

## Auditory Event Related Potentials in Violent and Nonviolent Prisoners\*

**Miles E. Drake, Ann Pakalnis, Modestine E. Brown, and Sharon A. Hietter**

EEG and Evoked Potential Laboratory, The Ohio State University Hospitals, Department of Neurology,  
The Ohio State University College of Medicine, Columbus, OH 43210, USA

**Summary.** The neurophysiologic substrates of violence and aggression have been extensively studied. Although the EEG is often normal in violent persons any abnormalities are generally nonspecific in nature. Evoked potentials have been infrequently used to study such behavior disorders. Long latency auditory event-related potentials (AEPs) were studied in prison inmates incarcerated for unmotivated violent crimes or violence during the commission of other crimes, and in prisoners jailed for non-violent offenses. Habitually violent inmates were compared with an equal number of non-violent prisoners and neurologically and audiotically intact controls. AEPs were recorded to an "oddball" paradigm from vertex and left and right temporal electrodes. None of the prisoners had latencies more than 3 standard deviations beyond the normal group mean. N1 and P2 components were longer in latency and lower in amplitude in prisoners than in controls, but this was not statistically significant. Violent and non-violent individuals did not differ in these measures. P3 was significantly prolonged in latency in violent inmates, but not in those who had committed a violent act. All components were longer in latency on the right in violent prisoners, while amplitudes were lower but not significantly so. This asymmetry was not present in non-violent prisoners or control subjects. Caution is needed in identifying differences between criminals and the general population, and in seeking markers of violent behavior. These findings may indicate cerebral dysfunction in some perpetrators of antisocial behavior, however, and suggest that evidence of non-dominant ce-

rebral hemisphere dysfunction, possibly subtle and chronic, may be found in some habitually violent individuals.

**Key words:** Auditory event related potentials – Prisoners – Violence

---

### Introduction

The physiological basis of violence and aggressive behavior has been the subject of intensive study [12]. Experimental lesions producing rage, ferocity, aggression, and combinations of these have been described [1]. Organic neurologic dysfunction, sometimes of subtle character, has been demonstrated in many patients with the episodic dyscontrol syndrome, in which violent and aggressive acts may occur with little or no provocation [8]. A variety of EEG abnormalities have been reported in violent prisoners, psychopathic patients and felons, and criminals generally, but they have been non-specific, and have correlated poorly with clinical features of violence or criminality, except for the frequent presence of neurologic signs of subtle brain disorder generally, and of frontal or temporal lobe dysfunction particularly [5]. Auditory evoked potentials have been studied in patients with antisocial behavior [10] and in brain injured individuals with rage attacks [4], and differences from controls have been demonstrated. Long latency auditory event related potentials, which have been related to limbic system function [13] and shown to change in temporal lobe and hippocampal disorders [7], have not been studied in violent or aggressive persons to our knowledge. We compared AEPs in normal con-

---

\*Presented in part at the 40th Annual Meeting, Central Association of Electroencephalographers, Chicago, April 1987. Supported in part by the Denman Fund for Epilepsy Research, The Ohio State University

Offprint requests to: A. Pakalnis

trols, prisoners incarcerated for non-violent offenses, and habitually violent offenders.

## Subjects and Methods

Prisoners with normal neurologic examinations, normal EEGs and computed tomographic brain scans, no history of prescribed medication use, no history of head trauma with hospitalization or prolonged unconsciousness, and no evidence of mental retardation or mitigating psychiatric diagnosis were identified during the course of neurologic consultation for the Ohio Department of Rehabilitation and Correction during 1986. 20 prisoners with a mean age of 27.1 years were incarcerated at a maximum security facility for more than one violent crime in which a domestic dispute, alcohol, or drugs did not play a role, or had been transferred from another penitentiary for habitual violent behavior during their incarceration for a non-violent offense. 20 additional inmates with a mean age of 29.7 years were studied from medium and minimum security facilities, to which they had been committed for nonviolent offenses with no recidivism or violence during their sentences. 19 normal individuals, taking no medications and without auditory disturbance or neurologic disorder, had a mean age of 31.7 years.

