Brain (1975) **98**, 113–128

SOMATOSENSORY CEREBRAL EVOKED POTENTIALS AFTER VASCULAR LESIONS OF THE BRAIN-STEM AND DIENCEPHALON

BY

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DEFINITE syndromes have been identified by clinical means among the vascular lesions of the brain-stem and diencephalon and their pathological correlates have been extensively studied. However, the pathophysiological mechanisms of the accompanying sensory signs and symptoms are little known. Problems may also arise when attempting to evaluate sensation in the so-called locked-in patients (Plum and Posner, 1966) or when trying to assess objectively the specific deficits for diagnostic and prognostic purposes. Some of these problems can be approached by applying to intact patients electrophysiological procedures which are appropriately designed for testing various brain-stem reflexes (cf. Kimura, 1973) or corticipetal pathways (cf. Halliday, 1967; Desmedt, 1971; Desmedt and Noël, 1973). The present paper is concerned with the functional assessment of the central somatosensory pathway in patients with several types of vascular lesions.

MATERIAL AND METHODS

Seven patients were selected on the basis of a fairly complete clinical report documenting a localized vascular lesion in the brain-stem or diencephalon. They were investigated in the acute stage and also in most cases after a delay which varied between six and twenty-two months. The patients were arranged in three groups as shown in Table I. Six of the patients are still alive and no pathological evidence is therefore available. The patients in the acute stage were transferred on a couch with due precautions from the intensive care unit of the Department of Medicine of St. Pierre Hospital to the near-by laboratory of the Brain Research Unit for a period of about three hours. In the patients of Group C, endotracheal aspiration through the tracheostomy was made whenever necessary during small interruptions of the testing runs. No sedation was used for the tests which were not unpleasant. The patients could actually be kept in a fairly relaxed state in spite of the unusual environment.

The testing room was at 24° C. The skin temperature of the limbs was maintained at or above 34° C, as measured by a thermistor plate. The stimulus was a square electrical pulse of 200 μsec duration delivered to the fingers II and III (index and middle) (silver ring electrodes) of one hand or to the median nerve at the wrist (thin steel needles inserted near the nerve). The stimulus intensity chosen between 3 and 20 mA was checked throughout each run with a Hewlett Packard current probe. The cerebral responses were recorded with thin steel needles inserted in the scalp. The active recording electrode for the hand stimulation tests was placed 7·5 cm from the mid-line over the

parietal contralateral projection, and sometimes also on the symmetrical ipsilateral region (C_3 and C_4 in the nomenclature of the Ten-Twenty EEG system). The reference electrode was usually placed on the mid-upper forehead. When stimulating the posterior tibial nerve at the ankle with similar techniques, the cerebral response was picked up on the mid-line, 2 cm behind the vertex (C_z). Whereas intervals of at least five seconds should be used between stimuli when studying the late components of the cerebral response, they can be reduced to two seconds when considering only the latency and features of the "primary" components (Desmedt, 1971; Desmedt and Noël, 1973).

The cerebral potentials were averaged with a FabriTek computer model 1062 or 1074, with analog to digital conversion of 10 bits accuracy and sweeps of 100 μsec per address when studying the early components. Such an averaging with 10 points per msec and a system bandpass extending from 1 to 3,000 Hz have been found necessary to extract without distortion the early "primary" components of the somatosensory cerebral potential (Desmedt, Brunko, Debecker and Carmeliet, 1974). In most patients 512 to 2,048 samples were averaged in order to obtain well-defined cerebral potentials on which the latencies could be accurately estimated. Several independent runs were programmed in order to resolve any remaining ambiguity due to the small size and to the abnormal configuration of some of the cerebral potentials. Fig. 1A shows the normal wave form of the cerebral potential evoked by stimulation in the upper limb. After the early surface-negative N_1 component, there is a generally surface-positive component whose second peak is usually at about 40 msecs (P_{40} in the nomenclature referring to polarity and peak latency). This is followed by a surface-negative component. The cerebral potentials evoked by stimulation in the lower limbs have a longer latency and they do not present an early surface-negative component (Desmedt, 1971;

