our findings are therefore not surprising. It is disappointing that the present study was not able to confirm the presence of a neurophysiological marker of schizophrenia, as described by Cooper *et al* (1985). It does, however, raise a note of caution to experimenters examining lateralising events or topographical data generally, that careful monitoring of EMG must be undertaken. Unfortunately, recordings from the temporal region were not carried out during the study by Cooper *et al* (1985), so the question as to whether myogenic activity in the temporal region produced an apparently abnormal result for their schizophrenic group, has to remain unanswered.

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