

Scalp, earlobe and nasopharyngeal recordings of the median nerve somatosensory evoked P14 potential in coma and brain death

Detailed latency and amplitude analysis in 181 patients

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Summary

Median nerve somatosensory evoked potentials (SEPs) were recorded in a total of 181 patients in coma and brain death. Special attention was paid to the derivation of P14 (the positive potential occurring ~14 ms after median nerve stimulation) with different electrode montages, using midfrontal scalp (Fz), linked earlobe (A_{1/2}), median nasopharyngeal (Pgz) and non-cephalic reference (NC) electrodes. The P14 amplitude (and, to a lesser extent, latency) were invariably lower in brain death than in coma. The potential was preserved in coma in all patients, but lost in brain death in 9.8% in Fz–NC and Pgz–NC recordings, in 23.2% in Fz–A_{1/2}, and in 100% in Fz–Pgz. Thus, Fz–Pgz

was the derivation yielding the most reliable results with respect to the distinction between coma and brain death and is therefore recommended as a confirmatory test, when other diseases interrupting the lemniscal pathway (isolated brainstem death, high cervical transverse cord lesion and focal bilateral lemniscal lesion) are excluded. Theoretical considerations lead to the hypothesis of different (rostral and caudal) segments of the P14 generator dipole being recorded by the different electrode montages. It is assumed that Fz–Pgz picks up the most rostral part of P14 (rP14) that is invariably lost in brain death and preserved in coma.

Keywords: evoked potentials; somatosensory; P14 far-field potential; nasopharyngeal electrodes; brain death; coma

Abbreviations: A_{1/2} = linked earlobes (electrode location); AEP = auditory evoked potential; Fz = midfrontal scalp (electrode location); NC = non-cephalic reference (electrode location); Pgz = median nasopharynx (electrode location); P14 = the positive potential occurring ~14 ms after median nerve stimulation; rP14 = the most rostral part of P14; SEP = somatosensory evoked potential

Introduction

In brain death, functions of the brain are lost, whereas functions of the spinal cord or peripheral nerves are typically preserved for some time. Brain death is primarily diagnosed by clinical evaluation (coma, brainstem areflexia, apnoea), but electrophysiological recordings like EEG, SEPs or auditory evoked potentials (AEPs) may play a role as additional tests confirming the clinical diagnosis.

The derivation of the median nerve somatosensory evoked P14 potential is of special importance in this context, as it is generated around the level of the foramen magnum, i.e. near the zone of transition between CNS structures whose

function is lost in brain death (brainstem) and those whose function may still be preserved (spinal cord). The typical derivation of P14 uses a scalp electrode (e.g. Fz) and a non-cephalic reference (NC; e.g. shoulder contralateral to stimulation) (Cracco and Cracco, 1976). This yields a series of so-called far-field potentials among which P14 is the last. The use of a non-cephalic reference, however, results in a relatively high inter-electrode distance, so that the injection of internal or external artefacts (ECG and other electrical fields) is rather high. Therefore, an earlobe instead of a non-cephalic reference is often used, especially in electrically

active environments like intensive care units. The SEP recordings from scalp to earlobe yield a P14 potential without a preceding P9 or P11 wave (these are cancelled, as they are identically recorded in scalp-NC and earlobe-NC derivations). Conventionally, the scalp-earlobe derivation is used as an alternative to scalp-NC recordings without considering any possible P14 latency or amplitude differences between the different electrode montages.

In a previous study in 50 patients (Wagner, 1991) we used a nasopharyngeal reference (Pgz) in SEP recordings in deeply comatose and brain dead patients and found different P14 amplitudes or latencies in Fz-NC, Fz-A_{1/2} and Fz-Pgz derivations. The most important differences were found in brain death, where there was a lack or an inversion of P14 in Fz-Pgz, whereas this potential was mostly preserved (with a low amplitude) in Fz-A_{1/2} and Fz-NC records. We also observed a decrease of P14-P9 interpeak interval with the progression from coma to brain death.

In this study, P14 amplitudes and latencies were systematically analysed with different electrode montages using scalp, earlobe, nasopharyngeal and non-cephalic leads in 181 comatose or brain dead patients. The practical value for brain death diagnosis as well as the theoretical significance for the understanding of P14 generation are discussed.

Patients and methods

Somatosensory evoked potential recordings

The median nerve was stimulated at the wrist with a stimulus strength sufficient to produce a clear thumb switch. Rectangular pulses of 200 µs duration were delivered at a rate of 5.1–5.4 per second.

For scalp and earlobe recordings, needle electrodes were used. The nasopharyngeal lead consisted of a slightly curved silver wire, isolated except for the ball shaped tip (5 or 2 mm in diameter). It was inserted through the inferior nasal meatus and moved forward until the tip reached the posterior nasopharyngeal wall (Wagner, 1988). Electrode resistance was <10 kΩ. The following electrode montages were used (the components derived are in parentheses): Fz-NC (P9, P11, P14); Pgz-NC (P9, P11, P14); Fz-Pgz (P14); Fz-A_{1/2} (P14). In addition, recordings from C₃'-Fz, C₄'-Fz (cortical N20) and Cv7-Jug (spinal cord N13) were done. Electrode locations are labelled as follows: Pgz = median nasopharynx, according to the 10–20 system, where Pg₁ and Pg₂ designate nasopharyngeal electrode locations on the left and right side, respectively (Jasper, 1958), A_{1/2} = linked earlobes, Cv7 = spinous process of the VIIth cervical vertebra, Jug = area above the jugular fossa, NC = non-cephalic reference (shoulder contralateral to stimulation), C₃', C₄', Fz = electrode locations at the scalp according to the international 10–20 system. Fz-Pgz recordings were done in all patients, Fz-A_{1/2} in 34 comatose and 75 brain dead patients, the other derivations in nearly all cases.

The filter bandpass was from 10 Hz to 1500 Hz (or

3000 Hz). Analysis time was 50 ms and 1000 (in a few cases, 2000) samples were averaged twice. Only the components clearly reproducible in both averages were evaluated.

The following P14 parameters were measured for analysis: peak latency, onset latency (in Fz-Pgz and Fz-A_{1/2}), interpeak interval P14-P9 (referred to P9 in Fz-NC), amplitude base-to-peak (in Fz-Pgz and Fz-A_{1/2}), amplitude ratio P14/P9 (in Fz-NC and Pgz-NC) (*see* García Larrea and Mauguière, 1988). When P14 was lacking, the amplitude (or amplitude ratio) was taken as 0.0 µV for calculation of mean and standard deviation, but these records were not considered for latency measurements. For statistical analysis, the Mann-Whitney rank sum test (Wilcoxon test) was used.

If there were multiple SEP recordings in coma or brain death in one patient, the records nearest to the moment of brain death were taken for analysis.

Patients

A total of 216 SEP in 181 patients were analysed. Of these, 108 SEP recordings were done after brain death and 108 in coma, and 35 patients were studied both before and after established brain death. Transcranial Doppler and EEG studies were additionally performed in some of the cases. We did CT scans in all cases to establish the neurological/neurosurgical diagnosis. All brain dead patients had primarily cerebral causes of brain death; most of the comatose patients had clinical or radiological evidence of brainstem involvement. Details are given in Table 1. Infratentorial lesions in nine brain dead patients included spontaneous posterior fossa bleeding (cerebellum, pons), tumour (acoustic neuroma, metastasis), arteriovenous malformation of the brainstem and traumatic brain injury. Brain death was diagnosed according to German guidelines (Stellungnahme des Wissenschaftlichen Beirates der Bundesärztekammer, 1991). According to these guidelines, the diagnosis of a severe, irreparable brain lesion is a basic prerequisite; reversible conditions (drugs, hypothermia, shock, endocrine disturbances, etc.) have to be excluded. Brain stem reflexes (pupillary, corneal, oculocephalic, cough and pharyngeal as well as reaction to painful stimuli in the trigeminal territory) must be absent. Apnoea has to be tested during hypercapnia (pCO₂ > 60 mmHg) with normoxaemia. These symptoms have to be repeatedly documented during an appropriate observation period (at least 12 h) to confirm brain death. Instead of the observation period, laboratory tests (EEG, transcranial Doppler, AEP) may be used. In primarily infratentorial lesions an EEG is obligatory.