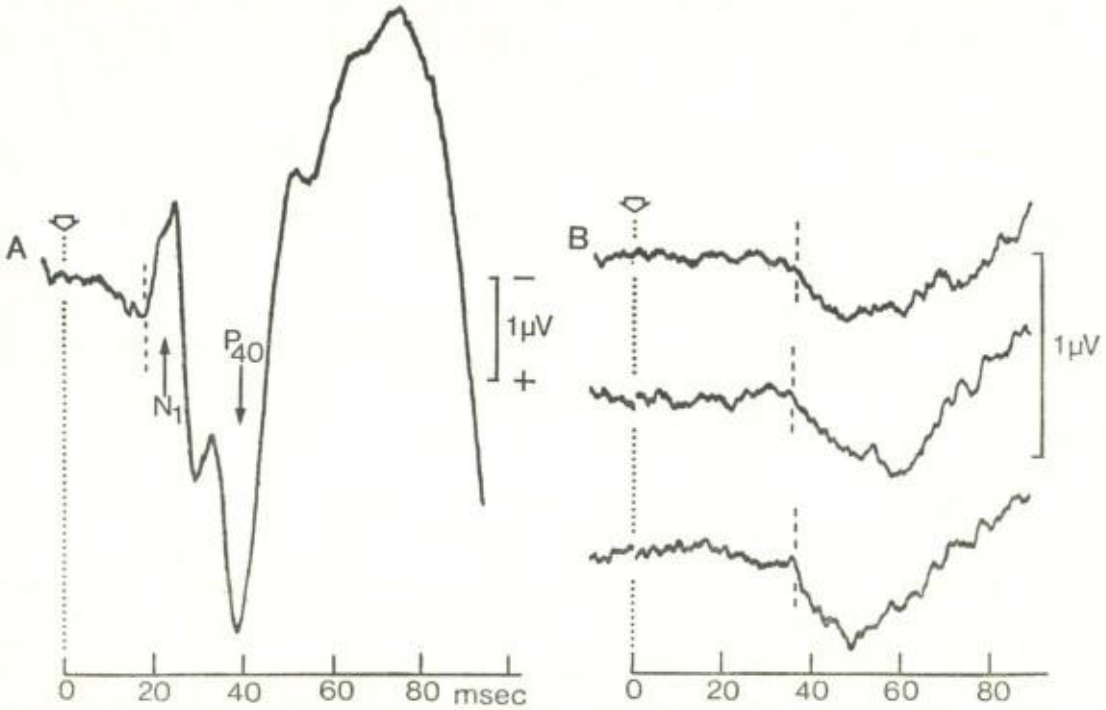

FIG. 1 (*Case I*).—Averaged cerebral potentials evoked by electrical stimulation (arrow) of the right (A) and left (B) median nerves at the wrist. Three separate trials performed on the same day are shown in B. The vertical interrupted lines indicate the onset latency of the cerebral potentials. N_1 early surface-negative component is present in A but not in B. Negativity of the active recording electrode drives the trace upwards in all the records.

Desmedt and Noël, 1973, 1975; Noël, 1974). In each session several tests were performed to compare the cerebral responses evoked from the different limbs and also to document the consistency of the pathological potentials. A second or even a third session on the following days was necessary in some of the patients.

Results

The somatosensory evoked potentials were found to present fairly normal features in the patients of group B while they exhibited marked alterations in the other patients.

Group A.—Patients with Unilateral Sensory Loss for All Modalities

Case 1.—C. D., aged 52, with a rather long-standing history of elevated blood pressure, suddenly developed a complete hemianæsthesia on the left side.

Table I

	Age (years)	*Sex*	*Clinical diagnosis*	*Time of EP test after initial accident*
Group A				
1 C. D.	52	M	Thalamic syndrome	1 week 8 months
2 R. R.	43	M	Thalamic syndrome	1 week 6 months 8 months
Group B				
3 M. L.	65	M	Wallenberg syndrome	9 days
4 W. H.	62	M	Wallenberg syndrome	3 weeks
5 F. B.	51	M	Weber syndrome	8 days
Group C				
6 P. L.	55	M	Pontine infarction	6 days
7 E. M.	25	F	Pontine infarction	14 days 22 months

On admission to hospital he was conscious, well orientated and presented no disorder of symbolic communication. The eye movements were completely paralysed in the vertical, but not in the lateral gaze (Parinaud syndrome). The pupils reacted normally to light. There was complete loss of sensation for pin-prick, temperature and tactile stimuli as well as for joint position changes and vibration on the left side of the body. No alterations of muscle strength were noticed but movements of the left limbs were grossly ataxic especially when performed with eyes closed. The tendon reflexes were normal. The plantar reflexes were flexor. The blood pressure was 180/100 mmHg. The CSF was clear and presented normal pressure and composition. The EEG revealed a fairly normal and symmetrical pattern with a 9–10 Hz background rhythm and occasional theta bursts in the temporal leads. The patient was discharged after two weeks with a diagnosis of right thalamic vascular lesion with little, if any, involvement of the cerebral cortex.

When re-examined eight months later, the patient could move his eyes in all directions but a gross vertical nystagmus was present. The patient was much disturbed by choreo-athetotic movements of large amplitude involving the left arm. The gait was made difficult but not impossible

by similar involuntary movements of the left leg. Vibration and position senses were still abolished on the left side. Tactile, pain and thermal sensations had returned to some extent but were still diminished on the left side. The patient did not report any spontaneous dysæsthesiæ.

Eight days after the acute episode the averaged cerebral potentials evoked by electrical stimulation (3 mA) of the right median nerve at the wrist (normal side) were of normal latency and configuration (Table II). No potentials could be identified on the opposite side of the scalp on stimulation of the left median nerve, even when using stimuli of large intensity (15 mA) which elicited vigorous twitching in the hand and which were reported by the patient to be faintly perceived. Eight months later small cerebral potentials could be evoked by stimulation of the left median nerve and their latency was increased to 35 msec (fig. 1B). Three separate averages showed the consistency of these responses whose early primary component was restricted to a surface-positive deflection. The potentials recorded from the opposite side on stimulation of the right median nerve (fig. 1A) presented the normal configuration (cf. Desmedt, 1971) with an initial surface-negative component (N_1) of 18 msec latency. The voltage of the early components was about ten times larger on the normal side (Table II). Late components were also delayed and reduced in

TABLE II

	Latency (msec)		*Voltage (μV)*	
	Affected side	*Intact side*	*Affected side*	*Intact side*
Case 1				
1 week	No response	18	No response	3
8 months	35	18	0·4	4·1
Case 2				
1 week	34·5 36·5	19·8	0·25	1·5
8 months	23·6	19·1	0·25	1·5
10 months	22	19·7	0·30	1·5

size when stimulating the median nerve on the affected side, both for contralateral and ipsilateral recordings. By contrast the late responses presented fairly normal features, even ipsilaterally, on stimulation of the right median nerve. These findings are in line with those described by Liberson (1966), Williamson, Goff and Allison (1970) and Tsumoto, Hirose, Nonaka and Takahashi (1973) in patients with vascular lesions at, and/or above, the thalamus.

Case 2.—R. R., aged 43, with no particular medical history, experienced sudden onset of fronto-occipital headache, more prominent on the left side. Consciousness was preserved.

Examination revealed a Parinaud syndrome with areflexic pupils and a complete loss of sensation to all modalities on the right side of the body. Muscle strength and tendon reflexes were normal on both sides. Blood pressure was 110/80 mmHg. The CSF was hæmorrhagic with 1,080 red cells per mm^3 but otherwise normal. Carotid and vertebral angiograms revealed no vascular abnormality. EEG showed normal symmetrical features. A few days later, the patient complained of continuous burning ache exacerbated by tactile and thermal stimuli on the right side of the body. Within a

couple of months the ocular signs subsided. The spontaneous pain has, however, severely disturbed the patient up to the present time (fourteen months after onset), in spite of several trials of diazepam, diphenylhydantoine and carbamazepine. The patient still presents a loss of position sense on the right side and a marked hypoæsthesia to tactile and vibratory stimuli. Pin-prick and thermal stimuli are perceived but elicit, on that side of the body, unpleasant dysæsthesiæ. The diagnosis of a left thalamic syndrome was made.

