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Human impulsive aggression: a sleep research perspective

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Abstract

Impulsive aggression is commonly associated with personality disorders, in particular antisocial and borderline personality disorders as well as with conduct disorder and intermittent explosive disorder. The relationship between impulsive aggression and testosterone is well established in many studies. One of the aims of this study was to characterize the relationship between earlier-mentioned different categorical psychiatric diagnosis describing human impulsive aggression and sleep using polysomnography and spectral power analysis. Another aim was to study the relationship between serum testosterone and sleep in persons with severe aggressive behaviour. Subjects for the study were 16 males charged with highly violent offences and ordered for a pretrial forensic psychiatric examination. The antisocials with borderline personality disorder comorbidity had significantly more awakenings and lower sleep efficiency compared with the subjects with only antisocial personality disorder. The subjects with severe conduct disorder in childhood anamnesis had higher amount of S4 sleep and higher relative theta and delta power in this sleep stage compared with males with only mild or moderate conduct disorder. The same kind of sleep architecture was associated with intermittent explosive disorder. In subgroups with higher serum testosterone levels also the amount of S4 sleep and the relative theta and delta power in this sleep stage were increased. The study gives further support to the growing evidence of brain dysfunction predisposing to severe aggressive behaviour and strengthens the view that there are different subpopulations of individuals with antisocial personality varying in impulsiveness. The differences in impulsiveness are reflected in sleep architecture as well.

Keywords: Human impulsive aggression; Testosterone; Sleep; Polysomnography; Spectral power analysis

1. Introduction

Impulsive aggressive behaviour that includes physical aggression directed towards others, self-mutilation, suicide attempts, domestic violence, substance use and property destruction presents a challenge to both research and health care system. The economic and social cost of aggressive behaviour is huge (Scott et al., 2001), and so far both pharmacological and behavioural treatment interventions have been quite ineffective (Malone et al., 2000). As a symptom, impulsive aggression cuts across a number of psychiatric disorders (Moeller, 2001), but it is commonly associated with personality disorders, in particular antisocial (APD) and borderline (BPD) personality disorders (Eronen et al., 1996; Virkkunen et al., 1996; Goodman and New, 2000, Skodol et al., 2002). In fact, genetic, neurobiological, and diagnostic studies suggest a dimensional approach to BPD symptomatology, with impulsive aggression as one of the core dimensions of the disorder (Goodman and New, 2000; Siever et al., 2002). APD is associated with a pervasive pattern of disregard for and the violation of the rights of others. Not surprisingly, the highest prevalence rates of APD are found in prisons and forensic settings (American Psychiatric Association, 2000). In a study by Fazel and Danesh (2002), 47% of male prisoners had APD. APD often co-occurs with BPD (Coid, 1993; Hudziak, 1996) and it has even been suggested that BPD represents a female form of male-predominant APD (Gunderson and Zanarini, 1987). APD is always preceded by conduct disorder (CD) before the age of 15 (American Psychiatric Association,

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2000). The essential feature of CD is a repetitive and persistent pattern of behaviour in which the basic rights of others or major age-appropriate societal norms or rules are violated (American Psychiatric Association, 2000). Impulsiveness has been found to be the best predictor of conduct problems (Vitacco and Rogers, 2001) and impulsiveness together with emotional lability may increase the likelihood of CD progressing to adult antisocial behaviour (McKay and Halperin, 2001). It has been argued that many individuals with personality disorders display clinically significant impulsive–aggressive behaviour, which cannot be specifically identified using axis II personality disorder diagnosis (Coccaro et al., 1998). In these cases, it would be better to use the diagnosis of intermittent explosive disorder (IED), which may best be regarded as a categorical expression of recurrent, problematic impulsive and aggressive behaviour (Coccaro, 2000). In addition, for research purposes, the diagnosis can also be made for individuals with APD and BPD, in cases where impulsive aggression is of specific clinical relevance (American Psychiatric Association, 2000).

The relationship between testosterone and impulsive aggression has been well established in many studies. High concentrations of testosterone have been shown to be associated with both CD and APD (Virkkunen et al., 1994; Brooks and Reddon, 1996; Stalenheim et al., 1998; Aromäki et al., 2002). In the study by Räsänen et al. (1999), personality-disordered criminals with multiple offences had higher serum testosterone levels than criminal schizophrenics or healthy controls. The role of testosterone in sleep regulation is still fairly unclear, but an association between slow wave sleep (SWS) and serum testosterone in healthy males has been reported (Leibenluft et al., 1997).

Patients with most psychiatric diagnosis have displayed significant changes in sleep parameters (Benca et al., 1992), but less is known about sleep in personality disorders including APD and BPD. In the study by Benson et al. (1990), non-affective BPD patients had less both total sleep and stage 4 sleep (S4). More waking time after sleep onset and reduced rapid eye movement sleep (REM) latency have also been reported (Battaglia et al., 1993). In the study by De La Fuente et al. (2000), BPD patients had less total sleep, longer sleep onset latency and a greater percentage of wakefulness than healthy control subjects. They also had a longer duration of REM sleep, less stage 3 sleep (S3), S4 and SWS, but there was no difference in REM latency. In APD only one sleep EEG study has been reported (Lindberg et al., 2003). In this study habitually violent offenders, all having antisocial personality disorder, had significantly more awakenings during the night and decreased sleep efficiency (SE) but quite contrary to BPD, the amount of both SWS and especially S4 were significantly increased compared with the healthy controls. In fact, APD appears to be the only psychiatric disorder associated with an increase in deep sleep. Coble et al. (1984) reported that in pre-adolescent boys with CD the number of delta waves during sleep was higher compared with healthy controls. The alcoholic, impulsive violent offenders with IED have been reported to have a profound diurnal activity rhythm disturbance (Virkkunen et al., 1994), but to our knowledge, there are no sleep EEG studies in this diagnosis group.