An important, but difficult question was the identification of the 'moment of brain death'. In cases of acute brainstem herniation with sudden changes in blood pressure and heart rate paralleling the abrupt development of brainstem areflexia, the progression from coma to brain death could be assessed rather accurately. However, in cases of a slow increase of intracranial pressure with a stepwise disappearance

Table 1 Patient data

	Coma	Brain death
Number of patients/records	108 (65 male, 43 female)	108 (60 male, 48 female)
Age (years)	12–92 (mean, 49.2)	15–83 (mean, 46.8)
Time delay between SEP and brain death (h)	1–456 (mean, 71.3)	1–72 (mean, 16.8)
Diagnoses (number of patients)		
Traumatic brain injury	42	41
Tumour	12	12
Subarachnoid haemorrhage	25	28
Arteriovenous malformation	1	1
Intracerebral haematoma	16	20
Intraventricular bleeding	1	–
Cerebral hypoxia	4	–
Cerebral ischaemia/infarction	6	5
Hydrocephalus	–	1
Encephalitis	1	–
Type of brain injury		
Primary	104	108
Secondary	4	–
Location of brain injury		
Supratentorial	87	96
Infratentorial	21	12

As both groups overlap by 35 patients (tested in coma and in brain death), the figures represent the number of patients recorded in each situation.

Table 2 P14 latencies and amplitudes (mean \pm SD) in different derivations in coma and brain death

	P14–P9: interpeak interval		P14 amplitude or P14/P9 ratio	
	Coma	Brain death	Coma	Brain death
Fz–NC	4.49 (\pm 0.76) ms	3.83 (\pm 0.66) ms	1.68 (\pm 1.12)	0.62 (\pm 0.54)
Pgz–NC	4.22 (\pm 0.73) ms	3.85 (\pm 0.69) ms	0.90 (\pm 0.68)	0.70 (\pm 0.61)
Fz–A _{1/2}	4.74 (\pm 0.77) ms	3.62 (\pm 0.74) ms	0.73 (\pm 0.30) μ V	0.09 (\pm 0.09) μ V
Fz–Pgz	4.86 (\pm 0.83) ms	3.99 (\pm 1.11) ms	0.65 (\pm 0.32) μ V	–0.08 (\pm 0.10) μ V

P14–P9 interpeak interval (ms) is referred to P9 in Fz–NC. For Fz–A_{1/2} and Fz–Pgz the amplitude (μ V) is measured base-to-peak; for Fz–NC and Pgz–NC the amplitude ratio P14/P9 is given.

of clinical signs of brainstem function (among which cessation of spontaneous respiration is generally the last), the exact ‘moment of brain death’ could not be identified and had to be estimated on the basis of the clinical data. The interval between brain death and SEP recording was \sim 1 h in one case, \sim 2 h in five cases, \sim 3 h in six cases, \sim 4 h in three cases, \sim 5 h in six cases, \sim 6 h in 10 cases and $>$ 6 h in the other brain dead patients. In the comatose patients, SEPs were recorded \sim 1 h before brain death in one case, \sim 3 h in one case, \sim 6 h in three cases and $>$ 6 h in the remaining comatose patients.

Results

Means and standard deviations of P14 latencies (interpeak intervals) and amplitudes (amplitude ratios) are summarized in Table 2.

Recordings from Fz–NC

The P14 wave was found in Fz–NC in all comatose patients; it was bilobed (P13/P14) in a few of them (in these cases, the larger peak was analysed). In brain death, P14 was lacking in 16.9% of the records (in 9.8% of the patients it was lacking on both sides) (Fig. 1). In some brain death derivations, it was difficult to differentiate between P11 and P14 far-field potentials, as P14 was broken by several notches.

The mean P14 latency in coma was 14.46 ms, and in brain death 13.94 ms. The P14–P9 interpeak intervals were 4.49 ms in coma and 3.83 ms in brain death; this difference was statistically significant ($P < 0.001$). The P14 amplitudes (or P14/P9 ratios), showed a marked decrease during the progression from coma to brain death. The mean P14/P9 amplitude ratio was 1.68 in coma and 0.62 in brain death ($P < 0.001$). The P14/P9 ratio was typically $>$ 1 in coma and $<$ 1 in brain death; there were, however, also patients

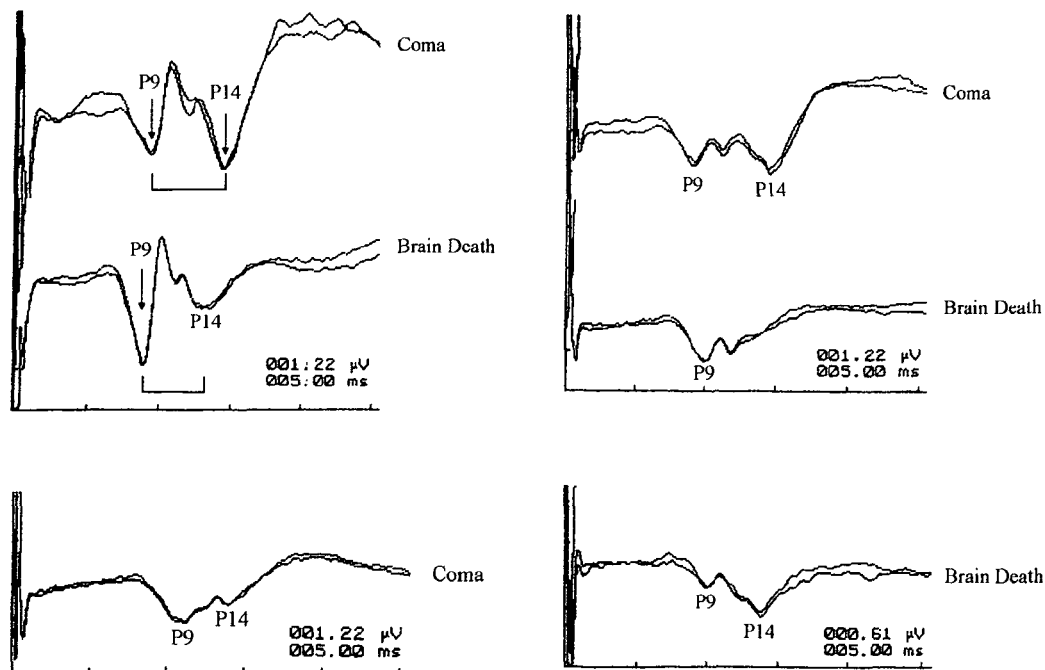


Fig. 1 Examples of P14 derivations from Fz–NC in coma and brain death. *Upper left*: decrease of P14–P9 interpeak interval and of P14/P9 amplitude ratio after progression from coma to brain death (37 years, female, brain injury, 72 h before and 3 h after brain death). *Upper right*: loss of P14 in brain death (28 years, male, brain injury, 48 h before and 24 h after brain death). *Lower left*: low P14 amplitude in coma (68 years, female, brain injury). *Lower right*: high P14 amplitude in brain death (16 years, male, brain injury, 38 h after brain death). In this and subsequent figures, positivity at the ‘active’ electrode is downward.

with low amplitude P14 in coma or high amplitude P14 in brain death (Fig. 1, lower traces). The parameter distributions in both patient groups (*see* Fig. 4) showed considerable overlap (latencies more than amplitudes), making the distinction between coma and brain death in a single patient impossible on the basis of latency or amplitude measurements in Fz–NC alone.

