Epilepsia, **44**(7):944–949, 2003
Blackwell Publishing, Inc.

Absence Epilepsy with Onset before Age Three Years: A Heterogeneous and Often Severe Condition

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Summary: *Purpose:* The classification of epilepsies and epileptic syndromes recognizes three syndromes with typical absences [TA, i.e., childhood and juvenile absence epilepsies (CAE and JAE), and epilepsy with myoclonic absences (EMA), none of which is characterized by onset in early childhood]. Although several other forms of absence epilepsies have been described recently, none concerns infants and very young children, and little is known about the nosology and prognosis of early-onset absences.

Methods: We retrospectively selected all cases with onset of absences as the only or major seizure type before age 3 years and ≥2 years of follow-up among cases newly referred between 1986 and 2002. Neurospychological assessments (generally IQ measure), behavior patterns, and schooling situations were reviewed for each child.

Results: We found 10 patients (7 F, 3 M). No child had sensory or motor deficits: neuroimaging was performed in nine and was normal in eight, with aspecfic findings in one. Only two could be characterized as CAE and EMA, respectively, both with seizure control and a good cognitive outcome. Among the remaining eight cases, four had a fairly homogeneous presentation with predominantly brief absences and clearly asymmetric interictal EEGs. All eight had neuropsychological and/or behavioral difficulties. Three had full seizure control, and five, persisting absences, with a follow-up ranging beetween 2 years 8 months to 9 years 4 months; only one child was older than 12 years.

Conclusions: Great heterogeneity exists among absence epilepsies of early onset, which are rare conditions. Only a few patients can be categorized into well-known syndromes. The overall prognosis is poor. Early onset of absences is uncommon, and multicenter studies should help clarify the nosology and prognosis. **Key Words:** Epilepsy—Typical absences—Absence epilepsy—Early onset—Prognosis.

Typical absences (TAs) are defined by a more or less complete loss of contact that may be isolated (simple TA) or associated with motor phenomena (complex TA), with a concomitant bilateral regular and symmetrical spike-and-wave (SW) discharge ~3 Hz on the EEG (1), and TAs are found mostly in idiopathic generalized epilepsies (IGEs) (2). Conversely, atypical absences are found in other settings (e.g., the Lennox–Gastaut syndrome (3). Several well-described epileptic syndromes include TA (4): childhood absence epilepsy (CAE), juvenile absence epilepsy (JAE), juvenile myoclonic epilepsy (JME), and epilepsy with grand mal on awakening (EGMA). Panayiotopoulos (5) described less common syndromes with TA, but the existence of these syndromic entities has not been validated. In CAE, TAs occur typically between 5 and 9 years, and the youngest age at onset has been set at 3 years (6). Given the heterogeneity of epilepsies with TA, some authors have tried to establish a clinical and prognostic classification (5,7), but age at onset has not been considered a discriminating criterion. In their classic work of epilepsies with TA in childhood, Doose et al. (8) found only seven cases (among a total of 149) with onset before age 3, and had classified them together with myoclonic–astatic epilepsy, the soon-to-be-described Lennox–Gastaut syndrome, and a "grand mal" epilepsy of childhood. Early-onset absences are uncommon and still difficult to classify into one of the established syndromes. To try to clarify the nosology and prognosis of such cases, we decided to study retrospectively the clinical and EEG characteristics and the course of absence epilepsies with onset before age 3, which is the lower limit of classic CAE.

PATIENTS AND METHODS

We included patients first referred to the Centre Saint Paul between 1986 and 2002, on the following criteria: onset of absences before age 3, with clinical and EEG documentation of loss of contact associated with 3-Hz SW bilateral and symmetrical discharges; absences as the

Accepted March 1, 2003.
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This work was published in poster form at the 5th European Epilepsy Congress, Madrid, October 6–10, 2002.

only or clearly predominant seizure type; a follow-up of ≥2 years. In the summer of 2002, we reviewed all available clinical and EEG records, as well as information from neuropsychological assessments, neuroimaging, and school reports, and contacted whenever necessary the patients' families and doctors.