Greater understanding of subgroups within the broad category of persons with impulsive aggression may help to create more effective treatment interventions (Hill, 2003).

Polysomnography may provide additional information in sub-typing persons with severe aggression problems. One aim of the study was to characterize the relationship between different categorical psychiatric diagnosis describing impulsive aggression and sleep using polysomnography and spectral power analysis. Another aim was to study the relationship between serum testosterone and sleep in persons with severe impulsive aggression.

2. Material and methods

2.1. Subjects

The subjects for the study were 16 males with a history of recurrent violent acts. They were charged with violent offences and ordered for a pretrial forensic psychiatric evaluation lasting approximately two months by the Finnish National Board of Medico-Legal Affairs. The evaluation took place in a special ward of a university psychiatric hospital. Diagnoses were made by the same senior forensic psychiatrist (H.N.) using structured clinical interview SCID I and II (First et al., 1997a, First et al., 1997b). All 16 males met the DSM IV criteria for APD, and in addition six of them also for BPD (American Psychiatric Association, 1994). Subjects with a DSM-IV axis I diagnosis other than drug and alcohol dependence were excluded, as were subjects with an axis II diagnosis other than the two earlier-mentioned personality disorders. The trial records and all available background information, including medical, family, school and criminal history from childhood and adolescence to adulthood, were studied. Using these data and information from SCID-interviews, the severity of the preceding CD and the possible diagnosis of IED were evaluated. The severity of the preceding CD was rated as mild (lying, truancy, staying out dark without permission), moderate (stealing without confronting a victim, vandalism) or severe (forced sex, physical cruelty, use of a weapon, stealing while confronting a victim, breaking and entering) using the descriptive guidelines of DSM-IV-R (American Psychia-

tric Association, 2000). The essential features of IED (the occurrence of discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property = criterion A and the degree of aggressiveness expressed during an episode is grossly out of proportion to any provocation = criterion B) were evaluated and, cases in with both criteria were positive, the diagnosis of IED was made. In the case of one subject, not enough information was available to decide whether or not he had IED. The distribution of subjects to different clinical diagnosis groups and the overlap in the distribution can be seen in Table 1. The laboratory tests including serum testosterone were taken at 8 a.m. after the second polysomnography recording. Fifteen males had the history of alcoholism; their average age when they started to use alcohol was 13 years. However, because of staying in prison before the psychiatric evaluation, subjects had a mean alcohol abstinence of ±SEM 4.8±0.41 months. In the laboratory tests, S-GT and S-CDT were within normal limits in all cases. Urine screening for illicit drugs was performed just before the sleep examination and it was negative in all cases. Brain MRI (1.5 T) disclosed no abnormality. Waking EEG was normal in 13 cases, while three subjects had mild slowing of EEG background activity. As part of a forensic psychiatric examination the WAIS-R IQ was evaluated. It was mean±SEM 93.5±3.06 with all subjects being scored above the border of mental retardation. The mean duration of formal education was 8.7 years. Subjects did not use any medication during the study period.

Eleven controls consisted of hospital staff and students. They were age-matched and healthy without a history of somatic, psychiatric or neurological disorders or substance abuse. As a part of psychiatric interview, the SCID non-patient version was filled in. Brain MRI (1.5 T) excluded structural brain abnormalities and blood tests (including liver function) and electrocardiograms disclosed no abnormality. The mean duration of formal education was 13.2 years. Controls were asked to avoid alcohol, drugs or medication 2 weeks prior the sleep examinations. Both the subjects and the controls filled in the Beck Depression Inventory Scale (BDI) before the sleep recordings.

2.2. Sleep examination

2.2.1. Polysomnography and scoring of sleep stages

The sleep recordings were made during two consecutive nights. The first night was the adaptation night; the second night was considered for the study. Subjects slept on the hospital ward. Controls entered the hospital in the afternoon and slept in the guest room, corresponding to the facilities at the ward. EEG was recorded using a mobile recording unit (Medilog 4-24 recorder, Oxford Medical Systems, UK) allowing the subjects to move freely. EMG surface electrodes were placed submentally, EOG electrodes according to Rechtschaffen–Kales standards (Rechtschaffen and Kales, 1968). During normal sleep the theta band has occipital predominance (Werth et al., 1997; Finelli et al., 2001), indicating that detection of changes in theta power is most sensitive from EEG derivations in this area. As we wanted to maximise the detection of theta power, an occipital derivation was chosen for recording. Right-handed persons were recorded on derivation O2–P4 and left-handed on derivation O1–P3 according to the 10–20 system. The signal was analysed with a Nightingale sleep analysator (Judex, Copenhagen, Denmark) (low-and high-pass filters 25 and 0.2 Hz). As the

Table 1
The overlap of different diagnostic subgroups among 16 male offenders

Subject	Age	Index crime	ASP	BPD	CDs	CDm	IED+	IED-
1	19	Attempted manslaughter	x		x			
2	46	Attempted manslaughter	x		x		x	
3	27	Murder	x			x		x
4	18	Assault	x			x	x	
5	34	Attempted manslaughter	x		x		x	
6	27	Attempted manslaughter	x		x			x
7	40	Manslaughter	x	x		x	x	
8	45	Manslaughter	x			x		x
9	39	Manslaughter	x	x	x		x	
10	39	Manslaughter	x	x		x		x
11	23	Assault	x	x		x	x	
12	20	Murder	x	x	x		x	
13	48	Attempted manslaughter	x	x		x		x
14	29	Manslaughter	x			x	x	
15	20	Murder and attempted manslaughter	x		x		x	
16	18	Attempted manslaughter	x		x		x	

ASP, antisocial personality disorder; BPD, borderline personality disorder; CDs, conduct disorder type severe; CDm, conduct disorder type mild or moderate; IED+, intermittent explosive disorder; IED−, no intermittent explosive disorder.

derivation deviates from the standard C4–A1 arrangement by Rechtschaffen–Kales, we carefully calibrated the signal by comparing it with the signal obtained from the standard derivation C4–A1. Details of the calibration procedure have been published previously (Lindberg et al., 2002). All data for the analysis were scored by the same scorer (N.L.).