**Table 1.** AEP Latency and Amplitude from vertex-linked ear montages control subjects, violent inmates and nonviolent prisoners

Waveform	Controls	Violent inmates	Other inmates
<i>Latency (MS)</i>			
N1	91.4 ± 11.3	95.3 ± 7.7	94.0 ± 10.1
P2	161.2 ± 11.1	164.9 ± 10.3	165.5 ± 14.3
N2	211.6 ± 21.6	223.3 ± 20.7	217.5 ± 21.8
P3	320.5 ± 27.1	334.5 ± 23.1	322.4 ± 18.7
<i>Amplitude (UV)</i>			
N1	11.1 ± 4.4	9.8 ± 5.4	10.0 ± 6.6
N2	10.5 ± 6.1	5.4 ± 2.9	6.6 ± 6.0
P3	12.1 ± 4.4	10.3 ± 0.3*	10.7 ± 1.5*

\* = < 0.05

**Table 2.** AEP Latencies from right and left temporal montages in control subjects, violent inmates, and non-violent prisoners

	N1	P2	N2	P3
<i>Controls</i>				
Cz-T3	92.9 ± 13.5	189.3 ± 31.6	244.5 ± 30.2	330.0 ± 30.2
Cu-T4	89.6 ± 10.6	186.8 ± 22.0	241.7 ± 37.3	332.2 ± 33.9
<i>Violent inmates</i>				
Cz-T3	95.4 ± 13.6	180.5 ± 33.3	254.4 ± 27.6	350.2 ± 39.2
Cz-T4	94.3 ± 11.2	189.3 ± 22.2	276.9 ± 26.1	379.2 ± 26.1*
<i>Other inmates</i>				
Cz-T3	90.6 ± 11.5	180.1 ± 19.3	264.8 ± 39.2	354.8 ± 39.2
Cz-T4	92.6 ± 15.3	178.6 ± 23.3	266.4 ± 36.1	352.2 ± 39.0

\* =  $P < 0.05$

Auditory event related potentials were recorded in an "odd ball" paradigm, in which rare (3,000 Hz) tones were interspersed among common (1,000 Hz) tone stimuli in a pseudo-random fashion. Patients and subjects were instructed to attend to infrequent high pitched tones, and to attempt to keep an accurate count of them. 1,000 and 3,000 Hz tone pips were presented through shielded headphones in an 80/20 ratio by a Nicolet SM 200 auditory stimulator (Nicolet Instrument Corporation, Madison, WI, USA). Tones had a 50 ms plateau time and 5 ms rise – fall time, and were presented at 70 dBHL bilaterally, with an interstimulus interval of 1.1 s. AEPs were recorded from Grass tin cup electrodes (Grass Instrument Company, Quincy, MA, USA) placed at midline (Cz), left temporal (T3), and right temporal (T4) positions of the International 10/20 system and on the ears (A1 and A2). Recording montages were Cz-A1 + A2, Cz-T3, and Cz-T4. Analysis time was 1,000 ms, filter band pass was 1–100 Hz, and 400 averages were replicated in each patient and subject. Trials with apparent drowsiness or a rare tone count in error by more than 5 were rejected, and automatic rejection of myogenic or ocular artifact was obtained.

The latencies and amplitudes of the N1, P2, N2, and P3 components of the AEPs were measured. Prison inmates and controls and violent and non-violent offenders were serially compared in two tailed *T* tests.

## Results

Latency and amplitude measurements recorded from the Cz-A1 + A2 montage are summarized in Table 1. Violent and non-violent inmates had longer latency and lower amplitude of N1 and P2 wave forms than did normal controls. P3 was significantly longer in latency among violent inmates ( $P < 0.05$ ), but did not differ between violent and non-violent individuals. Violent and non-violent inmates had lower N2 amplitudes which were not statistically significant, while P3 was significantly lower in amplitude ( $P < 0.05$ ). Violent and non-violent inmates did not differ from each other with respect to N2 and P3 amplitudes, however.