Neuropsychological assessments were carried out by a neuropsychologist with at least one IQ measurement. Three IQ scales were used: the Wechsler Intelligence Scale for Children–III (WISC III) (9), the Wechsler Preschool and Primary Scale for Intelligence–Revised (WPPSI-R) (10), and the Brunet–Lezine (11). Some patients had two assessments at different periods: in this case, the last assessment only was taken into consideration. Neuroimaging included either a computed tomography (CT) scan or a magnetic resonance imaging (MRI) scan. Video-EEGs were performed in all while awake and asleep.

RESULTS

Ten cases fulfilling our criteria were found. Six of these patients had been included in a prospective database on all newly referred patients of all ages maintained between 1986 and 1997, the four remaining cases were first referred later. Among 2,811 patients with confirmed epilepsy in our database, 639 (22.7%) had seizure onset before age 3. The main clinical characteristics have been summarized on Table 1. They were seven girls and three boys, with a mean age at onset of absences of 27.5 months (range, 15–36). The treatment delay (time elapsed between the first clinical manifestations and the onset of treatment) was short, between 4 and 8 weeks in all but three: 5 months in case 8, 12 months in case 9, and 48 months in case 3.

A first- or second-degree family history of epilepsy was found in only two cases: IGE with generalized tonic–clonic seizures (GTCSs) during childhood with full remission without treatment in adulthood in the father of case 1; epilepsy with GTCSs in a maternal great-aunt in case 8 (no further documentation). A perinatal history was present in four: intrauterine growth delay (case 7); twin pregnancy with uncomplicated premature birth at 36 weeks (case 8); twin pregnancy and cesarean for breech position (case 9); and premature birth at 32 weeks and emergency cesarean for toxemia (case 3). However, none had had signs of perinatal distress. No child had sensory or motor deficits; the mean age for walking was 14.9 months, and only one child was delayed in this respect (age 20 months, case 7). Delayed development of language was noted in three cases: it was first noted at first referral for absences in two (cases 4 and 10), but had been present before, and in case 9, a simple speech delay was noted during follow-up. Neuroimaging was performed in nine, and was consistently normal except for the detection of periventricular white matter increased T_2-weighted signal intensity on MRI scans at age 6 years (case 9), which were considered nonspecific.

Absences were classified as simple in five cases and complex in five because of associated motor components (eyelid myoclonia, myoclonic jerks, or automatisms). Case 3 had absences with bilateral and rhythmic myoclonic jerks and fulfills the criteria of epilepsy with myoclonic absences (EMA). None had atonic components and/or falls. In two cases, absences were first

TABLE 1. *Clinical characteristics and course of epilepsy*

Case	Sex	Age at onset (mo)	Family history	Neuro imaging	Absences at onset	Follow-up (duration)	Absences at follow-up	Course of epilepsy	Treatment at last visit
1	F	30	+	MRI N	Simple	3 yr 4 mo	Complex with myoclonic jerks	Uncontrolled Uncontrolled	VPA + ESM + TPM
2	F	30	–	CT N	Simple (+EE)	2 yr 8 mo	Complex with myoclonic jerks	Uncontrolled daily absences	VPA + CLB
3	F	24	–	CT N	Complex with myoclonic jerks (+EE)	4 yr 7 mo	No change No change	Controlled at 6 yr 3 mo	VPA
4	F	30	–	MRI N	Simple (+EE)	2 yr 11 mo	No change No change	Uncontrolled daily absences	LTG + ESM + CLB
5	M	36	–	MRI N	Complex with eyelid myoclonia (+EE)	9 yr 4 mo	Complex with automatisms	Uncontrolled daily absences	LTG + ESM + LEV
6	F	33	–	CT N	Simple (+EE)	4 yr 3 mo	No change	Uncontrolled daily absences	LTG + ESM + VPA
7	F	15	–	CT N	Complex with eyelid myoclonia (+EE)	2 yr 2 mo	No change	Controlled at 3 yr 2 mo	VPA
8	F	23	+	ND	Simple (+EE)	5 yr 7 mo	No change	Controlled at 6 yr	VPA + ESM
9	M	30	–	MRI A	Simple	7 yr 4 mo	No change	Controlled at 7yr	LTG
10	M	24	–	CT N	Complex with brief non-rhythmic myoclonia	17 yr	Simple (+EE)	Controlled at 8 yr 6 mo	None

F, female; M, male; N, normal; ND, not done; A, abnormal; EE, elevation of eyeballs; VPA, valproate; LTG, lamotrigine; ESM, ethosuximide; LEV, levetiracetam; CLB, clobazam; TPM, topiramate (see text for more details about history or development).