Averaged cerebral potentials evoked by stimulation of the right median nerve could be obtained in the first tests made after a week. They were, however, reduced in voltage to about one-third of those on the control side Their latency was increased to about 35·5 msec (Table II). The early surface-negative component was missing. Records from this patient have been illustrated elsewhere (Desmedt and Noël, 1975).

Group B.—Patients with Loss of Thermo-algesic Sensations

Case 3.—M. L., aged 65, with no previous neurological history, experienced a suddenly marked vertigo in the standing position and unsteadiness of gait.

Miosis of the right eye was noticed. The upper eyelid was drooping but could be voluntarily elevated (Horner syndrome). The ocular movements were full and the pupils reacted to light. The soft palate was paretic on the right side and swallowing was grossly impaired. Examination of the motor system and of the reflexes was normal. Touch, joint position and vibration were normally perceived. Thermal and pain sensation were lost over the right side of the face and the left side of the body. The EEG and CSF were normal. Spontaneous recovery set in progressively and by the third week only slight disturbance in thermal and pain sensations remained in the left leg. Normal sensation had returned in the upper limb and face. The diagnosis considered was a vascular lesion involving the right lateral medulla and the spinothalamic pathway (Wallenberg syndrome).

Nine days after onset and while the symptoms and signs were still conspicuous, cerebral potentials evoked by electrical stimulation of fingers II and III (index and

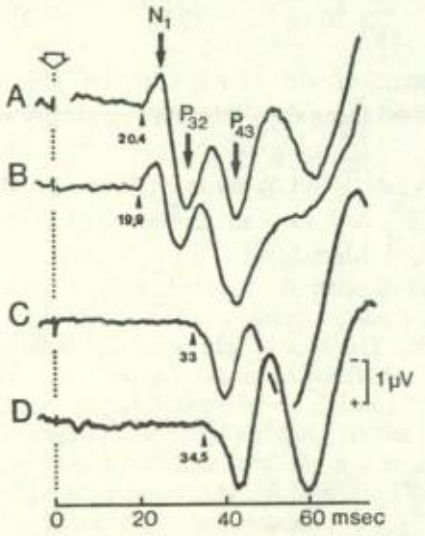

FIG. 2 (*Case 3*).—Averaged cerebral potentials evoked by electrical stimulation of fingers II and III (index and middle) of the right (A) and left (B) hands, and by stimulation of the right (C) and left (D) posterior tibial nerves at the ankle.

middle) of the right (fig. 2A) and left (B) hands presented a normal configuration. Their latencies were both within normal limits (Table III). Stimulation of the posterior tibial nerve at the ankle also evoked normal cerebral potentials whose latency was within the normal range for body size (cf. Noël, 1974; Desmedt and Noël, 1975).

Case 4.—W. H., aged 62, presented a similar syndrome of acute onset with a left Horner syndrome, left paresis of the soft palate and pharynx and a contralateral thermo-algesic sensory loss on the right side of the body. Spontaneous recovery occurred within two months. The diagnosis was lateral infarction of the left medulla (Wallenberg syndrome). Cerebral potentials evoked by finger stimulation were normal (Table III).

Case 5.—F. B., aged 51, with a rather long-standing elevation of blood pressure suddenly developed diplopia and left hemiplegia. Examination disclosed at that time a right oculomotor nerve palsy and a flaccid hemiplegia with a Babinski sign on the left side. Joint position and vibration sense were preserved while thermo-algesic sensations were diminished in the left leg. The patient was conscious and well orientated. The diagnosis was a vascular lesion of the ventrolateral right mesencephalon (Weber syndrome) with little, if any, involvement of the medial lemniscus (*see* Discussion). Cerebral potentials evoked by finger stimulation were normal in tests performed eight days after onset (Table III). Three weeks later the

TABLE III

	Latency (msec)		*Voltage (μV)*	
	Affected side	*Intact side*	*Affected side*	*Intact side*
Case 3	20·4	19·9	2·8	2
Case 4	20	20	1·5	2
Case 5	20·4	19·2	3·2	3

patient had acute aggravation with coma, complete bilateral ophthalmoplegia and bilateral Babinski. He died three days later and necropsy was opposed by the family.

Group C.—Patients with Locked-in Syndrome (Plum and Posner, 1966)

Case 6.—P. L., aged 55, with no known neurological history, developed an acute quadriplegia during sexual intercourse.

On examination he appeared conscious but could only communicate by blinking and vertical eye movements. Lateral gaze was paralysed. The pupils were equal and reacted normally to light. Corneal reflexes were absent. There was a right peripheral facial palsy. The patient understood speech but could only emit unintelligible sounds. The soft palate and pharynx were paralysed but the gag reflex was retained. There was a severe quadriparesis, more marked on the left side. Deep reflexes were brisk and symmetrical. Babinski's sign was present on both sides. Pin-prick evoked no obvious reaction. Due to the patient's condition examination of sensation could not be performed adequately. EEG disclosed a 9 Hz symmetrical background rhythm with temporal theta wave bursts. The CSF was colourless and of normal composition. The diagnosis was a vascular lesion of the medial basis pontis.