In order to study the distribution of sleep epochs through the night the sleep period was divided to first and second half of the actual sleep time (AST). The distribution of number of epochs was calculated as follows: (the number of epochs during the first half of the night−the number of epochs during the second half of the night)/ the number of epochs during the first half of the night×100. Thus a positive figure reflects the relative increase of a given sleep stage in the first half of AST.

2.2.2. Power spectrum

The EEG-signal was analysed after Fast Fourier transformation (sampling rate 50 Hz) in 5 frequency bins (band widths: δ: 0.5–3.5 Hz, θ: 3.5–8.0 Hz, α: 8.0–12.0 Hz, σ: 12.0–14.5 Hz and β: 14.5–25.0 Hz), separately for stages 2, 3 and 4. The spectral powers in the different bins were normalized in each recording to the total power in stages 2+3+4 to enable comparisons between different recordings in the same persons and in recordings between persons. Both the first half and the whole AST were analysed by normalizing the data to the total power of each period, respectively. Results are given as percentage amounts of the given frequency bin of the total power of the studied period. The distribution of the absolute spectral power in different frequency bins in each recording was calculated as follows: (the power during the first half of the night −the power during the second half of the night)/the power during the first half of the night×100.

We also compared the power values obtained from the two derivations in different bands (for details see Lindberg et al., 2002). When compared to the C4–A1 derivation (=100%), the power in the O2–P4 derivation was 92.9+5.3% in δ, 118.2±8.5% in θ, 129.0±13.1% in α, 81.3±5.1% in σ and 93.0±5.1% in the β band.

2.3. Hormone assays

Testosterone was quantitated with a coated-tube radioimmunoassay (Spectria, Orion Diagnostica, Espoo, Finland). The detection limit of the assay is 0.1 nmol/l. The inter-assay coefficient of variation is 7% at 1.2 nmol/l and about 5% in the concentration range of 4–23 nmol/l.

2.4. Statistical analysis

In comparison of the 16 subjects to 11 controls a *t*-test was applied. In cases where the parameters had non-normal distribution, the Mann–Whitney rank sum test was used. The results for different subgroups of subjects with impulsive aggression and healthy controls were compared using either one-way ANOVA with post-hoc Student–Newman–Keul's Method (normally distributed values) or Kruskal–Wallis ANOVA on ranks with post-hoc Dunn's method (non-normally distributed values). One-way analysis of covariance with age as an independent factor was performed for the following parameters: S4%, SWS%, delta power in stages 4 and 3+4, theta power in stages 4 and 3+4, and serum testosterone level measurements. When covariance should effect, the final analysis was performed using age-adjusted values.

2.5. Ethics

Informed consent was obtained from the subjects and controls and the study was accepted by the ethical committee of Helsinki University Hospital. The principles of declaration of Helsinki were adequately followed.

3. Results

3.1. General

The percentage distribution of various sleep stages in first half of the night compared with the second half of the night in 16 subjects was equal to that of 11 controls (S2 in subjects vs. controls −30.8±11.31 vs. −14.2±11.58; t (25) = −0.994, P = NS; S3 in subjects vs. controls 26.4±13.44 vs. −48.9±91.58; Mann–Whitney T = 165.00, P = NS; S4 in subjects vs. controls 70.4±7.96 vs. 63.2±12.10; t (25) = 0.522, P = NS; SWS in subjects vs. controls 60.4±7.41 vs. 32.5±23.83; Mann–Whitney T = 142.00, P = NS).

The percentage distribution of the total power of each frequency bin in various stages among the subjects was equal to that of the controls when the first half of the night was compared with the second half of the night (the total power in stage 2 in subjects vs. controls −2.5±19. 20 vs. −14.7±28.22; Mann–Whitney T = 146.00, P = NS; the total power in stage 3 in subjects vs. controls 46.0±9.51 vs. 43.6±16.80; t (25) = 0.136, P = NS; the total power in stage 4 in subjects vs. controls 70.3±8.38 vs. 60.2±13.14; t (25) = 0.680, P = NS; the total power in SWS in subjects vs. controls 64.7+7.90 vs. 48.8+16.23; t (25) = 0.968, p = NS).

3.2. Antisocial vs. antisocial and borderline personality disorders

3.2.1. General

There were no significant differences between *ages* in the subject groups and healthy volunteers (antisocials 28.1±3.40 years vs. antisocials with BPD comorbidity

34.8±4.45 years vs. controls 32.5±3.44 years; one-way ANOVA: $F(2, 24) = 0.793$, P = NS). *BDI* expressed mild depressive symptoms in both subject groups without significant differences between groups, while the controls were almost symptom free (antisocials 10.6±2.18 vs. antisocials with BPD comorbidity 12.0±2.35 vs. controls 1.6±0.78; one-way ANOVA: $F(2, 24) = 10.789$, $P < 0.001$, post-hoc Student–Newman–Keul's antisocials vs. antisocials with BPD comorbidity $q = 0.731$, P = NS, antisocials vs. controls $q = 5.529$, $P < 0.001$, antisocials with BPD comorbidity vs. controls $q = 5.503$, $P = 0.002$).

3.2.2. Polysomnography

For details of the polysomnography, see Table 2. Antisocials with BPD comorbidity had significantly more awakenings during the night, and, as a result, the sleep efficiency (SE%) in this subgroup was significantly lower compared with both the APD-group and controls. Both subgroups had significantly higher absolute and percentage amounts of S4 sleep compared with controls but there were no statistically significant differences between subgroups. The absolute and percentage amounts of S3 sleep were lower in both subgroups compared with controls but again there was no statistically significant difference between subgroups.