**Table 3.** AEP amplitudes from right and left temporal montage in control subjects, violent inmates, and non-violent prisoners

	N1	P2	N2	P3
<i>Controls</i>				
Cz-T3	6.4 ± 5.1	4.3 ± 1.9	7.8 ± 3.1	8.9 ± 5.7
Cz-T4	6.8 ± 6.0	4.9 ± 2.3	8.4 ± 5.2	8.9 ± 5.1
<i>Violent inmates</i>				
Cz-T3	6.6 ± 6.3	4.5 ± 1.3	6.9 ± 5.3	8.8 ± 3.3
Cz-T4	5.9 ± 2.3	4.2 ± 2.2	5.2 ± 4.0*	7.1 ± 5.6*
<i>Other inmates</i>				
Cz-T3	6.0 ± 3.8	4.5 ± 1.8	8.2 ± 3.8	8.8 ± 4.2
Cz-T4	6.5 ± 3.3	4.7 ± 2.2	8.3 ± 4.4	9.1 ± 5.5

\* =  $P < 0.05$

Latency measurements from left and right temporal recordings are summarized in Table 2, and Table 3 shows amplitude values from left and right temporal electrodes. P2 and N2 were longer in latency in Cz-T4 than in other derivations among the violent inmates, but this relative prolongation was not statistically significant. P3 ( $P < 0.05$ ) was significantly prolonged, however. The asymmetry was not observed in non-violent prisoners or in controls. A similar asymmetry in AEP amplitude was observed in the right temporal derivation among only the violent inmates, with an amplitude reduction of N2 which was not significant, and P3 amplitude reduction of a lesser degree of significance ( $P < 0.1$ ).

## Discussion

These findings suggest differences in long latency AEPs between some incarcerated offenders and age-matched controls. The significance of these differences is uncertain, and it is likely to reflect a variety of factors. The differences cannot be ascribed to identifiable neurologic disorder, however, nor to auditory impairment, as all of these were absent in all three groups. The both prisoner groups all had neurologic complaints, having been referred initially for neurologic evaluation, but these complaints were in all cases unattended by objective evidence of underlying neurologic disorder. The prisoners tested did not differ from the general inmate population in the presence of neurologic disorder, but some could conceivably have had a physical basis for their complaints not detectable at the time of their clinical evaluation and electrophysiologic study. Controls and both inmate groups were ostensibly taking no medications, which is an important factor since drugs are known to effect

AEPs [9]. It is certainly possible that some inmates could have taken surreptitiously medications which might have influenced their AEPs. Head trauma is also relevant, as head injury is unduly frequent in the incarcerated population [2, 14], and has been shown to effect AEPs [6]. The prisoners studied had no clearly identified episodes of major head injury, but could have had the cumulative effects of unreported or undocumented head trauma.

A discrepancy in AEPs was observed between violent and nonviolent offenders, with attenuation and latency prolongation of AEP components over the right hemisphere. This asymmetry was not observed in nonviolent offenders or in controls, and suggests the possibility of subtle right hemisphere dysfunction in the former group, although the etiology of such dysfunction in neurologically normal individuals, and its relationship to their violent offenses, is uncertain. Further neurophysiologic study of these and other violent individuals, perhaps utilizing computerized EEG frequency analysis or topographic brain mapping, would be appropriate to replicate and clarify this observation, and might be supplemented by neuropsychological assessment of various features of right and left hemisphere function.