The averaged cerebral potentials evoked by rather strong stimulation (15 mA) of the median nerves at the wrist presented reduced voltage and abnormal wave forms

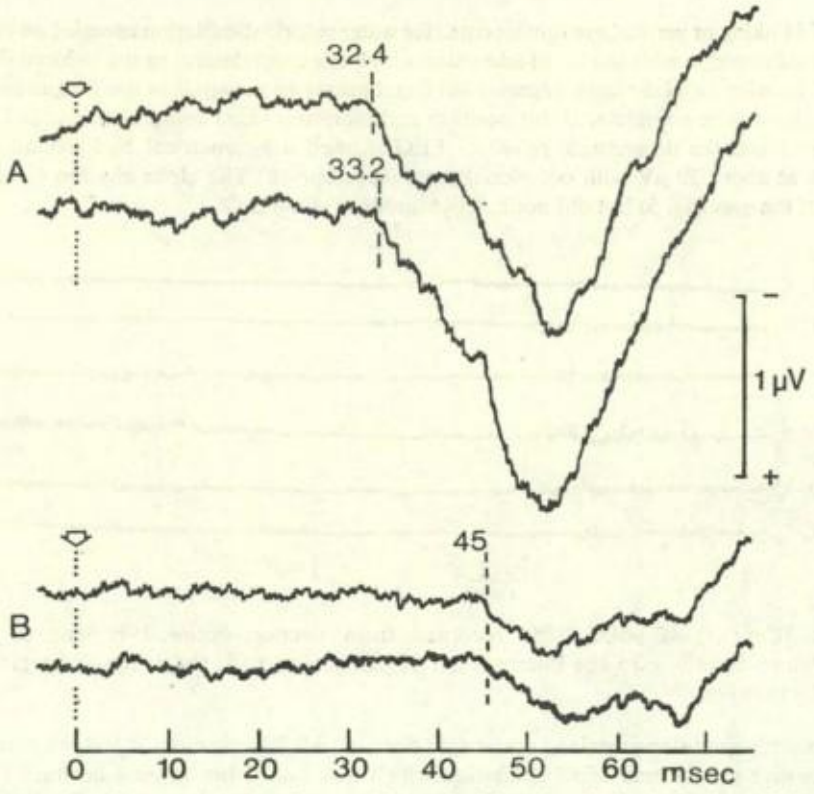

FIG. 3 (*Case 6*).—Averaged cerebral potentials evoked by electrical stimulation of the right (A) and left (B) median nerves at the wrist. Two separate trials on the same day show the consistency of the potentials.

with no clear early surface-negative component N_1 (fig. 3). The onset latency of the potential was 33 msec for stimulation of the right median nerve (A) and as much as 45 msec for stimulation of the left median nerve (B). Two independent trials on the same day showed consistency of the measurements of these rather small responses. The peak of the positive components had a latency of 52 and 60 msec respectively which also exceeded the normal range (cf. fig. 1A).

Case 7.—E. M., aged 25, presented an episode of sudden occipital headache, vomiting and blindness rapidly followed by loss of consciousness.

On examination she appeared comatose with decerebrate posturing of all four limbs after noxious stimuli. Respiration was 22 per minute. The blood-pressure was 130/80 mmHg. Eyes were deviated downward and to the right. The right eye presented occasional roving movements while the left eye remained immobile. The right pupil was midposition (diameter 4 mm) and the left pupil was pin-point. Both reacted to light. A corneal reflex was present on the right side and absent on the left. The teeth were tightly clenched. Hypertonia was present in all four limbs. The deep reflexes were hyperactive and the plantar reflexes extensor bilaterally. The CSF was bloodless and of normal composition. A vertebral angiogram revealed a complete basilar artery occlusion (fig. 4, Plate XIII). During the ensuing days, the patient progressively regained consciousness and appeared aware of her surroundings. She now followed moving objects in the vertical plane. She blinked to menace but only on the right side and she attempted to answer written, but not verbal, commands by voluntary

unilateral blinking or vertical eye movements. Ice water caloric stimulation revealed an internuclear type ophthalmoplegia with failure of adduction of the eye contralateral to the induced direction of the gaze. Inferior facial diplegia, superior left facial paralysis, paralysis of the tongue and pharynx and tetraplegia were complete. Joint position and vibration sense could not be tested. Noxious stimuli enhanced the decerebrate rigidity. EEG showed a symmetrical background rhythm at 8 to 9 Hz at about 30 μV with occasional theta wave bursts. The alpha rhythm disappeared to opening of the eyes (fig. 5) but did not react to auditory stimuli.

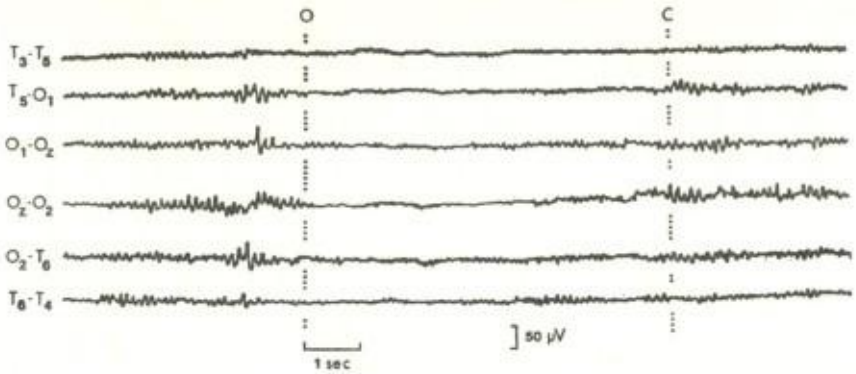

FIG. 5 (*Case 7*).—Bipolar EEG recorded from temporo-occipital regions of the scalp (nomenclature according to the international 10–20 system) with the effect of opening (O) and closing (C) the eyes.

The patient's condition remained unchanged during the following months and she was submitted to an intensive programme of rehabilitation which maintained her general health. Twenty-two months after the accident she was still paralysed and anarthric but fully alert. She had learned to communicate by using appropriate oculomotor responses. Eye movements were full. The pupils were in mid-position and reacted symmetrically to light. A central facial paresis was still present on the left side. She could open her mouth and protrude her tongue beyond the edge of the lower teeth. She was able to swallow water and puréed foods. The quadriplegia was complete with brisk symmetrical deep reflexes and bilateral Babinski sign. Painful stimulation of the face, trunk or extremities elicited some grimacing but no longer enhanced the muscular hypertonia. The position of the extremities was obviously perceived on the left side but perception appeared impaired to some extent on the right side. Examination of sensation by clinical means was difficult to achieve.