Recordings from Fz–A_{1/2}

When using an earlobe reference in scalp recordings, a P14 potential is normally recorded without preceding P9 and P11 components. This pattern was found in all comatose patients. In brain death, P14 in Fz–A_{1/2} was lacking in 33.9% of the records (in 23.2% of the patients it was lacking on both sides) (Fig. 2).

The mean P14 latency in Fz–A_{1/2} was slightly longer in coma (14.61 ms) than in brain death (13.59 ms). Interpeak intervals (referred to P9 in Fz–NC) were 4.74 ms and 3.62 ms in coma and brain death, respectively ($P < 0.001$). The latency distributions overlapped considerably (*see* Fig. 4). Mean onset latency was 10.96 ms in coma and 9.75 ms in brain death. There was a mean P14 amplitude (base-to-peak) of 0.73 μ V in coma and 0.09 μ V in brain death (the mean in the records with preserved P14 in Fz–A_{1/2} was 0.14 μ V; $P < 0.001$). As an ‘untypical’ finding, we also saw low P14 amplitudes in coma and high amplitudes in brain

death (Fig. 2, lower traces); there was less overlap in amplitude distributions than in latency distributions (*see* Fig. 4).

Recordings from Fz–Pg_z

In coma, records from Fz–Pg_z were very similar to those from Fz–A_{1/2}. The P14 component was found bilaterally in all comatose patients (except for one patient with a high cervical tetraplegia and cerebral hypoxia after odontoid fracture); in one patient with a bulbopontine haemorrhage and ischaemia after subarachnoid bleeding, the potential was nearly lost on one side, i.e. it was of very low amplitude (0.05 μ V). In brain death P14 was invariably lacking in all Fz–Pg_z records (Fig. 3). Instead of that, in 54.8% of the records (58.3% of the patients on one or both sides), we found an inversion of this component, i.e. a scalp-negative potential (‘inverse P14’). In some brain death records, a shallow triphasic wave with a negative-positive-negative pattern was seen, where the positive part only slightly passed beyond the baseline, but this wave form was clearly different and easily distinguishable from the monophasic P14 in coma. Both inverse and triphasic P14 potentials disappeared when serial recordings were possible (Fig. 3, lower traces); they were never seen in Fz–A_{1/2} records. The pattern of a preserved P14 in coma and a loss of P14 in brain death in Fz–Pg_z records was independent of the P14/P9 ratio in Fz–NC

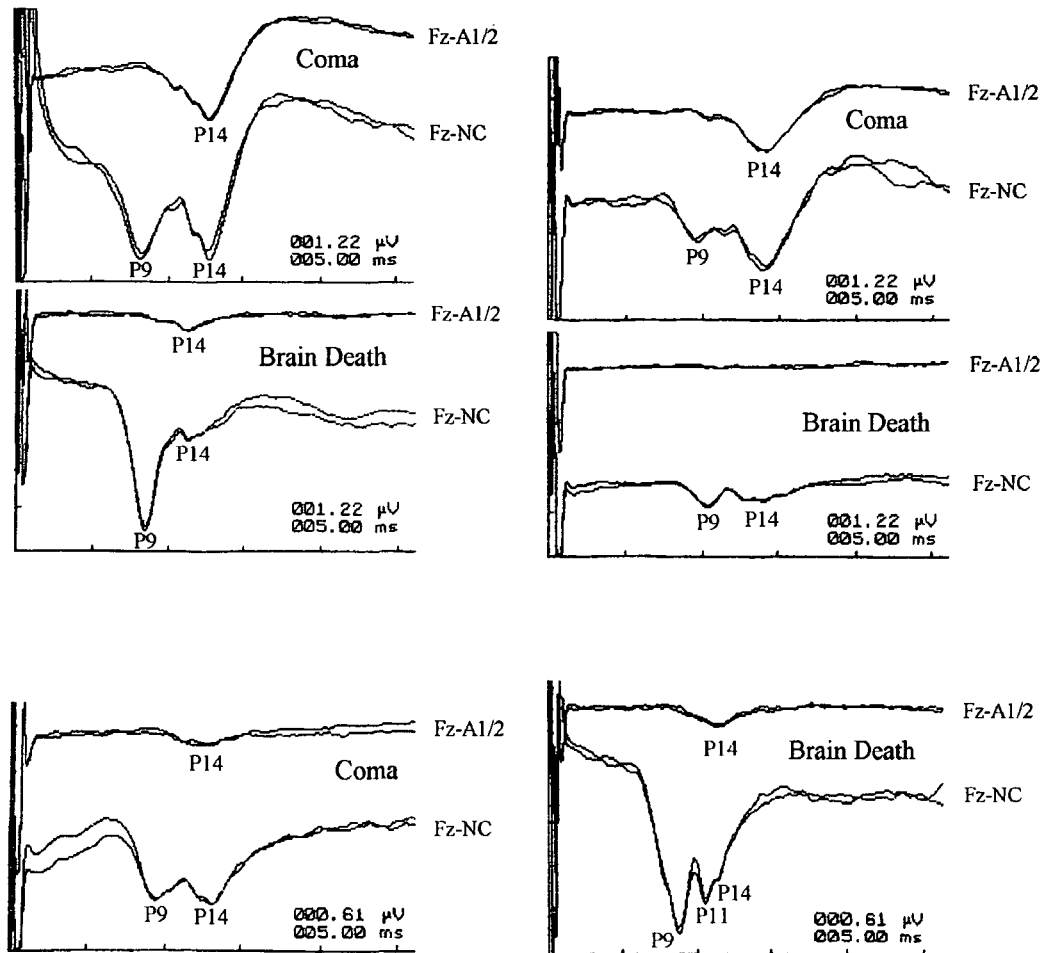


Fig. 2 P14 derivations from Fz-A_{1/2} and Fz-NC in coma and brain death. *Upper left*: decrease of P14 latency and amplitude after progression from coma to brain death (24 years, female, subarachnoid haemorrhage, 24 h before and 72 h after brain death). *Upper right*: loss of P14 in brain death (62 years, female, middle cerebral artery ischaemia, 6 h before and 65 h after brain death). *Lower left*: low P14 amplitude in coma (30 years, female, brain hypoxia). *Lower right*: relatively high P14 amplitude in brain death (39 years, female, intracerebral haematoma, 11 h after brain death).

or the preservation or loss of P14 in Fz-A_{1/2} (Fig. 5, upper traces).

The mean P14 latency in Fz-Pgz was 14.81 ms in coma and the mean latency of the 'inverse P14' in brain death was 14.14 ms. Interpeak intervals were 4.86 ms and 3.99 ms in coma and death, respectively ($P < 0.001$). There was a considerable overlap between the latency distributions (Fig. 4). The mean onset latency in coma was 11.99 ms, and in brain death 10.37 ms. The mean P14 amplitude in coma was 0.65 μV and in brain death (the overall mean inverse P14) 0.08 μV (but the mean of only those records with inverse P14 in Fz-Pgz was 0.14 μV ; $P < 0.001$). As there was no preserved P14 in brain death and no lacking P14 in coma, the P14 amplitude in Fz-Pgz was the only parameter showing no overlap in the distributions of group data.

Recordings from Pgz-NC

The non-cephalic referenced records from Pgz showed identical P9 and P11 components compared with Fz-NC, i.e.

Fz-Pgz did not show P9 or P11 (see Fig. 3). However, the P14 amplitude (or P14/P9 ratio) in coma was always lower in Pgz-NC than in Fz-NC; in brain death, they were nearly identical (or P14/P9 was slightly higher in Pgz-NC than in Fz-NC) (Fig. 5, lower traces). The P14 component in Pgz-NC was preserved exactly in those brain death records with a preserved P14 in Fz-NC.