3.2.3. Spectral power analysis

For details of the spectral power analysis, see Table 3. During the first half of the sleep period, there were no statistically significant differences between the subgroups in theta and delta power in stages 4 and 3+4. The APD-group had higher theta power in stage 4 and delta power in stages 4 and 3+4 compared with controls. The antisocials with BPD comorbidity had higher theta power in stage 4 compared with controls. During the whole sleep period, there were no statistically significant differences between the subgroups. The APD-group had higher theta and delta power in stages 4 and 3+4 compared with controls. The antisocials with BPD had higher theta and delta power in stage 4 compared with controls.

3.2.4. Serum testosterone levels

There were no significant differences between the serum testosterone levels of antisocials with and without BPD comorbidity (mean±SEM 20.4±1.74 nmol/l vs. 20.6±2.66 nmol/l; t (14) = 0.815, P = NS).

3.3. Severe conduct disorder vs. mild or moderate conduct disorder

3.3.1. General

There were no significant differences between *ages* in the subject groups and healthy volunteers (CD type severe 27.8±3.81 years vs. CD mild/moderate 33.5±3.92 years vs. controls 32.5±3.44 years; one-way ANOVA: $F(2, 24) = 0.631$, P = NS). *BDI* expressed mild depressive symptoms in both subject groups without significant differences between groups, while the controls were almost symptom free (CD type severe 11.6±2.60 vs. CD type mild/moderate 10.6±1.98 vs. controls 1.6±0.78; one ANOVA: $F(2, 24) = 10.674$, $P < 0.001$, post-hoc Student–Newman–Keul's CD type severe vs. CD type mild/moderate $q = 0.538$, P = NS, CD type severe vs. controls $q = 5.778$, $P = 0.001$, CD type mild/moderate vs. controls $q = 5.200$, $P = 0.001$).

3.3.2. Polysomnography

For details of the polysomnography, see Table 2. The subgroup with preceding type severe CD had significantly higher absolute and percentage amounts of S4 sleep and SWS but less S2 sleep compared with those subjects with only mild or moderate CD and controls. Both subgroups had lower absolute and percentage amounts of S3 sleep compared with controls, but there was no statistically significant difference between subgroups. The group with mild or moderate CD had significantly higher absolute and percentage amounts of S4 sleep compared with controls.

3.3.3. Spectral power analysis

For details of the spectral power analysis, see Table 3. During the first half of the sleep period, the subgroup with preceding type severe CD had higher theta and delta powers in stages 4 and 3+4 compared with males with mild or moderate CD and controls. The subgroup with preceding type mild or moderate CD had higher theta power in stage 4 compared to controls. During the whole sleep period, the subgroup with preceding type severe CD had higher theta and delta power in stages 4 and 3+4 compared with males with mild or moderate CD and controls. The subgroup with preceding type mild or moderate CD had higher theta and delta power in stage 4 compared with controls.

3.3.4. Serum testosterone levels

The group with preceding type severe CD had significantly higher serum testosterone levels than those with only mild or moderate CD (mean±SEM 24.1±1.83 nmol/l vs. 16.9±1.27 nmol/l; t (14) = 2.882, $P = 0.01$).

3.4. Intermittent explosive disorder vs. no intermittent explosive disorder

3.4.1. General

There were no significant differences between *ages* in the subject groups and healthy volunteers (IED+ 28.5±3.36 years vs. IED−37.2±4.41 years vs. controls 32.5±3.44 years; one-way ANOVA: $F(2, 24) = 1.106$, P = NS). *BDI* expressed mild depressive symptoms in both subject groups without significant differences