Fig. 6 illustrates averaged cerebral potentials evoked by electrical stimulation (6 mA) of the posterior tibial nerves at the level of the ankle on both sides. Two weeks after the onset of the accident, the potential evoked by stimulation of the right nerve was reduced in size and markedly delayed (latency 43 msec) considering the small body size of the patient (153 cm) (fig. 6A). The stimulation of the left nerve elicited a fairly normal potential whose onset latency of 27 msec was within normal range for body size (B). Definite improvement in the response to right nerve stimulation was recorded twenty-two months later (C), the latency having decreased to the same value as that of the opposite side (D). A small difference in the voltage of the responses was still present.

Fig. 7 displays on fast sweeps the cerebral potentials evoked by electrical stimulation (10 mA) of fingers II and III, index and middle, of either hands at nearly two years' interval. Stimulation on the right side evoked a small response with

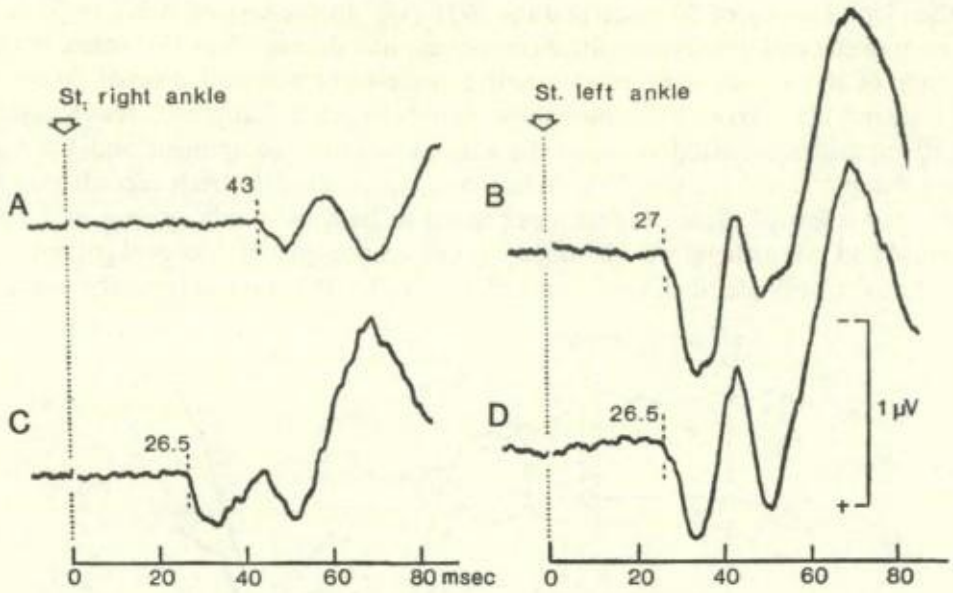

FIG. 6 (*Case 7*).—Averaged cerebral potentials evoked by electrical stimulation of the right (A, C) and left (B, D) posterior tibial nerves at the ankle, in tests performed either fourteen days (A, B) or twenty-two months (C, D) after the vascular accident.

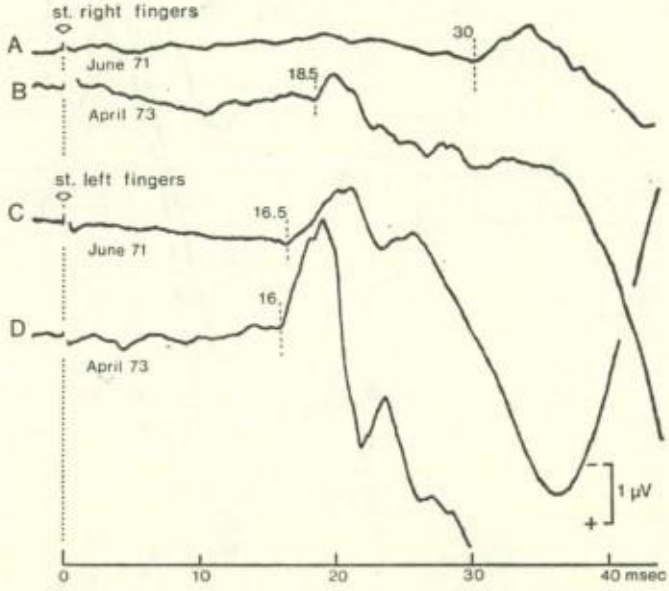

FIG. 7 (*Case 7*).—Averaged cerebral potentials evoked by electrical stimulation of fingers II and III of the right (A, B) or left (C. D) hands in tests performed either fourteen days (A, C) or twenty-two months (B, D) after the vascular accident.

rather long latency of 30 msec in June 1971 (A). In the test of April 1973, onset latency of the early surface-negative component had decreased to 18·5 msec, but the latency of the subsequent surface-positive component was still delayed compared with the normal. When examining these records in detail it appeared that a number of irregularities occurred between the earliest negative component and the large downswing of the trace (fig. 7B). When comparing separate trials recorded on the same day a few of these irregularities proved to be consistently present at a given latency and were therefore identified as subcomponents of the evoked potential (fig. 8A; for example, deflexions marked 1 and 2). This unusual pattern could be