The P14 latency was slightly higher in coma than in brain death in the Pgz-NC derivation too. The mean values were 14.19 ms and 13.89 ms, respectively (interpeak intervals: 4.22 ms and 3.85 ms; $P < 0.001$). The P14/P9 ratio was slightly, but significantly, lower in brain death than in coma (0.90 versus 0.70; $P < 0.001$). Parameter distributions are shown in Fig. 4.

Comparison of the various P14 derivations

Typical examples of different P14 derivations are shown in Fig. 5 and the overall parameter distributions are given

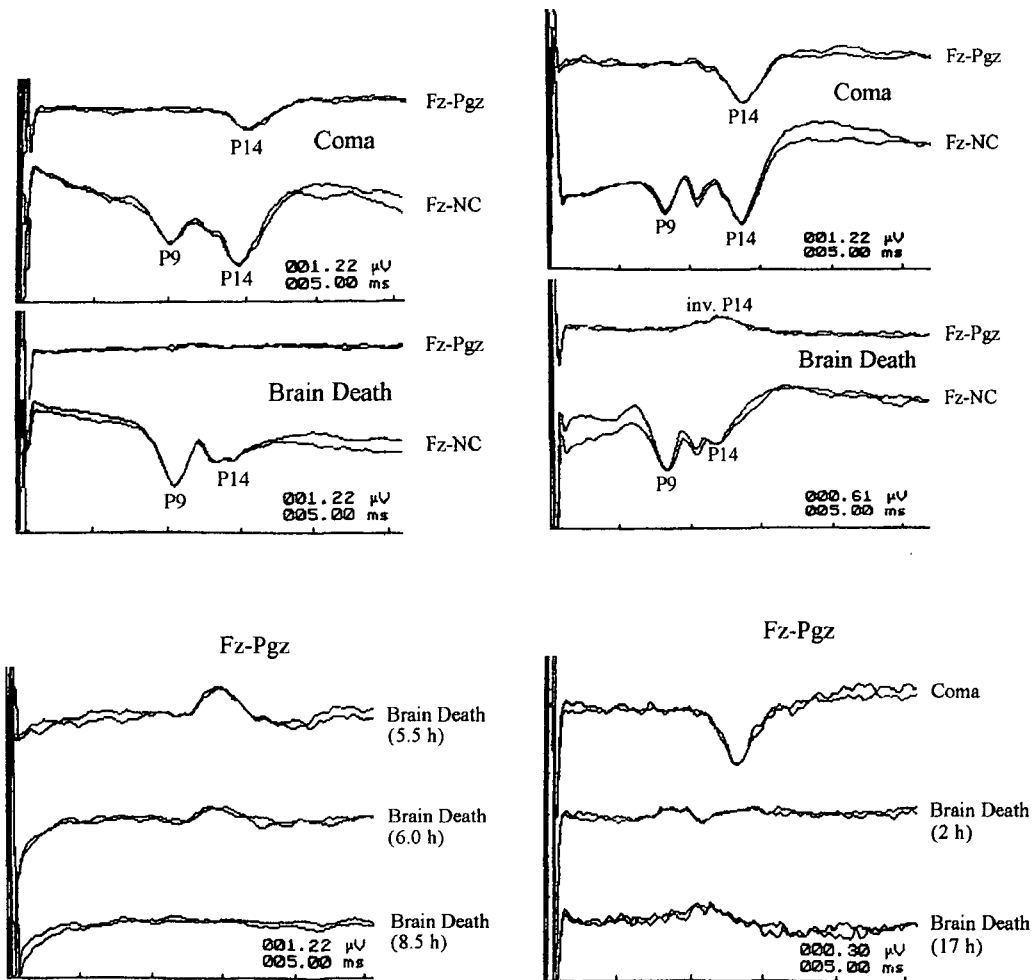


Fig. 3 P14 derivations from Fz-Pgz and Fz-NC in coma and brain death. *Upper left*: loss of P14 with the progression from coma to brain death (48 years, male, subarachnoid haemorrhage, 60 h before and 12 h after brain death; same patient as in Figure 5, lower traces). *Upper right*: loss of P14 and appearance of a scalp-negative 'inverse P14' potential in brain death (22 years, male, brain injury, 12 h before and 12 h after brain death). The inverse P14 potential is shown with a fourfold higher display gain. *Lower left*: disappearance of an inverse P14 potential 8.5 h after brain death (69 years, female, subarachnoid haemorrhage). *Lower right*: transition from a normal P14 in coma to a triphasic P14 2 h after brain death and to an inverse P14 15 h later (46 years, female, intracerebral haematoma).

in Fig. 6. The P14–P9 interpeak interval in coma was shortest in Pgz–NC (4.22 ms) and longest in Fz–Pgz (4.86 ms); the latency in Fz–NC lay in-between (4.49 ms). These differences were statistically significant (Fz–Pgz/Pgz–NC: $P < 0.001$; Fz–Pgz/Fz–NC: $P < 0.001$; Fz–NC/Pgz–NC: $P < 0.001$). Earlobe derivations (Fz–A_{1/2}) yielded a latency slightly faster (4.74 ms) than in Fz–Pgz (4.86 ms) ($P = 0.049$). In brain death, P14–P9 interpeak intervals were nearly identical in all four derivations (i.e., there were no statistically significant differences; $P > 0.35$).

The P14/P9 ratio in coma was invariably higher in Fz–NC than in Pgz–NC (mean 1.68 versus 0.90, respectively; $P < 0.001$), resulting in a scalp-positive P14 potential in Fz–Pgz. In brain death, amplitude ratios in both derivations were either identical or slightly higher in Pgz–NC (mean 0.62 in Fz–NC and 0.70 in Pgz–NC, difference not significant).

This led occasionally to an inverse P14 potential in Fz–Pgz. The P14 amplitude in coma was higher in Fz–A_{1/2} than in Fz–Pgz (mean 0.73 μ V versus 0.65 μ V, $P = 0.033$); in brain death, there was a mean amplitude of 0.09 μ V in Fz–A_{1/2} and of –0.08 μ V (inverse P14) in Fz–Pgz ($P < 0.001$) without overlap between the parameter distributions.

Derivations of other potentials (N13, N20)

Spinal N13 (negative wave at 13 ms in Cv7–Jug, see Fig. 7 for electrode positions) was found in all records in coma and in nearly all records (93.3%) in brain death. In two patients with bilateral loss of N13, post-mortem pathoanatomical studies revealed pathologies extending down to the vertebral canal; tumour infiltration was found in one case and massive bleeding from a basilar artery aneurysm in the other case.