Table 2
The polysomnography parameters

	A $n=10$	B $n=6$	CO $n=11$	Statistics
Polysomnography				
TSL (min)	429.7±24.37	500.3±35.29	461.8±21.47	$F=1.606$, $P=$NS
AST (min)	396.9±18.66	455.3±28.09	442.6±22.61	$F=1.778$, $P=$NS
SE (%)	95.3±0.86	89.3±2.85	95.7±1.51	**$F=3.890$, $P=0.03$**
				A vs. B $q=3.400$, $P=0.02$
				B vs. CO $q=3.705$, $P=0.04$
sleep lat (min)	21.4±8.21	17.9±5.40	18.5±7.00	$H=0.877$, $P=$NS
awakenings (n)	10.7±1.65	24.7±4.53	11.7±1.26	**$F=9.815$, $P<0.001$**
				A vs. B $q=5.823$, $P=0.001$
				B vs. CO $q=5.489$, $P<0.001$
S1 (min)	23.6±5.79	26.9±2.12	23.3±5.38	$H=2.878$, $P=$NS
S1%	6.7±1.39	6.0±0.64	5.3±1.15	$H=1.610$, $P=$NS
S2 (min)	176.8±12.53	226.3±26.54	228.7±11.91	**$F=3.873$, $P=0.04$**
				A vs. CO $q=3.648$, $p=0.042$
				A vs. B $q=2.942$, $p=0.048$
S2%	44.3±1.83	49.0±3.44	51.7±1.33	**$F=4.194$, $P=0.03$**
				A vs. CO $q=4.077$, $P=0.02$
S3 (min)	38.5±3.66	36.6±5.78	63.5±5.74	**$F=8.916$, $P=0.001$**
				A vs. CO, $q=5.200$, $P=0.001$
				B vs. CO, $q=4.810$, $P=0.006$
S3%	9.9±1.17	7.7±0.89	14.6±1.40	**$F=7.144$, $P=0.004$**
				A vs. CO, $q=3.899$, $P=0.01$
				B vs. CO, $q=4.934$, $P=0.005$
S4 (min)	68.2±10.34	63.8±11.21	24.9±4.29	**$F=8.856$, $P=0.001$**
				A vs. CO, $q=5.528$, $P=0.002$
				B vs. CO, $q=4.279$, $P=0.006$
S4%	17.2±2.48	14.4±2.63	6.3±0.96	**$F=9.107$, $P=0.001$***
				A vs. CO, $q=3.960$, $P<0.001$
				B vs. CO, $q=2.997$, $P=0.005$
SWS (min)	104.8±9.93	100.3±11.99	88.4±7.56	$F=0.914$, $P=$NS
SWS%	27.1±2.38	22.1±2.32	21.0±1.31	$F=2.209$, $P=$NS*
REM lat (min)	111.9±8.25	83.2±10.84	105.3±16.75	$F=0.951$, $P=$NS
REM (min)	86.2±6.10	101.7±6.70	98.4±9.69	$F=0.934$, $P=$NS
REM%	21.7±1.18	22.7±2.05	21.8±1.32	$F=0.128$, $P=$NS
	CDs $n=8$	CDm $n=8$	CO $n=11$	
TSL (min)	425.4±25.90	478.0±28.33	461.8±21.47	$F=1.055$, $P=$NS
AST (min)	402.9±24.1	434.6±23.57	442.6±22.61	$F=0.773$, $P=$NS
SE (%)	94.8±1.38	91.3±2.28	95.7±1.51	$H=5.765$, $P=$NS
sleep lat (min)	26.1±9.67	30.9±16.57	18.5±7.00	$F=0.346$, $P=$NS
awakenings (n)	14.5±2.87	21.6±4.01	11.7±1.26	**$F=3.630$, $P=0.04$**
				CDm vs. CO $q=3.765$, $P=0.04$
S1 (min)	17.9±3.21	31.9±5.71	23.3±5.38	$F=1.720$, $P=$NS
S1 (%)	5.6±1.07	7.4±1.40	5.3±1.15	$F=0.843$, $P=$NS
S2 (min)	157.1±24.65	218.8±21.35	228.7±11.91	**$F=4.154$, $P=0.03$**
				CDs vs. CDm $q=3.115$, $P=0.04$
				CDs vs. CO $q=0.029$, $P=0.03$
S2 (%)	42.6±1.91	49.5±2.47	51.7±1.33	**$F=6.596$, $P=0.005$**
				CDs vs. CDm $q=3.528$, $P=0.02$
				CDs vs. CO $q=5.045$, $P=0.004$
S3 (min)	36.4±5.24	39.1±3.41	63.5±5.75	**$F=8.968$, $P=0.001$**
				CDs vs. CO $q=5.289$, $P=0.003$
				CD m vs. CO $q=4.776$, $P=0.003$
S3 (%)	9.2±1.55	9.0±0.74	14.6±1.40	**$F=6.238$, $P=0.007$**
				CDs vs. CO $q=4.138$, $P=0.008$
				CDm vs. CO $q=4.281$, $P=0.02$
S4 (min)	83.4±9.18	49.7±8.66	24.9±4.29	**$F=17.409$, $P<0.001$**
				CDs vs. CDm $q=4.472$, $P=0.004$
				CDs vs. CO $q=8.345$, $P<0.001$
				CDm vs. CO $q=3.533$, $P=0.02$

(*continued on next page*)

Table 2 (*continued*)

	CDs $n=8$	CDm $n=8$	CO $n=11$	Statistics
S4 (%)	20.6±1.91	11.6±2.19	6.3±0.96	**$F=19.167$, $P<0.001$***
				CDs vs. CDm $q=3.394$, $P=0.002$
				CDs vs. CO $q=6.191$, $P<0.001$
				CDm vs. CO $q=2.567$, $P=0.02$
SWS (min)	119.9±9.88	88.6±7.75	88.46±7.56	**$F=4.397$, $P=0.02$**
				CDs vs. CDm $q=3.524$, $P=0.02$
				CDs vs. CO $q=3.813$, $P=0.03$
SWS (%)	29.8±1.93	20.6±2.00	21.0±1.31	**$F=8.250$, $P=0.002$***
				CDs vs. CDm $q=3.455$, $P=0.002$
				CDs vs. CO $q=3.696$, $P=0.001$
REM lat (min)	125.7±18.77	72.4±11.43	105.3±16.75	$F=2.404$, $P=$NS
REM (min)	88.3±7.13	95.6±6.74	98.4±9.69	$F=0.367$, $P=$NS
REM (%)	21.9±1.28	22.3±1.72	21.8±1.32	$F=0.034$, $P=$NS
	IED+ $n=10$	IED- $n=5$	CO $n=11$	
TSL (min)	455.3±21.78	458.5±47.77	461.8±21.47	$F=0.018$, $P=$NS
AST (min)	428.7±21.41	409.2±33.55	442.6±22.61	$F=0.376$, $P=$NS
SE (%)	94.0±1.01	90.5±3.87	95.7±1.51	$H=4.084$, $P=$NS
sleep lat (min)	33.1±13.59	11.0±5.32	18.5±7.00	$H=3.109$, $P=$NS
awakenings (n)	17.8±1.84	21.0±7.31	11.7±1.26	$H=4.752$, $P=$NS
S1 (min)	21.1±2.49	36.2±8.59	23.3±5.38	$F=1.779$, $P=$NS
S1 (%)	5.8±0.74	8.8±2.03	5.3±1.15	$H=4.479$, $P=$NS
S2 (min)	195.9±18.47	172.5±45.94	228.7±11.91	$F=1.595$, $P=$NS
S2 (%)	45.0±2.50	47.2±2.66	51.7±1.33	$F=3.159$, $P=$NS
S3 (min)	36.3±3.95	42.0±5.74	63.5±5.75	**$F=8.445$, $P=0.002$**
				IED+ vs. CO $q=5.605$, $P=0.002$
				IED- vs. CO $q=0.019$, $P=0.02$
S3 (%)	8.3±0.75	10.7±2.19	14.6±1.40	**$F=6.658$, $P=0.005$**
				IED+ vs. CO $q=5.122$, $P=0.004$
S4 (min)	79.4±8.50	38.8±8.26	24.9±4.29	**$F=18.784$, $P<0.001$**
				IED+ vs. IED- $q=5.057$, $P=0.002$
				IED+ vs. CO $q=8.509$, $P<0.001$
S4 (%)	18.9±2.07	9.6±2.22	6.3±0.96	**$F=15.131$, $P<0.001$***
				IED+ vs. IED- $q=2.822$, $P=0.01$
				IED+ vs. CO $q=5.484$, $P<0.001$
SWS (min)	115.7±8.95	80.6±8.37	88.4±7.56	**$F=4.345$, $P=0.03$**
				IED+ vs. IED- $q=3.555$, $P=0.05$
				IED+ vs. CO $q=3.464$, $P=0.02$
SWS (%)	27.2±2.02	20.4±3.36	21.0±1.31	$F=2.653$, $P=$NS*
REM lat (min)	86.6±9.53	126.8±35.08	105.3±16.75	$F=1.006$, $P=$NS
REM (min)	92.0±4.52	96.6±12.46	98.4±9.69	$F=0.165$, $P=$NS
REM (%)	21.7±1.54	23.4±2.39	21.8±1.32	$F=0.317$, $P=$NS