TABLE 2. *Electroencephalographic features*

Case	Background activity	Sleep organization	Absences duration	Onset of discharge	End of discharge	Paroxysms during sleep	HV	Photic stimulation
1	N	Decreased sleep transients	4–8 s	Abrupt; sometimes asymmetric	Sometimes progressive and asymmetric	↗	+	—
2	N	N	15–18 s	Abrupt	Abrupt	↗	+	—
3	N	N	7–8 s	Abrupt	Sometimes progressive	↗	+	—
4	N	N	≤50 s	Abrupt	Progressive	↗	−	Driving at low frequencies
5	N	N	3–4 s	Abrupt	Abrupt	↗	+	Triggers Absences
6	N	N	3–4 s	Abrupt	Abrupt	↗	+	—
7	N	N	3–4 s	Abrupt	Abrupt	↗	−	—
8	N	N	10–12 s	Abrupt	Abrupt	↗	+	Driving at low frequencies
9	N	N	3–4 s	Abrupt	Abrupt	↗	−	—
10	Posterior and/or diffuse slow waves	N	6–10 s	Abrupt	Abrupt	↗	+	—

N, normal; ↗, increase; HV, hyperventilation; +, activating effect on absences.

simple, but myoclonic jerks appeared at age 5 years (cases 1 and 2). Another patient (case 10) had initial absences with nonrhythmic, brief, diffuse myoclonic jerks, but 5 years later, only simple absences. An isolated febrile convulsion was noted before the first absences in cases 6 and 8. In case 5, the patient also had four generalized tonic seizures during follow-up, but no GTCSs or partial seizures.

EEG features are summarized in Table 2. The background activity was normal at the beginning and during follow-up in all except case 10, and sleep organization was normal in nine. Interictal changes were generalized and symmetric, but sometimes asymmetric with right or left predominance in four cases (cases 5, 6, 9, 10). These asymmetric activities were consistently found on several distinct EEG recordings (Fig. 1). In two other patients, the EEG demonstrated focal abnormalities. In case 2, a persistent left temporal spike focus seemed independent of the ictal and interictal activities. In case 1, a right frontal spike focus appeared during the course of epilepsy, and in some EEG recordings, the TA appears in continuity with the frontal focus (Fig. 2). The duration of TA was between 4 and 20 in five, <4 s in four, and >20 s in one. The ictal EEG showed generalized, symmetric spike- or polyspike- (with a maximum of three spikes) and slow wave complexes at 2.5–3.5 Hz. Hyperventilation octivated TA in seven. No activation of TA occured during photic stimulation, except in case 5, in whom TAs were triggered by intermittent light stimulation or by watching cartoons on television. Spontaneous absences and absences triggered by hyperventilation also were noted in this patient.