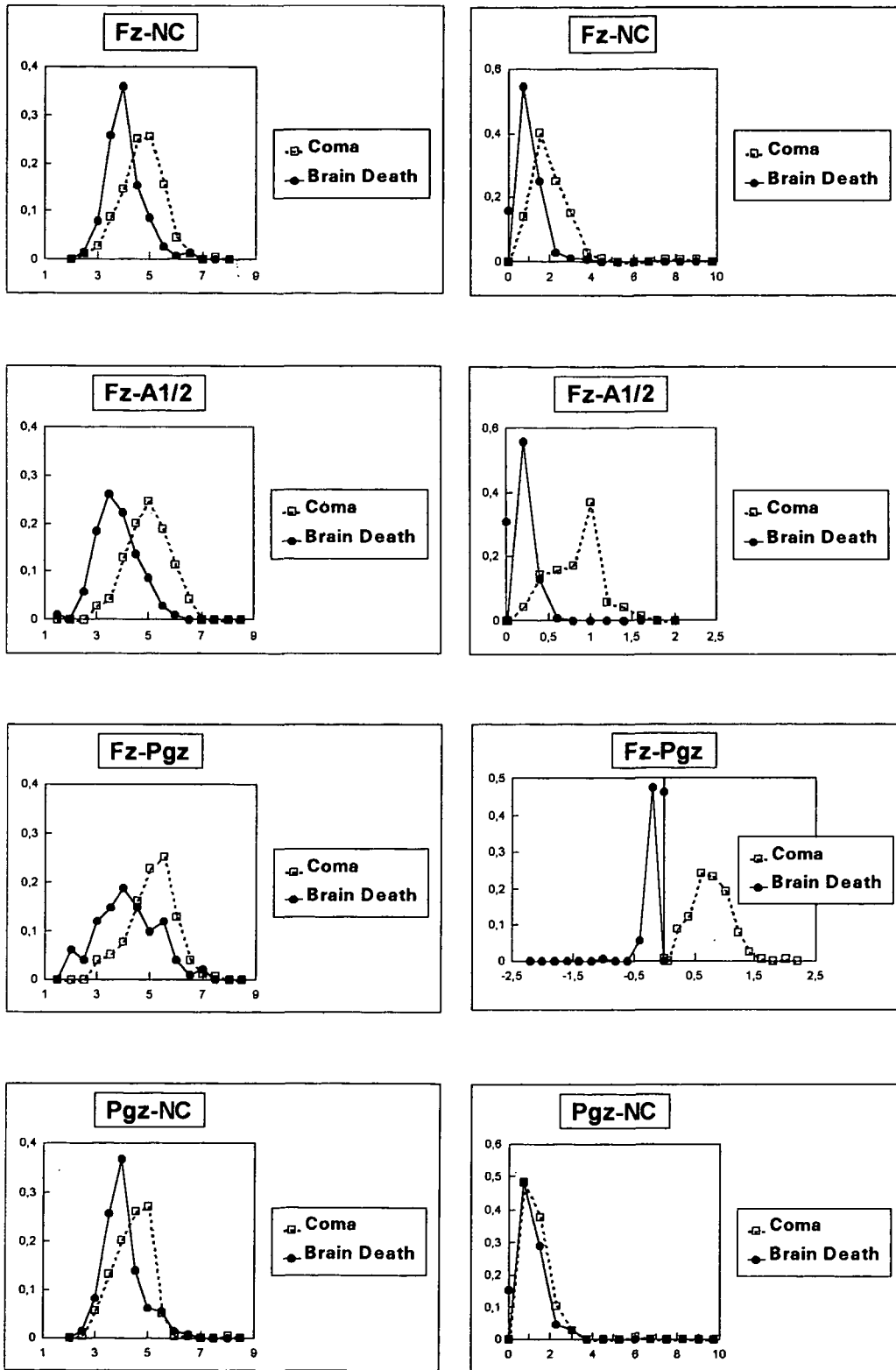


Fig. 4 Distribution of P14 latencies and amplitudes from different derivations in coma versus brain death. *Left column:* P14/P9 interpeak intervals (ms) (referred to P9 in Fz-NC). *Right column:* amplitudes (μV) (Fz-A_{1/2}, Fz-Pgz) and P14/P9 amplitude ratios (Fz-NC, Pgz-NC). For recordings from Fz-Pgz in brain death, values of the inverse P14 potential (negative amplitude) are given. Vertical axes indicate relative frequencies.

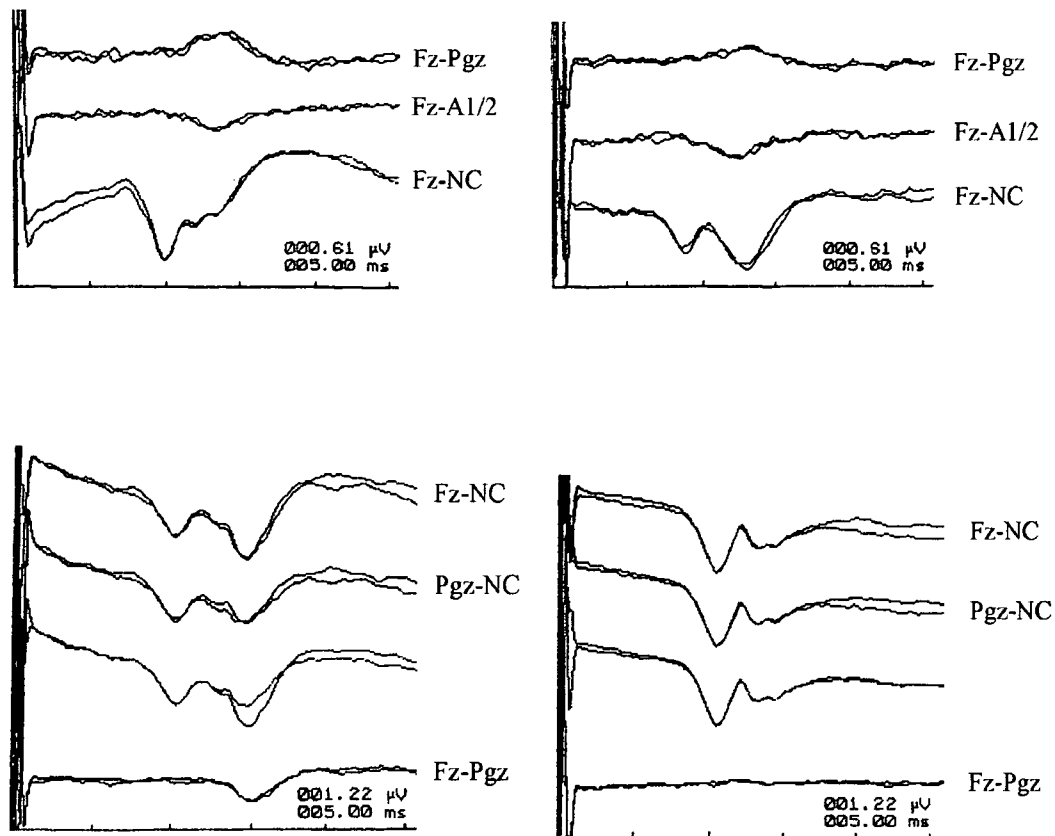


Fig. 5 Comparison of different P14 derivations in coma and brain death. Note the phase reversal between Fz-A_{1/2} and Fz-Pgz in brain death that is independent of the P14/P9 ratio (*top*), and the P14 amplitude difference between Fz-NC and Pgz-NC in coma that is lost in brain death (*bottom*; the third pair of traces from the top show a superposition of Fz-NC and Pgz-NC). Upper left: 18 years, male, brain injury, 30 h after brain death. Upper right: 36 years, female, brain injury, 12 h after brain death. Lower traces: 48 years, male, subarachnoid haemorrhage, 60 h before (left) and 12 h after (right) brain death.

Cortical N20 (in C₃'-Fz and C₄'-Fz) was per definitionem lacking in all cases of brain death, but was also bilaterally lost in 36.1% of the comatose patients. Nearly all (94.9%) of the latter patients eventually died. All comatose patients whose SEP are shown in Figs 1, 2, 3, 5 and 7 had lost cortical potentials.

Diseases 'imitating' the brain death SEP pattern

Two clinical pictures different from (whole-)brain death presented with SEP findings that could not be distinguished from those in brain death: isolated brainstem death (one case) and complete tetraplegia after odontoid fracture (one patient). In these patients, additional tests including transcranial Doppler ultrasound, EEG and brainstem AEP revealed the preserved function or blood circulation in supratentorial structures or in the brainstem.

Possible findings with caudal displacement of the nasopharyngeal electrode

In two brain dead patients with an inadvertent caudal displacement of the nasopharyngeal lead (bending down

along the tracheal tube), a predominantly scalp-positive triphasic potential was observed in Fz-Pgz. After correction of the electrode position there was an inverse P14 potential. This phenomenon could not be constantly reproduced in other patients (when the electrode was deliberately displaced).