A, antisocial personality disorder; B, antisocial and borderline personality disorders; CDs, conduct disorder type severe; CDm, conduct disorder type mild or moderate; IED+, intermittent explosive disorder; IED-, no intermittent explosive disorder; CO, controls; TSL, total sleep length; AST, actual sleep time; SE, sleep efficiency; S1–S4, sleep stages 1–4; SWS, slow wave sleep; REM, rapid eye movement sleep; lat, latency; NS, change is not statistically significant. Comparisons made using one-way ANOVA with post-hoc Student–Newman–Keul's method (normally distributed values) and Kruskal–Wallis ANOVA on ranks with post-hoc Dunn's method (non-normally distributed values). Significant differences between groups are indicated with bold typing.

* one-way analysis of covariance with age as an independent factor. All values expressed as mean±SEM.

between groups, while the controls were almost symptom free (IED+ 12.6±2.00 vs. IED-10.0±2.55 vs. controls 1.6±0.78; one-way ANOVA: $F(2, 24)=13.824$, $P<0.001$, post-hoc Student–Newman–Keul's IED+ vs. IED-$q=1.364$, $P=$NS, IED+ vs. controls $q=7.211$, $P<0.001$, IED-vs. controls $q=4.456$, $P=0.005$).

3.4.2. Polysomnography

For details of the polysomnography, see Table 2. The only statistically significant differences in sleep parameters between males with IED and males without this diagnosis were found in S4 sleep and in SWS, with higher absolute and percentage amounts of S4 sleep and

Table 3
The percentual spectral power in relation to the total power in stages 2 + 3 + 4

	A $n=10$	B $n=6$	CO $n=11$	Statistics
first half of AST				
theta power (%)				
stage 4	13.6±1.17	10.8±2.55	5.0±0.93	***F* = 11.546, *P* < 0.001**
				A vs. CO $q=6.679$, $P<0.001$
				B vs. CO $q=3.883$, $P=0.01$
stage 3 + 4	17.4±0.81	14.8±2.47	13.9±0.88	$F=2.442$, $P=$ NS
delta power (%)				
stage 4	20.9±5.03	12.4±3.25	4.9±1.17	***F* = 5.907, *P* = 0.008**
				A vs. CO $q=4.861$, $P=0.006$
stage 3 + 4	24.2±5.13	15.6±3.21	10.9±1.52	***F* = 3.850, *P* = 0.04**
				A vs. CO $q=3.900$, $P=0.03$
total AST				
theta power (%)				
stage 4	11.7±1.16	7.7±1.66	3.8±0.63	***F* = 15.267, *P* < 0.001***
				A vs. CO $q=5.490$, $P<0.001$
				B vs. CO $q=2.696$, $P=0.01$
stage 3 + 4	15.4±0.88	11.3±1.58	11.3±0.95	***F* = 4.194, *P* = 0.03***
				A vs. CO $q=2.739$, $P=0.01$
delta power (%)				
stage 4	17.7±4.73	9.0±2.41	3.5±0.80	***F* = 8.938, *P* = 0.001***
				A vs. CO $q=4.082$, $P<0.001$
				B vs. CO $q=2.594$, $P=0.02$
stage 3 + 4	21.2±4.88	12.1±2.51	8.3±1.11	***F* = 5.040, *P* = 0.02***
				A vs. CO $q=3.154$, $P=0.004$
	CDs $n=8$	CDm $n=8$	CO $n=11$	
first half of AST				
theta power (%)				
stage 4	15.5±1.14	9.6±1.56	5.0±0.93	***F* = 20.152, *P* < 0.001**
				CDs vs. CDm $q=4.706$, $P=0.003$
				CDs vs. CO $q=8.977$, $P<0.001$
				CDm vs. CO $q=3.913$, $P=0.01$
stage 3 + 4	18.8±0.97	14.0±1.48	13.9±0.88	***F* = 6.202, *P* = 0.007**
				CDs vs. CDm $q=4.142$, $P=0.007$
				CDs vs. CO $q=4.560$, $P=0.01$
delta power (%)				
stage 4	25.9±5.44	9.6±1.60	4.9±1.17	***F* = 13.140, *P* < 0.001**
				CDs vs. CDm $q=5.112$, $P=0.002$
				CDs vs. CO $q=7.084$, $P<0.001$
stage 3 + 4	28.9±5.67	13.0±1.55	10.9±1.52	***F* = 9.244, *P* = 0.001**
				CDs vs. CDm $q=4.728$, $P=0.003$
				CDs vs. CO $q=5.767$, $P=0.001$
total AST				
theta power (%)				
stage 4	12.9±1.26	7.5±0.99	3.8±0.63	***F* = 24.204, *P* < 0.001***
				CDs vs. CDm $q=3.616$, $P=0.002$
				CDs vs. CO $q=6.951$, $P<0.001$
				CDm vs. CO $q=3.096$, $P=0.005$
stage 3 + 4	16.0±1.0	11.8±1.17	11.3±0.95	***F* = 4.734, *P* = 0.02***
				CDs vs. CDm $q=2.323$, $P=0.03$
				CDs vs. CO $q=2.968$, $P=0.007$
delta power (%)				
stage 4	21.5±5.36	7.4±1.05	3.5±0.80	***F* = 11.230, *P* < 0.001***
				CDs vs. CDm $q=3.986$, $P=0.002$
				CDs vs. CO $q=4.662$, $P<0.001$
				CDm vs. CO $q=2.674$, $P=0.01$