These observations accord with some evidence that the "minor" hemisphere may be of crucial importance in affective communication [15–17]. Right hemisphere lesions or disorders might interfere with interpersonal relations and might result in violent altercations or social disability. An association between predominantly right temporal EEG paroxysms and such dramatic personality disturbances as dissociative states and multiple personality has been reported [11]. Weintraub and Mesulam [18] described a developmental disorder of the right cerebral hemisphere, characterized by affective misidentification and difficulty in acquiring skills and interpersonal communication; it is possible that some individuals prone to violent behavior, who have a high incidence of "minimal brain dysfunction" [8], may represent an exaggerated form of this developmental disorder. Some neurophysiologic evidence [3] suggests dysfunction of the non-dominant hemisphere in some personality disorders. We are not aware of previous studies utilizing long-latency auditory event related components, but believe that this may be an appropriate tool with which to study cognitive function and information processing, and to assess differences in hemispheric function which might be behaviorally and socially meaningful. It is premature to assign any clinical significance to such differences, however, and medical or social application of findings suggesting cerebral dysfunction in violent persons would not be appropriate.

*Acknowledgement.* The authors appreciate the preparation of the manuscript by Mrs. Victoria L. Grimes.

## References

1. Bard T (1928) Diencephalic mechanism for the expression of rage. *Am J Physiol* 89:490–515
2. Bell CC (1986–87) Coma and the etiology of violence. *J Natl Med Assoc* 78:1167–1176; 79:79–85
3. Braun BG (1983) Neurophysiologic changes in multiple personality due to integration: A preliminary report. *Am J Clin Hypnosis* 26:84–92
4. Cannon PA, Drake ME (1986) EEG and brainstem auditory evoked potentials in brain-injured patients with rage attacks and self injurious behavior. *Clin Electroencephalogr* 17:169–172
5. Cummings JL (1985) *Clinical neuropsychiatry*. Grune and Stratton, New York, pp 127–129
6. Donchin E, Ritter W, McCallum WC (1978) Cognitive psychophysiology: The endogenous components of the ERP, in: Calloway E, Tueting P, Koslow SH (eds): *Event-related potentials in man*. Academic Press, New York, pp 349–411
7. Drake ME, Burgess RJ, Gelety TJ, Brown ME, Ford CE (1986) Long latency auditory event-related potentials in epilepsy. *Clin Electroencephalogr* 17:10–13
8. Elliott FA (1984) The episodic dyscontrol syndrome and aggression. *Neurol Clin* 2:113–129
9. Frazier SH (1974) Murder-single and multiple. *Res Publ Assoc Nerv Ment Dis* 52:304–312
10. Josef NC, Lycaki H, Chayasirisobhon S (1985) Brainstem auditory evoked potentials in anti-social personality. *Clin EEG* 16:91–92
11. Mesulam MM (1981) Dissociative states with abnormal temporal lobe EEG: Multiple personality and the illusion of possession. *Arch Neurol* 38:176–181
12. Moyer KE (1968) Kinds of aggression and their physiological basis. *Comm Behav Biol* A2:65–86
13. Okada YC, Kaufman L, Williamson SJ (1983) The hippocampal formation as a source of the slow endogenous potentials. *Clin Neurophysiol* 55:417–426
14. Pakalnis A, Hietter S, Huber SJ, Drake ME (1988) Effects of carbamazepine and valproate on long-latency auditory event-related potentials. *J Epilepsy* (in press)
15. Ross ED, Mesulam MM (1979) Dominant language functions of the right hemisphere? prosody and emotional gesturing. *Arch Neurol* 36:144–148
16. Ross ED, Harney JH, Delacoste-Utamsing C, Purdy PD (1981) How the brain integrates affective and propositional language into a unified behavioral function: Hypothesis based on clinicoanatomic evidence. *Arch Neurol* 38:745–748
17. Tucker DM, Watson RT, Heilman KM (1977) Discrimination and evocation of affectively intoned speech in patients with right parietal disease. *Neurology* 27:947–950
18. Weintraub S, Mesulam MM (1983) Developmental learning disabilities of the right hemisphere: Emotional, interpersonal, and cognitive components. *Arch Neurol* 40:463–468

Received December 1, 1987