Discussion

The median nerve somatosensory evoked far-field potential P14 is bilobed in some of the recordings in normals or patients; therefore, some authors distinguish between P13 and P14 or refer to this potential as the P13/P14 complex (Desmedt and Cheron, 1982; Yamada *et al.*, 1986). In this discussion of our own data, we will use only the term P14. The exact site of origin of P14 is still under discussion. There is no doubt that the origin lies near the level of, and above, the foramen magnum. The assumptions concerning the anatomical structures generating P14 (or the P13/P14 complex) include the medial lemniscus, the cuneate nucleus, the rostral part of the dorsal columns and the brainstem nuclei getting inputs from medial lemniscal fibres. Possible physiological mechanisms are the propagation of action potential volleys (in the medial lemniscus or dorsal columns)

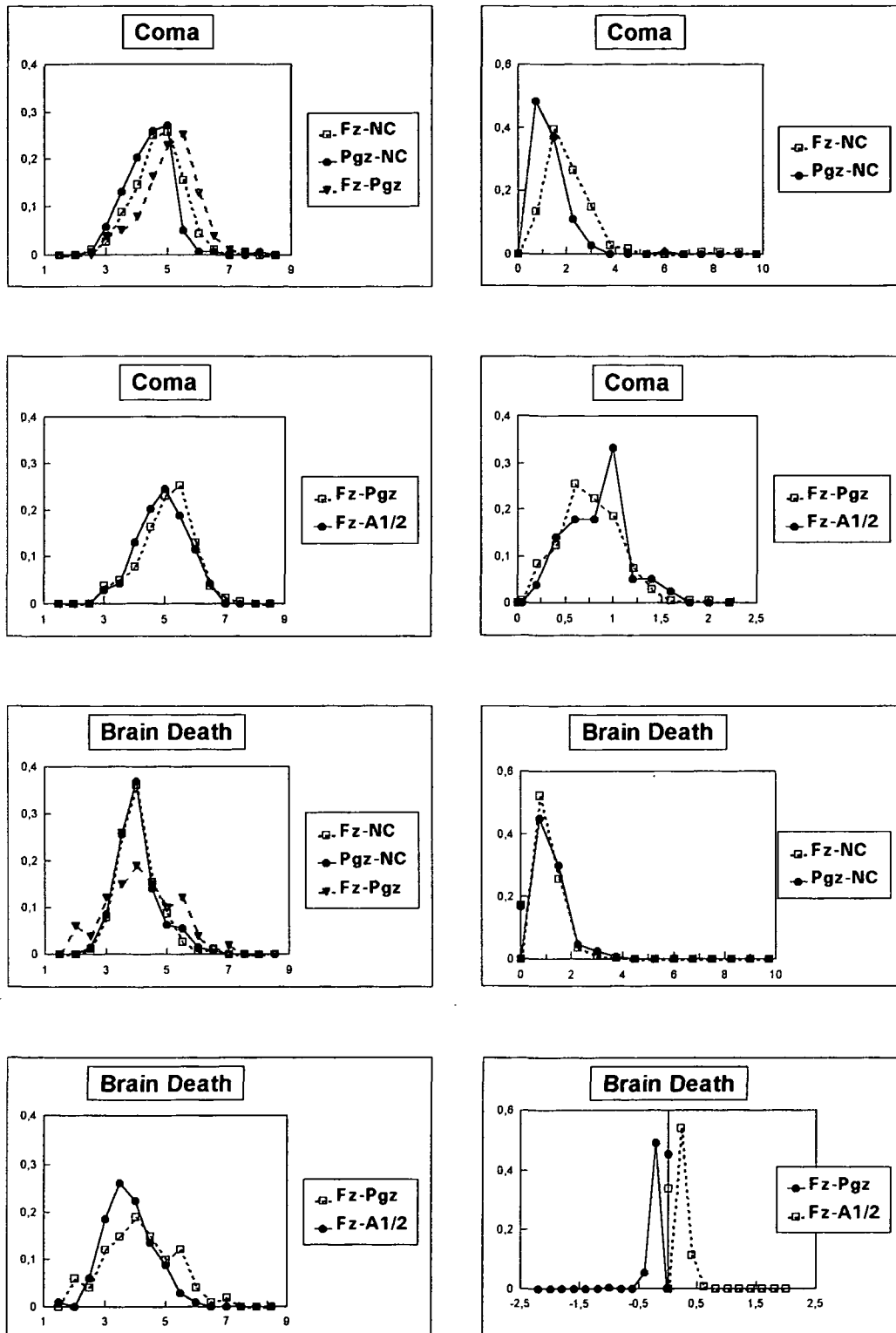


Fig. 6 Comparison of P14 parameter distributions from different derivations in coma and brain death. *Left column:* P14-P9 interpeak intervals (ms; referred to P9 in Fz-NC); *right column:* P14 amplitudes (μV) (Fz-A1/2, Fz-Pgz) and P14/P9 amplitude ratios (Fz-NC, Pgz-NC). For recordings from Fz-Pgz in brain death, values of the inverse P14 potential (negative amplitude) are given. Vertical axes indicate relative frequencies.

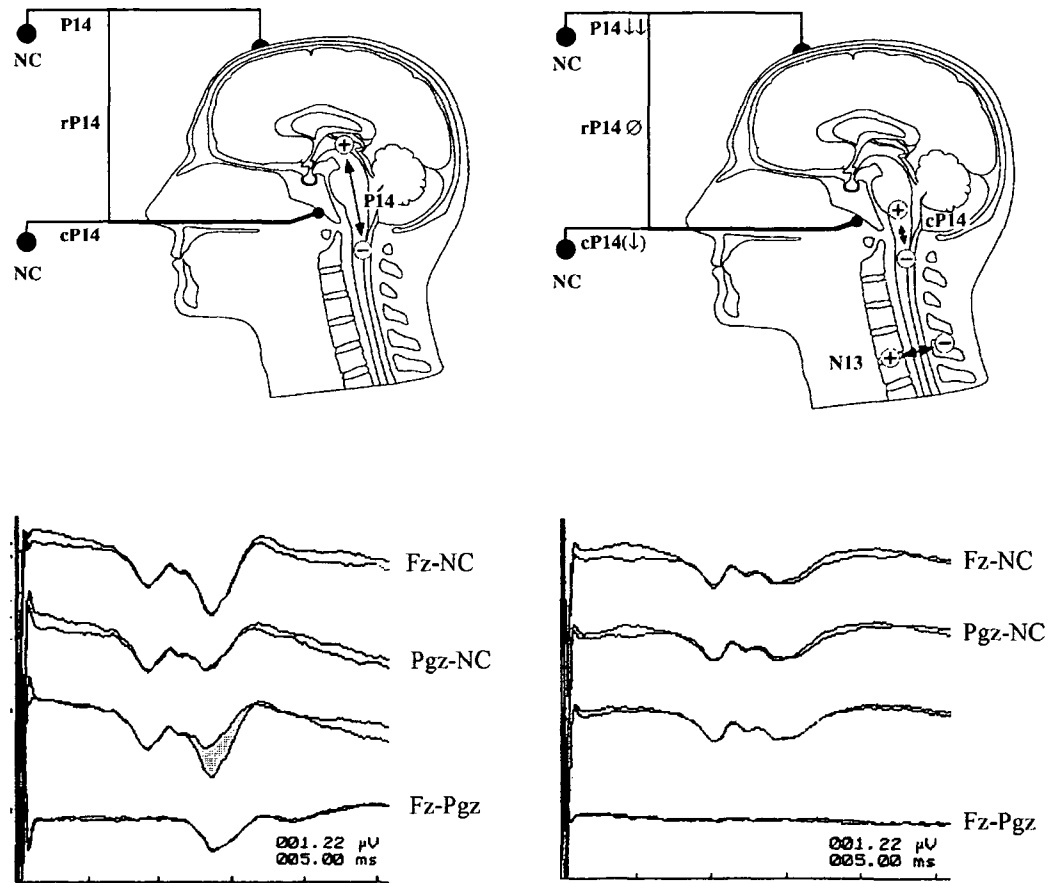


Fig. 7 Hypothesis of the projection of different rostral and caudal components (rP14, cP14) of the P14 dipole into Fz-NC, Pgz-NC and Fz-Pgz recordings. The third pair of traces from the top are a superposition of the upper two (averaged). In coma (*left*), the shaded area indicates the rostral P14 component picked up by Fz-Pgz. In brain death (*right*), the major rostral part of P14 is lost and the difference between Fz-NC and Pgz-NC (equivalent to Fz-Pgz) is zero. *Left*: 58 years, female, subarachnoid haemorrhage. *Right*: 22 years, male, brain injury, 10 h after brain death.

or postsynaptic responses (originating from synapses in the cuneate nucleus or in the brainstem nuclei).