(*continued on next page*)

Table 3 (*continued*)

	CDs $n=8$	CDm $n=8$	CO $n=11$	Statistics
stage 3+4	24.7±5.62	10.8±1.28	8.3±1.11	**$F=7.762$, $P=0.003$*** CDs vs. CDm $q=2.306$, $P=0.03$ CDs vs. CO $q=3.938$, $P<0.001$
	IED+ $n=10$	IED- $n=5$	CO $n=11$	
first half of AST theta power (%)				
stage 4	14.4±1.46	9.0±1.66	5.0±0.93	**$F=15.745$, $P<0.001$** IED+ vs. IED- $q=3.688$, $P=0.02$ IED+ vs. CO $q=7.925$, $P<0.001$
stage 3+4	18.1±1.14	13.4±1.90	13.9±0.88	**$F=4.981$, $P=0.02$** IED+ vs. IED- $q=3.499$, $P=0.05$ IED+ vs. CO $q=3.980$, $P=0.01$
delta power (%)				
stage 4	18.1±3.39	9.6±2.92	4.9±1.17	**$F=7.879$, $P=0.002$** IED+ vs. IED- $q=2.857$, P 0.05 IED+ vs. CO $q=5.584$, $P=0.002$
stage 3+4	21.0±3.16	13.1±3.28	10.9±1.52	**$F=4.731$, $P=0.02$** IED+ vs. CO $q=4.246$, $P=0.02$
total AST theta power (%)				
stage 4	11.3±1.42	7.9±1.42	3.8±0.63	**$F=12.700$, $P<0.001$*** IED+ vs. IED- $q=2.462$, $P=0.04$ IED+ vs. CO $q=4.944$, $P<0.001$ IED- vs. CO $q=2.628$, $P=0.02$
stage 3+4	14.8±1.16	12.5±1.80	11.3±0.95	$F=2.107$, $P=$NS*
delta power (%)				
stage 4	14.3±3.49	8.2±2.23	3.5±0.80	**$F=7.890$, $P=0.003$*** IED+ vs. IED- $q=3.012$, $P=0.02$ IED+ vs. CO $q=3.803$, $P=0.001$ IED- vs. CO $q=2.393$, $P=0.03$
stage 3+4	17.2±3.48	12.0±2.95	8.3±1.11	**$F=4.008$, $P=0.03$*** IED+ vs. CO $q=2.751$, $P=0.01$

A, antisocial personality disorder; B, antisocial and borderline personality disorders; CDs, conduct disorder type severe; CDm, conduct disorder type mild or moderate; IED+, intermittent explosive disorder; IED-, no intermittent explosive disorder; CO, controls; AST, actual sleep time; NS, change is not statistically significant. Comparisons made using one-way ANOVA with post-hoc Student–Newman–Keul's method (all values normally distributed). Significant differences between groups are indicated with bold typing.

* One-way analysis of covariance with age as an independent factor. All values expressed as mean±SEM.

absolute amount of SWS in males with IED. The absolute and percentage amounts of S4 sleep and absolute amount of SWS were higher in males with IED compared with controls, while absolute and percentage amounts of S3 sleep were lower. The absolute amount of S3 sleep was significantly decreased in males without IED compared with controls.

3.4.3. Spectral power analysis

For details of the spectral power analysis, see Table 3. During the first half of the sleep period, the subgroup with IED had higher theta power in stages 4 and 3+4 and delta power in stage 4 compared to males without this diagnosis. The subgroup with IED had higher theta and delta power in stages 3 and 3+4 compared with controls. There were no statistically significant differences between subjects without IED and controls. During the whole sleep period, the subgroup with IED had higher theta and delta power in stage 4 compared with males without the diagnosis. The subgroup with IED had higher theta power in stage 4 and delta power in stage 4 and 3+4 compared with controls. The subgroup without IED had higher theta and delta power in stage 4 compared with controls.

3.4.4. Serum testosterone levels

There was a tendency towards higher serum testosterone levels in males with IED compared with subjects without this diagnosis, although the difference was not statistically significant (mean±SEM 21.8±1.65 nmol/l vs.16.4±2.10; t (13)=1.567, $P=0.08$).

4. Discussion

The population of this study with crimes against human life is highly unusual and selected even for a criminal population. The subjects were evaluated as highly aggressive with major psychological and social problems (lack of empathy, ability to plan, vocational training, occupation and housing accommodation, inability to take care of family members), but however, responsible of the violent acts they were being charged with. We intended to select a population with no axis I psychiatric disorders which, however, proved to be impossible: APD is frequently combined with substance-related disorders (Robins, 1998), which was also the case in this study with almost all males having Cloninger type II alcoholism. However, as the duration of alcohol abstinence before the sleep examination was several months we regard it improbable that the sleep would have been affected by alcohol withdrawal syndrome. Because of the rarity of this kind of highly aggressive group of antisocial persons and the strict exclusion criteria that we adopted the number of the subjects is small and the findings of the study must be regarded as indicative.