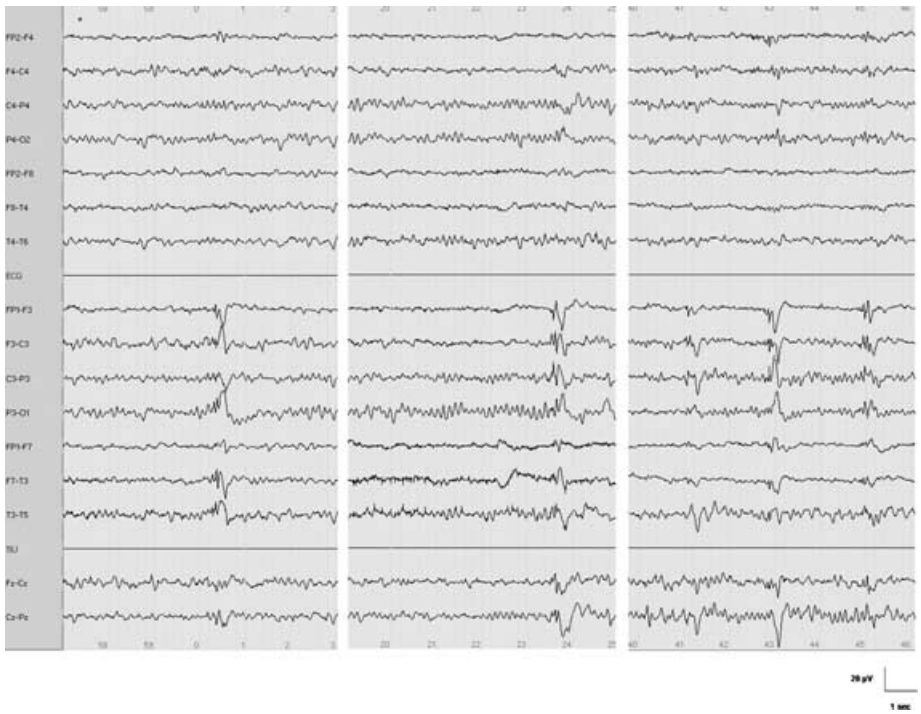

FIG. 1. Case 9. Persistent asymmetric interictal paroxysms with left predominance on three separate EEG recordings at age 17 yr 3 mo **(Left)**, at age 18 yr 2 mo **(Central)**, and at age 19 yr **(Right)**.

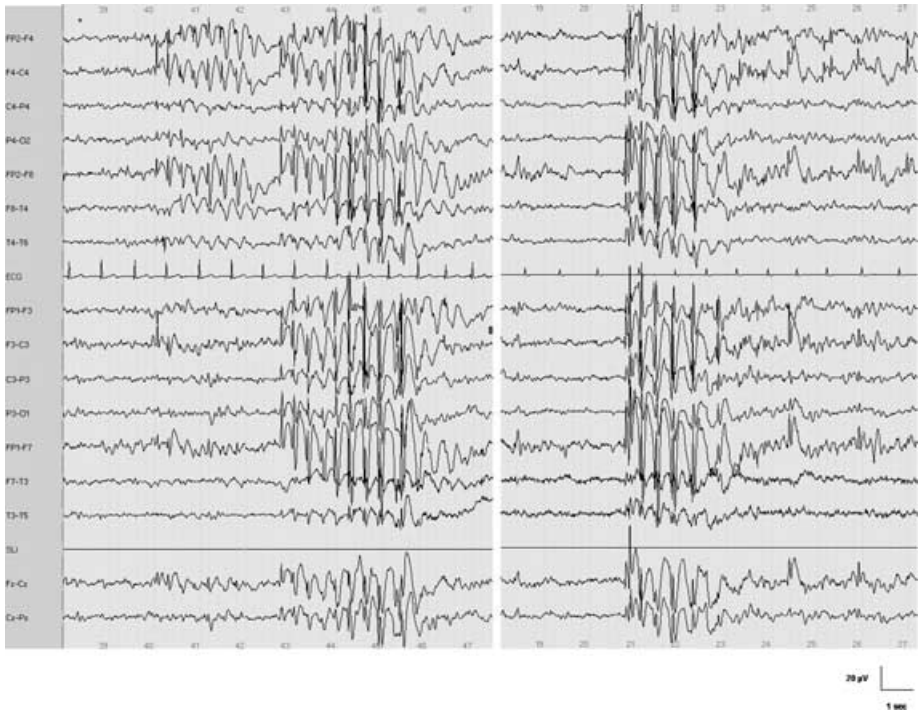

FIG. 2. Case 1. **Left:** absence with asymmetric onset with right predominance recorded at age 6 yr 5 mo. **Right:** Absence with symmetric onset and asymmetric interictal spike-and-wave at age 6 yr 2 mo.

The mean follow-up was 71.6 months (range, 26–204). Control of absences was obtained in five cases. Only one child (case 10) was seizure free without therapy since age 12 years. The others were seizure free with valproate (VPA; cases 3 and 7) or lamotrigine (LTG; case 9). In case 8, epilepsy is controlled with VPA and ethosuximide (ESM). Tapering of antiepileptic drugs (AETs) at age 7 resulted in relapse. Five children had persisting frequent absences with polytherapy (details on Table 1).