The physical and physiological basis of the generation of far-field activity has been dealt with in a number of theoretical and empirical studies (Kimura *et al.*, 1983, 1986; Yamada *et al.*, 1985; Cunningham *et al.*, 1986; Stegemann *et al.*, 1987; Deupree and Jewett, 1988; Kaji and Sumner, 1990; Dumitru and Jewett, 1993; Dumitru and King, 1993). Mechanisms relevant to the generation of far-field potentials are changes in geometry or conductivity of the volume conductor, bending or cutting of nerves or nerve tracts, etc. Therefore, the traversing of the afferent action potential volley through the foramen magnum is considered one of the mechanisms of P14 generation (Kimura *et al.*, 1983; Lüders *et al.*, 1983).

Several clinical studies and observations in patients with circumscribed lesions have shown that the neural structures generating P14 undoubtedly involve the medial lemniscus, but may encompass also the cuneate nucleus or the rostral

part of the dorsal columns (Desmedt and Cheron, 1980; Anziska and Cracco, 1981; Mauguière *et al.*, 1983a, b; Iragai 1984; Suzuki and Mayanagi, 1984; Emerson and Pedley, 1986; Yamada *et al.*, 1986; Tomberg *et al.*, 1991; Restuccia *et al.*, 1995). Many clinical findings and experimental data speak in favour of a postsynaptic origin (Mauguière *et al.*, 1983b; Hashimoto 1984; Mauguière and Ibáñez, 1985; Delestre *et al.*, 1986; Katayama and Tsubokawa, 1987; Jacobson and Tew, 1988; Convers *et al.*, 1989; Ibáñez *et al.*, 1989; Urasaki *et al.*, 1990; Morioka *et al.*, 1991b; Buchner *et al.*, 1992; Mavroudakos *et al.*, 1993; Urasaki *et al.*, 1993; Mavroudakos *et al.*, 1994) but several studies give evidence for a presynaptic origin, too (Lüders *et al.*, 1983; Møller *et al.*, 1986; Kaji and Sumner, 1987; Morioka *et al.*, 1991a; Buchner *et al.*, 1992; Mavroudakos *et al.*, 1993; Restuccia *et al.*, 1995). In particular, a dissociation between normal P13 and abnormal P14 in non-cephalic referenced scalp recordings, that was described in individual patients with lesions in the lower brainstem or at the cervicomedullary

junction, led to the hypothesis of a presynaptic origin for P13 and a postsynaptic origin for P14 (Delestre *et al.*, 1986; Kaji and Sumnet, 1987; Mavroudakos *et al.*, 1993; Restuccia *et al.*, 1995). In any case, the clinical data speak in favour of (i) the generation of P14 (or P13/P14) by neural structures belonging to the medial lemniscal system, (ii) the origin being around the level of the foramen magnum and (iii) rostro-caudal extension of the structures generating that potential.

Practical aspects

For SEP testing in brain death diagnosis, it follows that P14 must show at least an amplitude decrease (or a loss) during the progression from coma to brain death. This has indeed been reported in several studies (Anziska and Cracco, 1980; Mauguière *et al.*, 1982; Belsh and Chokroverty, 1987; Brunko and Zegers de Beyl, 1987; Stöhr *et al.*, 1987; Besser *et al.*, 1988; Buchner *et al.*, 1988; Wagner, 1989, 1991; Facco *et al.*, 1990). As was shown, e.g. by Buchner *et al.* (1988), this amplitude decrease may be rather gradual or it may be abrupt, probably reflecting a slow or steep rise in intracranial pressure leading to a rostro-caudal deterioration of CNS structures. A reduced P14 amplitude (or a loss) in brain death was invariably found in our study too. Interestingly, we also observed a (statistically significant) decrease in P14 latency in all derivations studied; this finding has, to the best of our knowledge, as yet not been reported by others. This latency decrease, however, was of no direct practical significance as it did not allow an electrophysiological distinction between comatose and brain dead patients.

When comparing the different electrode montages, it is important to note that a preserved P14 potential in Fz-NC, Fz-A_{1/2} and Pgz-NC has been found in ~90% of brain dead patients and that P14 in these derivations might be of relatively low amplitude even in coma in some cases. In brain death the Fz-Pgz recordings were the only ones which never yielded the scalp positivity in the 14 ms range that was constantly found in coma. This result has also been confirmed in a preliminary report by others (Roncucci *et al.*, 1994). So, Fz-Pgz was the only electrode montage where a P14 parameter (amplitude) showed no overlap between comatose and brain dead patients. However, isolated brainstem death or a high cervical transverse lesion of the cord still had to be excluded (Wagner *et al.*, 1993).

Another important question in this context is the temporal relationship between brain death and the changes in the P14 potential. In >30 of the 181 patients, SEP were recorded only a few hours before or after clinical criteria for brain death were met. In one patient, brain death obviously occurred during SEP testing. Even in these cases, when the interval between SEP testing and brain death was short, P14 in Fz-Pgz was preserved in coma and lost in brain death. It is therefore highly probable that the loss of P14 in Fz-Pgz coincides with the establishment of brain death.

From an empirical standpoint, the median nerve SEP from

Fz-Pgz can be recommended for the electrophysiological confirmation of brain death, when the following conditions are satisfied: (i) there is a diffuse brainstem impairment (focal lesion of the medial lemniscus may result in a loss of P14 without brain death), and other causes of lemniscal pathway dysfunction, like isolated brainstem death or a high cervical transverse cord lesion, have been excluded by case history, CT/MR scans, transcranial Doppler and EEG recordings and/or AEP recording (Frowein *et al.*, 1987); (ii) spinal (N13) and peripheral (P9) potentials are preserved, verifying valid stimulation and recording techniques; (iii) there is a primary brain lesion underlying the clinical syndrome (patients with brain injury secondary to extracerebral diseases have not been studied as yet) (Wagner, 1994).

In clinical experience, these conditions are fulfilled in nearly all patients who are found to be brain dead after a severe and primary brain injury.

Theoretical considerations

Besides the practical aspects, theoretical conclusions with regard to the origin of P14 may be drawn from the findings in the various SEP derivations. The following hypotheses are proposed in order to explain the differences in P14 latency and amplitude in the different derivations in coma (Fig. 7): (i) the P14 generating structures have some longitudinal extension; (ii) electrodes Fz, Pgz and NC have different topographical relationships to the P14 generator dipole—Fz is far rostral to the positive pole, NC is far caudal to the negative pole, Pgz lies at the level of the caudal part of the P14 dipole, with the main rostral part of the generator being above, the smaller caudal part of the generator being below the nasopharyngeal lead; for the projection of the P14 generator into the different electrode montages it appears that (iii) Fz-NC picks up the whole P14 dipole, (iv) Pgz-NC picks up only the (smaller) caudal part of P14 ('caudal P14', cP14) and that (v) Fz-Pgz picks up only the (main) rostral part of P14 ('rostral P14', rP14). Also (vi) as earlobe electrodes lie somewhat more caudal than Pgz, Fz-A_{1/2} records a rostral P14 component that reaches slightly more caudally than rP14 in Fz-Pgz.