To our knowledge, this is the first study to compare sleep in APD with and without BPD comorbidity. The most striking finding was the disruption in the continuity of sleep in persons with both personality disorders. In fact, "pure" antisocials did not differ from healthy controls when it came to the number of awakenings and SE%. This finding resembles the results reported in polysomnographic studies of borderline persons with no APD comorbidity (Battaglia et al., 1993; De La Fuente et al., 2000). Both increased number of awakenings and decreased SE% are also typical findings in acute depression (Kupfer et al., 1980). However, the degree of depressive symptoms in our subjects was only mild and did not differ significantly between the two groups. In APD with and without BPD comorbidity, the amount of S4 sleep was equally high and significantly higher than in controls. So, in spite of many overlapping clinical features, sleep architecture in these two personality disorders appears to differ and, in cases with comorbidity, both disorders have their own characteristics that influence sleep. The result is in agreement with the finding that APD differs from that with BPD comorbidity both in research settings and clinical outcome (Coid, 1993; Soloff et al., 1994; Virkkunen et al., 1996; Herpertz et al., 2001).

The total number of CD symptoms was reported to be the most important predictor of future APD (Robins, 1991). In the study by Stattin and Magnusson (1989), high ratings for aggressiveness were characteristic of boys who subsequently committed violent crimes and damage to public property. In our study of habitually violent offenders, half the subjects were estimated to have severe CD and half were regarded as having either mild or moderate CD in their medical history. The subjects with high number of CD symptoms in childhood anamnesis had significantly higher amount of delta sleep compared with males with only mild or moderate disorder. This raises the interesting question of whether this exceptional deep-sleep pattern had already developed in childhood or adolescence in these males. In a sleep study by Coble et al. (1984), a higher number of delta waves were found in boys with a primary diagnosis of CD compared with age-matched healthy controls, suggesting that this deep-sleep pattern may indeed already develop in childhood. The subjects in the Coble study were 17 pre-adolescent boys and in fact 13 of them represented the undersocialized aggressive subtype according to DSM-III, which was regarded as the most serious form of CD. To qualify as a case of the aggressive form, the conduct had to include robbery or violence against persons or property and, for a case to qualify as undersocialized, there could be no more than one of five indicators of being "socialized": enduring friendships, altruistic behaviour, feeling guilt or remorse, refraining from blaming others, and showing concern for others (American Psychiatric Association, 1980). It is possible to speculate that the boys in the study by Coble et al. would be the most likely to become antisocial in adulthood. Raine et al. (1990) reported a retrospective waking EEG study of 101 males which showed that adult criminals at the age of 24 had significantly more slow-frequency (δ and θ) electroencephalographic activity than non-criminals when measured at the age of 15 years. The researchers speculated that, in addition to social and psychological variables, measures of both autonomic nervous system and central nervous system underarousal may facilitate the early prediction of subsequent antisocial behaviour and even elucidate the etiological basis of criminality. Although it is problematic to extrapolate findings from waking EEG to sleep EEG, daytime EEG abnormalities have been found to reflect in sleep as well (Michael, 2001).

The notion that explosive violence may be linked to a discrete diagnosable condition such as IED is still controversial. S4 sleep was significantly higher in males with IED compared with subjects without this diagnosis. In fact, non-IED subjects did not differ in this respect from healthy controls. The relationship between high amount of S4 sleep and repetitive impulsive violence underlines the dimensional aspect of human aggressive behaviour. The result is also in agreement with previous studies suggesting that there might be different subpopulations of individuals with APD varying in impulsiveness (Linnoila et al., 1983; Coccaro et al., 1989; Virkkunen et al., 1996; Barratt et al., 1997; Coccaro et al., 1998; Coccaro 2000).

In this study, there were no differences in single-sample serum testosterone levels between antisocials with and without BPD comorbidity. On the other hand, there was a significant difference in serum testosterone

levels between antisocials with preceding type severe CD and those with only mild or moderate CD. This finding is in accordance with the study by Brooks & Reddon (1996), which reported higher single morning serum testosterone levels in 15–17 year old violent offenders with CD compared with boys committing non-violent or sexual offences. The antisocials with IED displayed a tendency towards higher serum testosterone levels than subjects without the diagnosis. If IED is regarded as a categorical expression of recurrent problematic impulsive and aggressive behaviour as suggested by Coccaro (2000), it is possible to speculate that, in this subgroup of antisocials, the criminal recidivism would be emphasized. In the study by Räsänen et al. (1999), recidivists with personality disorder had higher testosterone levels than non-recidivists with personality disorder. The limitation of the present work is the absence of serum testosterone measurements in control subjects. We limited the comparisons to the different subgroups of APD. However, in the study by Räsänen et al. (1999), the serum testosterone levels of the healthy males of approximately the same age as our controls (36.4 years, S.D. 8.0) were lower (16.8 nmol/l, S.D. 4.7) than the levels in antisocials with extreme aggression in the present study. In both patient groups with higher serum testosterone levels (preceding type severe CD and IED), also the percentage amount of S4 sleep and the relative theta and delta power in this sleep stage were significantly increased. The role of diurnal testosterone secretion in regulating normal human sleep is still fairly unclear. Serum testosterone levels have been described as being lower when young healthy adult men were awake than during sleep (11 p.m.–7 a.m.). The levels began to rise when the subjects fell asleep, and reached their peak value at about the time of the first REM cycle, remaining at the same levels until awakening (Luboshitzky et al., 1999). In the sleep EEG study by Leibenluft et al. (1997), leuprolide acetate was used to produce pharmacologically induced short-term hypogonadism in males between the ages of 18–48 years. Interestingly, this procedure only caused significant reductions in the amount of S4 sleep compared with measures taken during testosterone replacement. This connection between S4 sleep and testosterone, despite being associated with the testosterone-replaced state, offers the opportunity to speculate about whether, in violent offenders with APD, the increased amount of S4 sleep is perhaps at least partly mediated via elevated testosterone levels.

The study gives further support to the growing evidence of brain dysfunction in severe aggressive behaviour and strengthens the view that there are different subpopulations of individuals with antisocial personality varying in impulsiveness. The differences in impulsiveness are reflected to sleep architecture as well.

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