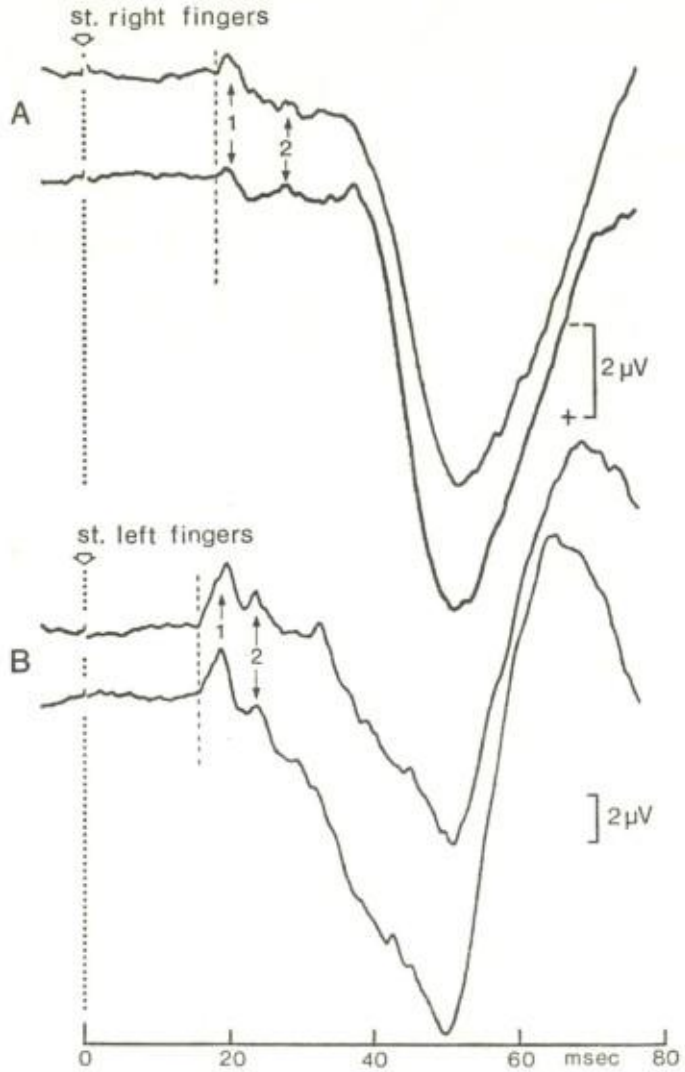

FIG. 8 (*Case 7*).—Averaged cerebral potentials evoked by finger stimulation twenty-two months after the vascular accident. Two separate trials show the consistency of several sub-components (for example arrows 1 and 2) at the beginning of the response.

interpreted as resulting from the desynchronization of the early surface-negative component (*see* Discussion). Stimulation of the left fingers evoked potentials of about 16 msec onset latency in the two tests of fig. 7C and D. The voltage of the potential was much increased in the second test in April 1973. A desynchronized pattern was also noticed in the early negative component on that side (fig. 8B).

Discussion

Appropriate clinical neurophysiological procedures can be designed to provide unique evidence about the functional capabilities of various pathways coursing along the brain-stem. For the somatosensory modality, joint position and vibration senses travel along the medial lemniscus while pain and temperature senses are mediated by the laterally situated spinothalamic pathway (Rose and Mountcastle, 1959). The pathways converge rostrally to the ventral posterior nuclei of the thalamus. These ascending tracts are known to be involved separately or simultaneously by certain types of vascular lesions which then produce various clinical syndromes of dissociated or total sensory loss. Our patients have been arranged in three groups (Table I) for the purpose of the present study.

The two patients of Group A present a unilateral sensory loss for all modalities. Spontaneous burning pain (so-called thalamic pains of Dejerine and Roussy, 1906) was a conspicuous feature of patient 2. Such a painful syndrome is generally related to a contralateral thalamic infarction but it has also been reported in lesions of the parietal cortex (Hamby, 1961; Cooper, 1965). In our 2 patients however the associated paralysis of vertical gaze and convergence (Parinaud syndrome) pointed to a deeply located lesion at the level of the pretectal area and argued for the sensory loss and spontaneous pain being related to a thalamic rather than to a cortical lesion. This interpretation was further supported by the absence of focal signs in the EEG. In patients of Group A the somatosensory evoked potentials presented a marked augmentation of the onset latency (Table II). The wave form was altered with an absence of the early surface-negative N_1 component. We cannot say whether the latter finding will eventually prove to be a constant feature. The marked changes in latency can be contrasted with the fairly normal latencies described in patients where lesions involved the cortex of the parietal lobe (Liberson, 1966; Laget, Mamo and Houdart, 1967; Williamson *et al.*, 1970; Noël and Desmedt, 1972; Tsumoto *et al.*, 1973). The voltage of the contralateral and ipsilateral responses was reduced in the thalamic lesions (Table II) whereas in the patients with cortical parietal lesions there was generally a similar general reduction except in some of the patients where a selective increase of the late negative component could be observed (Laget *et al.*, 1967; Noël and Desmedt, 1972; Tsumoto *et al.*, 1973).

Careful evaluation of the onset latency of the cerebral evoked potential in two or more trials (fig. 1B) might thus help to distinguish the cortical and the thalamic location of the lesions in patients with such clinical signs.

The patients of Groups B and C presented clinical evidence of vascular lesions in the brain-stem. At that level, the two main afferent somatosensory pathways can be

differentially affected by localized vascular lesions in view of the pattern of arterial supply by collateral branches of the vertebral and basilar arteries (Gillilan, 1964). Patients 3 and 4 were typical examples of the Wallenberg syndrome involving the lateral medulla (Wallenberg, 1895; Dejerine, 1914; Isch, 1957). This syndrome, by far the most common ischæmic injury of the brain-stem, results from an obstruction of the vertebral or posterior cerebellar artery (Fisher, Karnes and Kubik, 1961; Metzinger and Zülch, 1971). The rather stereotyped infarct of the medulla is dorsolateral to the upper border of the inferior olive and extends dorsally up to the fourth ventricle. The lesion involves the laterally situated spinothalamic tract but not the medial lemniscus which is located between the inferior olives at this level (Giok, 1956), which explains the dissociated sensory loss.

In both Wallenberg patients the somatosensory evoked potentials presented latency, configuration and amplitude within the normal range, with no significant difference between the cerebral responses recorded from the two sides (fig. 2 and Table II).