The cognitive outcome is summarized in Table 3. Only two patients had normal academic achievement (cases 3 and 8). Cases 5 and 10 required special schooling. In case 10, the initial language delay persisted, and a significant dissociation was seen between low verbal skills and somewhat better performance skills. Because of abnormal behavior, psychiatric follow-up was necessary, and pervasive developmental disorder was the psychiatric diagnosis in this case. Case 5 received special education because of severe behavioral disorder (oppositional defiant disorder) despite a normal IQ. The others were maintained in the normal schooling system but experienced major difficulties. Neuropsychological assessment showed low IQ in cases 4 and 6: results were homogeneous for verbal and performance skills. Visual-spatial and visual-construction deficits were noted in cases 1 and 2, who retained better verbal abilities. In case 9, school difficulties were secondary to attention and concentration deficit, whereas speech delay improved during childhood. In case 7, a motor and a cognitive delay was noted, but the patient was lost to follow-up at 3 years 5 months, and IQ was not evaluated.

TABLE 3. *Neuropsychological outcome*

Case	Academic achievement	Behavior	Neuropsychological Assessment
1	Normal school with difficulties	Normal	VIQ 94, PIQ 74 (WPPSI-R) at 5 yr 10 mo
2	Normal school with difficulties	Hyperactivity	DQ 100 (Brunet–Lezine) at 5 yr 6 mo; Visual-spatial deficit
3	Normal	Normal	ND
4	Normal school with difficulties	Hyperactivity	VIQ 69, PIQ 68 (WPPSI-R) at 5 yr 4 mo; language delay
5	Special school	Oppositional defiant disorder	VIQ 87, PIQ 96 (WISC III) at 9 yr 11 mo
6	Normal school with difficulties	Normal	VIQ 55, PIQ 56 (WISC III) at 7 yr 4 mo
7	Normal school with difficulties	Normal	Cognitive delay; IQ ND
8	Normal	Normal	VIQ 113, PIQ 107 (WISC III) at 8 yr 6 mo
9	Normal school with difficulties	Attention deficit	DQ 80 (Brunet–Lezine) at 5 yr speech delay improved
10	Special school	Psychiatric follow-up; pervasive developmental disorder	VIQ 55, PIQ 83 (WISC III); language delay persistant 12 yr 6 mo

WPPSI-R and WISC III, Wechsler scales; DQ, developmental quotient.

DISCUSSION

Early onset of absences is not a common situation: in our experience, such patients represent ~1% of epilepsies with onset before age 3. In our study, only two cases fulfill clinical and EEG criteria of well-recognized epileptic syndromes: CAE in case 8, who also had a family history of idiopathic epilepsy, and EMA in case 3 (4,12). Both patients enjoyed a good outcome. Thus there may be an early onset in CAE or EMA, but such patients are only a minority among those with early-onset absences. In CAE, onset before age 24 months is considered rare, occurring in only 2.4–2.6% of cases (13,14). Cavazzuti et al. (15) reported a single case with TA onset at age 6.5 months. Most patients in our study share unusual features, and the same conclusion applies to two previous studies (16,17). None of our patients had or developed paroxysmal dyskinesia, as recently reported in patients with early-onset absences (18).