The assumption that different parts or components of the P14 dipole project differently onto these electrode montages would explain, on the one hand, the amplitude differences, as the amplitude of the whole P14 dipole (P14 in Fz-NC) must be larger than that of its caudal (cP14 in Pgz-NC) or rostral (rP14 in Fz-Pgz) parts. On the other hand, latency differences would be explained by the fact that the action potential volley passes earlier through caudal than through rostral parts of the lemniscal pathway. Indeed, we found statistically significant latency differences between P14 (in Fz-NC), cP14 (in Pgz-NC) and rP14 (in Fz-Pgz). If conduction velocity in the medial lemniscus is estimated at 40 m s⁻¹ (Desmedt and Cheron, 1980), the latency difference of 0.64 ms between cP14 in Pgz-NC (4.22 ms) and rP14 in Fz-Pgz (4.86 ms) would indicate a spatial separation of

~25 mm between the amplitude maxima of the two potentials, which reasonably matches the anatomical data. This is in line with the findings from recordings with deep electrodes reported by Suzuki and Mayanagi (1984), who observed a longitudinal extension of P14 generating structures (medial lemniscus) of several centimetres and described in one patient records from different levels ventral to the brainstem with a P14 latency difference between medulla oblongata and midbrain of 0.7 ms, exactly matching our data. Similar conclusions with respect to the longitudinal extension of the P14 dipole have been drawn by other authors from intracranial recordings (Hashimoto 1984; Urasaki *et al.*, 1990; Morioka *et al.*, 1991b; Urasaki *et al.*, 1993) and by Anziska and Cracco (1981) from earlobe derivations (scalp-A_{1/2}, A_{1/2}-NC). On the other hand, the hypothesis of an involvement of rostral parts of the medial lemniscus in the generation of P14 is at variance with findings in (conscious) patients suffering from pontine lesions encompassing the lemniscal fibres, who show a normal P14 in non-cephalic referenced scalp recordings, as was reported by Convers *et al.* (1989). In a recently published detailed study on P13 and P14 recordings using different electrode montages, Restuccia *et al.* (1995) found in normal subjects mean P14 latency differences between Fz-NC (14.09 ms) or other scalp derivations (up to 14.15 ms) and Pgz-NC (13.84 ms) of 0.25–0.31 ms. These were not significant differences, probably due to the small number of subjects ($n = 6$), but they closely matched our findings (the P14 difference between Fz-NC and Pgz-NC was 0.27 ms; *see* Table 2). In patients with circumscribed lesions of the cervico-medullary junction, they found a dissociation between a normal P13 and an abnormal P14 in scalp derivations. These authors interpreted their data by suggesting that P13 was generated below the cervico-medullary junction and P14 above: their findings in patients with circumscribed lesions of the upper cervical cord confirmed some longitudinal extension of the neural structures generating the P13/P14 complex.

The differences between caudal and rostral P14 found in our patients cannot be explained merely by a reflection of pre- and postsynaptic activity, or a distinction between P13 and P14. First, the amplitude and latency differences between cP14 (in Pgz-NC), P14 (in Fz-NC) and rP14 (in Fz-Pgz) were seen independently of the presence of one or two of the peaks in the P14 ms range (a separate P13 component was the exception rather than the rule in our patients). Secondly, the statistically significant P14 latency and amplitude differences between nasopharyngeal and earlobe derivations (Fz-Pgz and Fz-A_{1/2}) can hardly be explained on the basis of a distinction between a presynaptic P13 and a postsynaptic P14 alone. The hypothesis of a single P14 dipole model is, of course, a simplification and does not exclude other possible interpretations. However, it does allow us to explain the main features of our data.

To explain the findings in brain death, the following hypotheses are proposed (*see* Fig. 7). (i) The greater rostral

part of the P14 generator dipole is inactivated in brain death; the very caudal segments, however, may be intact for some time. In other words, P14 is reduced to less than the cP14 component in brain death. (ii) The nasopharyngeal electrode invariably lies above the (positive) rostral end of the residual P14 generator dipole in brain death, irrespective of the longitudinal extension of the remaining dipole. (iii) It follows that both Fz and Pgz are on the positive side of the dipole field in brain death, Pgz being nearer to the dipole and therefore lying on an isopotential line with a higher positive potential; this results in a scalp-negative component (inverse P14) in Fz-Pgz in brain death. (iv) Earlobe electrodes (lying somewhat more caudal than Pgz) pick up a rostral P14 segment reaching more caudally, occasionally going below the rostral end of the residual P14 dipole in brain death. This results in a preserved (scalp-positive) P14 in Fz-A_{1/2} in brain death.

These hypotheses would explain the P14 amplitude and latency differences between coma and brain death, as the remaining caudal P14 segment in brain death must have a lower amplitude and latency than the whole dipole in coma. The differences between Fz-Pgz and Fz-A_{1/2} recordings can be attributed to the lower position of A_{1/2} compared with Pgz with respect to the neuraxis; this different position of these electrodes is clearly seen in X-rays too (*see* Wagner, 1991, fig. 10). That even slight rostro-caudal differences in electrode position may yield different potentials, is demonstrated by the finding that displacement of the nasopharyngeal lead can result in a predominantly scalp-positive potential also in brain death, when this electrode lies a few centimetres more caudally. The interesting phenomenon of an inversion of P14 in Fz-Pgz has been found exclusively in brain death, never in coma. Its explanation by a special topographical relationship of both electrodes to the residual dipole field is supported by intracranial derivations reported by Suzuki and Mayanagi (1984) who found a scalp-negative P14 potential when recording from Fz with a thalamic reference electrode. The low amplitude triphasic wave in Fz-Pgz, observed in some of our brain dead patients, may reflect a transition between a normal monophasic P14 in coma and an inverse P14 in brain death (*see* Fig. 3, lower left).

The most probable reason for a residual caudal P14 activity in brain death seems to be a contribution of (not affected) rostral parts of the dorsal columns to the generation of that potential. This would be in line with the findings of Restuccia *et al.* (1995) who clearly demonstrated that the upper cervical spinal cord contributes to the generation of the P13/P14 complex. Another explanation would be the origin of cP14 in very caudal parts of the medial lemniscus in the lower brainstem that have been displaced by an elevated intracranial pressure through the foramen magnum, thus leaving blood circulation intact for some time. This possibility has already been discussed by Goldie *et al.* (1981).

Whether the SEP findings in brain death merely reflect the preservation of a presynaptic component, while postsynaptic

P14 activity is lost, is debatable. Besides the above-stated arguments against a simple association of cP14 with presynaptic P13, and rP14 with postsynaptic P14, two points are important. First the statistically significant P14 latency and amplitude differences in Pgz-NC, between coma and death, show that the residual P14 activity in brain death is not just the same as the P14 activity picked up by Pgz-NC in coma, i.e. changes of P14 in brain death cannot be fully explained by the reduction of P14 to that activity that is recorded from nasopharyngeal electrodes in coma. Secondly, we have noticed in some serial recordings gradual decreases in P14 amplitude and latency after brain death (see Wagner 1991, fig. 8) as was described in detail by Buchner *et al.* (1988), suggesting a step-wise rostro-caudal failure of P14 generating structures. Again, the assumption of a simple (and simplifying) single dipole model, with a 'shrinking' of that dipole in brain death, explains the SEP findings better than a simple distinction between pre- and postsynaptic P13/P14 activity. In any case, within the longitudinally extended neural structures generating that potential, a rostral-to-caudal sequential failure during brain death must be postulated. This failure/destruction may start above the cuneate nucleus and eventually reach presynaptic structures anywhere in the upper dorsal columns.

In conclusion, P14 recordings with different electrode montages are of practical value in brain death diagnosis (in particular, when using Fz-Pgz) as well as of theoretical interest with regard to the site of origin of P14 and the differential projection of varying rostral and caudal segments of the generator dipole into various SEP recording configurations.

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