In patient 5 with a Weber syndrome the joint position sense was preserved, suggesting the integrity of the medial lemniscus which at this level of the mesencephalon lies transversely in the base of the tegmentum. The clinical evidence was compatible with an infarct restricted to the right peduncular region, interrupting the oculomotor fibres as they cross the pyramidal tract, a distribution which is not exceptional in that area. Among 12 patients with mesencephalic infarcts and obstruction of the basilar artery, Metzinger and Zülch (1971) found 9 in whom the destruction was confined to one or both peduncles and spared the tegmental and tectal segments. In our patient 5 the somatosensory evoked potentials were normal on both sides.

The findings in Group B patients are in line with previous studies on patients with dissociated sensory loss which showed that the impulses evoking the somatosensory cerebral potentials travel along the dorsal column pathway of the spinal cord, the potentials being generally unaffected in patients with loss of pin-prick and temperature sensations, but reduced in voltage and delayed in latency when there is loss of joint position and vibration senses (Halliday and Wakefield, 1963; Halliday, 1967).

Such evidence can now be used to interpret the evoked potential findings in other patients with severe brain-stem lesions in whom the clinical evaluation of somatic sensation is difficult or impossible. Patients with the locked-in syndrome (Table I, Group C) present alert wakefulness but cannot communicate except with eye movements because of their brain-stem tetraplegia (Plum and Posner, 1966). In such cases the extent of the lesion and the actual sensory loss, if any, are difficult to resolve by clinical examination only and should be assessed in the living patient with appropriate electrophysiological techniques not requiring the patient's participation. We found the cerebral somatosensory evoked potentials useful in approaching this problem (cf. Feldman, 1971; Desmedt, 1971) and other procedures such as blink reflex studies (Kimura, 1973) and investigations of the short latency brain-stem

auditory evoked potentials (Jewett and Williston, 1971; Picton, Hillyard, Krausz and Galambos, 1974) seem also to be of potential value.

In these locked-in patients the clinical findings are best accounted for by an extensive infarction of the basis pontis which interrupts the descending motor pathways but spares at least partially the tegmentum pontis (Plum and Posner, 1966). The clinical responsiveness of patients with pontine infarct appears to be roughly related to the extent of the tegmental involvement: patients with nearly total transection of basis and tegmentum pontis fail to respond in a significant way to any form of stimulus while those with unilateral and/or partial tegmental involvement retain the ability to communicate by means of eye movements (Chase, Moretti and Prensky, 1968). In our patient 6 the finding of a right peripheral facial palsy documented a lesion more extensive on the right side. In patient 7 a pin-point light-reactive pupil on the left side suggested a tegmental lesion on that side only (Plum and Posner, 1966). The EEG with a fairly normal alpha rhythm (8 to 9 Hz) which was clearly reduced by visual or somatosensory stimuli (fig. 5) is not an unusual finding in patients with extensive but incomplete lesions of the pons (Loeb, Rosadini and Poggio, 1959; Chatrian, White and Shaw, 1964; Chase *et al.*, 1968).

Since the medial lemniscus is situated in the ventral part of the pontine tegmentum, it is in a critical position in relation to the above considered pontine infarct of the locked-in patients. One could expect the medial lemniscus to be involved more or less severely depending on the dorsalward tegmental extent of the lesion on either side. This concept is supported by the detailed findings of abnormal somatosensory evoked potentials in our patients 6 and 7. The cerebral responses were reduced in voltage to a variable extent with respect to the normal range but they were not found to be abolished, provided the tests were carried out under appropriate conditions and with sufficient resolving power (cf. Desmedt, 1971; Desmedt and Noël, 1973; Desmedt, Brunko, Debecker and Carmeliet, 1974). The onset latencies were increased, in one instance by as many as 25 msec (fig. 3B), above the normal range. Furthermore the increase of the onset latency was more or less proportional to the severity of the reduction of the response voltage and of the distortion of the wave form (figs. 3, 7). The distortion consisted of reduction or disappearance of the early surface-negative component N_1 and of a somewhat increased duration of the surface-positive component (P_{40} in fig. 1A; *see* fig. 3). When comparing the cerebral potentials evoked from the upper or lower limbs and from either side of the body, one could even surmise the probable extent of encroachment of the lesion into the tegmentum. For example in patient 7, responses to right or left hand stimulation were both abnormal, but more so for right hand stimulation (fig. 7A, C) while responses to leg stimulation proved normal on the left side and reduced and delayed on the right side (fig. 6A, B). Since the projection from the lower extremity travels laterally compared to that from the upper limb in the medial lemniscus, these evoked potential data would predict a lesion involving both the left medial lemniscus and the medial part of the right one.

The mechanism responsible for the increased latency and reduced size of the averaged cerebral response in Groups A and C patients is not yet clear although it can be said to involve a localized segment of the somatosensory pathway. The recording of the afferent volley along the peripheral nerve indicated maximum conduction velocity within normal range in these patients. It would appear reasonable to suppose that the corticipetal conduction proceeds normally up to the level of the lesion in the brain-stem.

The volley must undergo some interference at the level of the anatomical lesion: for example, a number of axons may be blocked or even anatomically interrupted and could therefore not contribute to the eliciting of the cerebral response. The question whether the other, still conducting, lemniscal axons present a segment of slowed conduction cannot be answered at present although this would seem likely to be the case in a number of them. In this respect it is important to quote the marked slowing of corticipetal conduction which has been recorded in patients with multiple sclerosis (Halliday and Wakefield, 1963; Namerow, 1968; Desmedt and Noël, 1973; Halliday, McDonald and Mushin, 1972, 1973). In the latter case, localized segmental demyelination of central axons is a prominent feature and the mechanisms for slowing have been analysed in cat preparations with experimental demyelination lesions (McDonald and Sears, 1970*a*, 1970*b*; Mayer, 1971; McDonald, 1973). If localized slowing of conduction had indeed a significant incidence among axons adjacent to, or somehow involved by, a localized vascular lesion, it would be important to investigate the responsible pathophysiological mechanisms which are at present unknown (Segmental demyelination? Reduction in fibre diameter through local compression? Localized ischæmia? etc.). Another question is whether the still functional axonal segments beyond the localized vascular lesions would present impaired conduction and/or reduced synaptic efficiency at the next (thalamic or cortical) relays.