Analysis of the clinical and EEG features allows us to lump together four cases (cases 5, 6, 7, and 9). This group is characterized by frequent daily brief absences, lasting >4 s, and frequently asymmetric interictal changes with a left predominance in three. In this group, the prognosis is poor because of neurodevelopmental deficits and/or uncontrolled epilepsy. Cases 5 and 7 had absences associated with eyelid myoclonias, and case 5 also exhibited photosensitivity. A syndrome of absences with eyelid myoclonias was first reported by Jeavons (19). However, such patients typically have seizure onset between ages 2 and 5 years, earlier than in CAE, and also are characterized by brief absences associated with eye closure, a high degree of photosensitivity; GTCSs are common (5). For case 7, no photosensitivity was found. In case 5, the occurrence of four tonic seizures, reported by the family but not documented otherwise, do not allow us to establish a precise syndromic diagnosis. Cases 1 and 2 share the appearance during follow-up of a myoclonic component after an initial period of simple absences. Myoclonias appeared at age 5 years, with a possible time relation with the use of LTG (case 2), or a worsening effect of LTG after the occurrence of myoclonias (case 1). Aggravating effects of LTG on the myoclonic components of absences has been reported (20). In our two cases, EEG records showed focal abnormalities, but neuroimaging was normal. In both, seizures remained uncontrolled. In a large cohort of patients with IGE and a mean follow-up of 16 years, Lombroso et al. (21), found 56% of patients with focal abnormalities in the temporal and the frontal areas. In the group with absence seizures, this percentage was highest (60%). Cortical microdysgenesis was reported in patients with IGE by Meencke and Janz (22). An explanation for these focal features found in IGE is that EEG focalities might originate from preexisting cortical microdysgenesis. Clinically typical absences were produced by electrical stimulation of the mesial frontal lobe by Bancaud et al. (23). In cases 1 and 2, the presence of clear focal changes may orient the diagnosis toward a focal epilepsy with bilateral synchrony, but we must stress that our patients had no other seizure type and that a long-term epilepsy with absences as the only seizure type is not characteristic of focal epilepsies. The last two cases (cases 4 and 10) were not related to the previous subgroup, but both shared poor neuropsychological outcome.

The main characteristic of patients with early-onset absences seems to be the poor overall prognosis. Most IGEs are considered benign in childhood, and children with TA tend to have normal development. However, some authors have a less optimistic view. Loiseau et al. (24) reported that one third of patients with TA had inadequate social adjustment. In a recent controlled study, Echenne et al. (25) observed low IQ and significant impairment of various cognitive areas, which led to variable school difficulties in most children with absences. In most studies, however, none of psychometric data were collected before the onset of TA, which makes it difficult to determine whether the impairment of mental development is acquired and related to epilepsy, or preexisting.

A clear relation between age at onset of TA and prognosis is not established. Late onset after age 8 years is associated with increased occurrence of GTCSs (24). In their uncontrolled study, Hirsch et al. (7) reported that neither the age of onset nor the ictal symptoms are correlated with the prognosis of epileptic syndrome with TA. These authors found TA with early onset in three of their four prognosis groups: five cases hade an onset earlier than 4 years, and two of these had a benign outcome, two developed uncontrolled seizures, the last patient was characterized by the association of absences and photosensitivity. Covanis (26) studied 19 children with early-onset TA before age 3 years: two groups were identified (group A, seven children, with simple absences, and group B, 12 children, with absence associated to a marked myoclonic component). This author concluded that early-onset TAs have a more unfavorable prognosis than does classic CAE.

Absences persisted despite polytherapy in five of 10 children in this group. However, the follow-up is not sufficient for a definitive conclusion because four of the five resistant cases have not reached age 12 years. Only two patients had a favorable outcome with fully controlled epilepsy and normal school performance; three had controlled epilepsy but lingering neuropsychological and/or behavioral problems, and this may be an indication that the difficulties are not necessarily related to the persistence of absences. Early-onset absences were thus associated in this study with neurodevelopmental or behavioral difficulties in eight children. The association with developmental delay or mental retardation also was observed in four of the five patients reported by Aicardi (9).

CONCLUSION

The clinical and EEG analysis of 10 consecutive patients with absences with onset before age 3 years led to a diagnosis of a classic absence syndrome in only two, who may represent the beginning of the age-distribution curve of CAE or EMA. Eight of 10 were unclassifiable, and obviously heterogeneous. We noted some unusual, yet unspecific characterictics in some: a frequent myoclonic component, the brief duration of the absences, or the evidence of asymmetric EEGs or focal abnormalities. The global prognosis appears less favorable than that in CAE, because of frequently uncontrolled epilepsy and of neurodevelopmental difficulties, even when seizures are under control. Early-onset absence epilepsies are uncommon and a multicenter study would be useful to delineate specific diagnostic and prognostic features.

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