All the processes just considered could contribute to the recorded increase of the onset latency of the cerebral response. It should also be emphasized that the alterations of the evoked potentials in Groups A and C patients were found to be at least partially reversible (Table II; figs. 6 and 7). This suggests that the lesion cannot be viewed as a mere anatomical interruption of the central axons in which case no regeneration could occur. Furthermore the data about functional recovery after the accident suggest that the evoked potentials afford a valuable method to document not only the severity of the sensory defects due to the vascular lesion, but also their potential improvement thereafter.

An interesting new feature was observed in the cerebral potentials evoked by finger stimulation in Case 7, namely, a seemingly desynchronized pattern of the early surface-negative N_1 component in tests performed twenty-two months after the accident (fig. 8). This might be the result of a temporal dispersion of the corticipetal volley, possibly related to marked variation in the stage of recovery of the axons at the level of the pontine lesion.

Summary

Clinical and electrophysiological observations are described in 7 patients with clinically well-identified vascular lesions of the brain-stem or diencephalon. In the patients of Group A with a thalamic syndrome, the somatosensory cerebral evoked potentials had a reduced voltage and increased latency on the affected side. No significant anomalies were recorded in the patients of Group B with a Wallenberg or Weber syndrome. In patients of Group C with a locked-in syndrome, the cerebral evoked potentials presented marked bilateral anomalies which provided interesting data about the extension of the pontine vascular lesions into the tegmentum. The pathophysiological mechanisms involved in the changes of average cerebral evoked potentials and in the slowing of corticipetal conduction are discussed.

Acknowledgments

This research was supported by grants from the Fonds de la Recherche Scientifique Médicale and from the Fonds National de la Recherche Scientifique. We wish to thank Dr. A. Cornil (Intensive Care Unit of the Department of Medicine, St Pierre University Hospital) for his co-operation.

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(Received September 26, 1974)

LEGEND FOR PLATE

PLATE XIII

FIG. 4 (*Case 7*).—Vertebral angiogram showing the obstruction of the basilar artery (arrow).

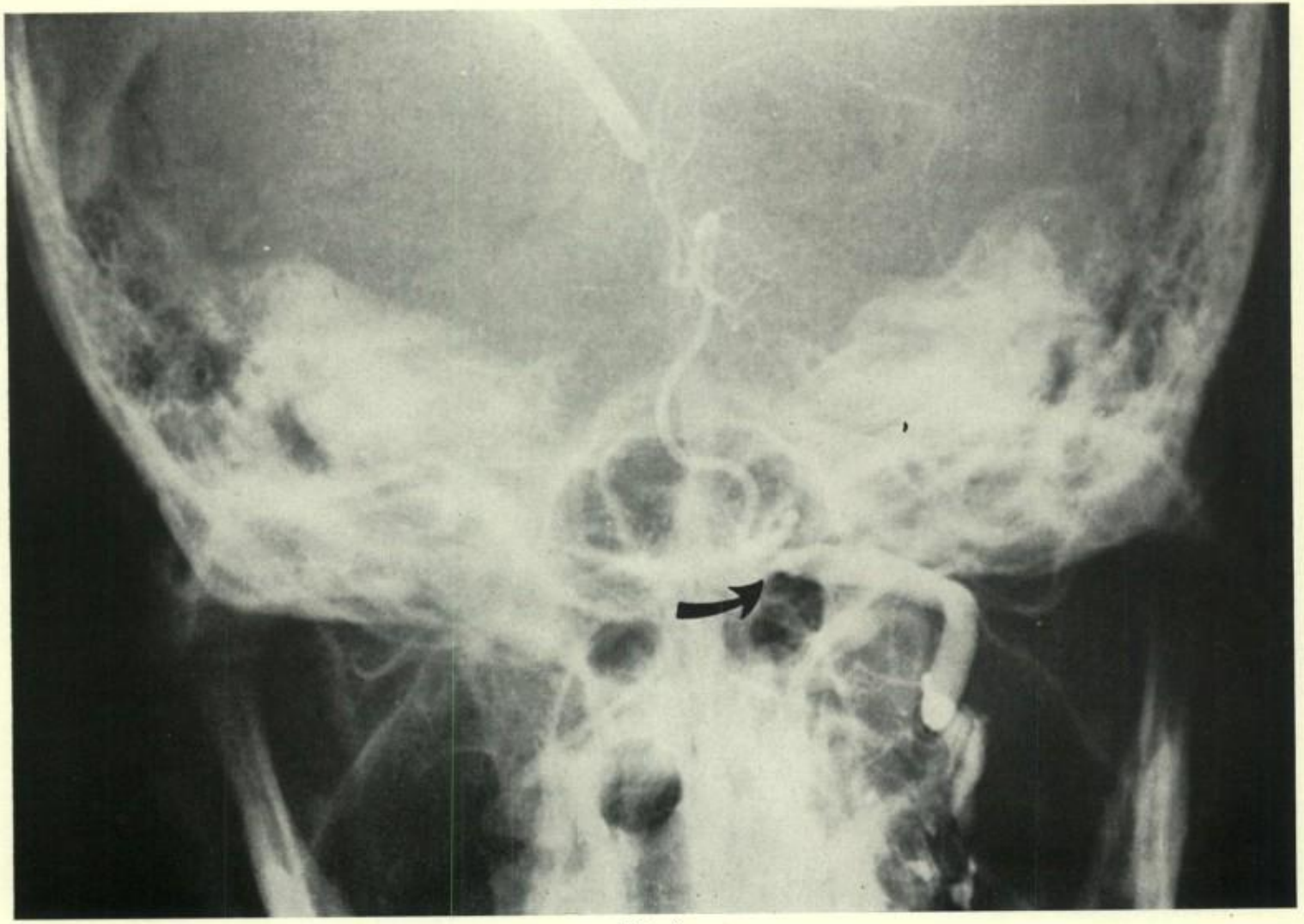

FIG. 4.

To illustrate article by P. Noel and John E. Desmedt.