APPENDIX 6

Epilepsy syndromes

Table A6.1 Onset in neonatal period and infancy

SELF-LIMITED FOCAL EPILEPSY SYNDROMES	SELF-LIMITED NEONATAL SEIZURES AND SELF-LIMITED FAMILIAL NEONATAL EPILEPSY (SeLNE)
Age at onset	2 to 7 days.
Resolves by	6 weeks; may persist up to 6 months; up to one-third will have seizures later in life.
Seizures	Focal tonic seizures, most often affecting the head, face, and limbs. May experience vocalization or automatisms, and about one-third will present with cyanosis (bluish skin color) and apnea (breathing stops). Seizures are brief but frequent, appearing in clusters and may occur over hours or several days.[1]
Management and treatment	Antiseizure medication (carbamazepine is usually used for *KCNQ2*); often able to stop within weeks without recurrence.[1]
Cause	Genetic.
Genes	*KCNQ2, KCNQ3*.
Inheritance pattern	Inherited or *de novo*.[1]
Developmental impact	Typical development.[1]
Comorbidities	A minority of individuals may have learning difficulties or mild motor impairment.[1]

Cont'd.

SELF-LIMITED FOCAL EPILEPSY SYNDROMES

SELF-LIMITED FAMILIAL INFANTILE EPILEPSY AND SELF-LIMITED INFANTILE EPILEPSY (SeLIE)	
Age at onset	3 to 20 months.[1]
Resolves by	Typically within 1 year of onset.[1]
Seizures	Focal seizures with behavioral arrest, staring, cyanosis, clonic movements often frequent (5 to 10 per day over several days). Seizures may progress from focal to bilateral tonic-clonic.[1]
Management and treatment	Antiseizure medication.
Cause	Genetic.
Genes	*PRRT2, SCN2A, KCNQ2, KCNQ3*.[1]
Inheritance pattern	Inherited or *de novo*.[1]
Developmental impact	Typical development.[1]
Comorbidities	Movement disorders may develop later in life.[2]
GENETIC EPILEPSY WITH FEBRILE SEIZURES PLUS (GEFS+)	
Age at onset	Under 6 months.[1]
Resolves by	May resolve by puberty.[1]
Seizures	Febrile seizures occurring outside the typical time frame for febrile seizures (i.e., before 6 months of age, or after 6 years of age); additional generalized or focal seizures also occur.[1]
Management and treatment	Antiseizure medication.
Cause	Genetic.
Genes	*SCN1A, SCN1B, GABA, PCDH19, STX1B*.[1]
Inheritance pattern	Typically inherited, may be *de novo*.[1]
Developmental impact	Developmental delay may occur.[1]
Comorbidities	Typically none associated.
MYOCLONIC EPILEPSY IN INFANCY	
Age at onset	6 months to 2 years.[3]
Resolves by	6 months to 5 years from onset;[3] approximately 10 percent of individuals will develop another type of epilepsy during late childhood or adolescence.[1]

Cont'd.

SELF-LIMITED FOCAL EPILEPSY SYNDROMES	**Seizures**	Myoclonic seizures occurring in frequent clusters involving the head and upper arms; may be triggered by sound, light, or touch.[4]
	Management and treatment	Antiseizure medication, which can often be stopped after 3 to 5 years.[4]
	Cause	Likely genetic.
	Genes	None identified.
	Inheritance pattern	Family history of febrile seizures or epilepsy in about 10 percent.
	Developmental impact	Developmental progress is typically normal before onset of seizures; 60 to 85 percent have normal developmental progress at long-term follow-up.[1]
	Comorbidities	Mild learning difficulties and attention problems may occur.[1]
DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHIES (DEES)	**EARLY-INFANTILE DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHY (EIDEE) (OHTAHARA SYNDROME OR EARLY MYOCLONIC ENCEPHALOPATHY)**	
	Age at onset	First 3 months of life.[1]
	Seizures	Generalized or focal, tonic (Ohtahara syndrome), or myoclonic (early myoclonic encephalopathy). Other types of seizures might be seen, such as epileptic spasms. Seizures occur frequently in clusters. Interictal EEG is abnormal.[1] May progress to West syndrome or Lennox-Gastaut syndrome.[5]
	Management and treatment	Often drug-resistant seizures. May be treated with ACTH,* high-dose steroids, antiseizure medication, ketogenic diet, neuromodulation device (vagus nerve stimulator), or epilepsy surgery.[1,6]
	Cause	Genetic, structural, metabolic.[1,7]
	Genes	*SCN2A, SCN8A, KCNQ2, STXBP1, CDKL5, KCNT1, UBA5*.[1]
	Inheritance pattern	Family history not usually present; *de novo*.[7, 8]
	Developmental impact	Developmental delay may occur prior to onset of seizures; moderate to profound developmental delay develops over time.[1]
	Comorbidities	Intellectual disability, movement disorders, or abnormal tone often present.[1,6]
	EPILEPSY OF INFANCY WITH MIGRATING FOCAL SEIZURES	
	Age at onset	First 6 months of life.[1]
	Seizures	Focal clonic or tonic that migrate to multiple areas of the brain; seizures may increase in frequency over time and may lead to status epilepticus.[9]
	Management and treatment	Drug-resistant seizures; multiple antiseizure medications may be tried. Ketogenic diet or neuromodulation (vagus nerve stimulation) may be options.[10]
	Cause	Genetic.
	Genes	*KCNT1, SCNA1, SCNA2, TBC1D24,* and others.[1]

Cont'd.

DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHIES (DEES)	**Inheritance pattern**	Typically *de novo*.[10]
	Developmental impact	Developmental delay is severe. Typically, a poor prognosis with decreased life expectancy.[1]
	Comorbidities	Intellectual disability, microcephaly,† movement disorders, gastrointestinal disorders.[10,1]
	INFANTILE EPILEPTIC SPASMS SYNDROME (IESS) (WEST SYNDROME)	
	Age at onset	3 to 12 months.[7]
	Seizures	Epileptic spasms ranging from a few to over a hundred in clusters while awake or during sleep. Focal seizures may also occur at the beginning or end of the spasms. [1,7] Hypsarrhythmia on interictal.[12] Individuals with this syndrome often develop other epilepsy syndromes.
	Management and treatment	ACTH/high-dose steroids and/or antiseizure medication (e.g., in case of tuberous sclerosis, vigabatrin might be a good option); ketogenic diet might be considered.[13]
	Cause	Structural (30 percent), genetic, metabolic.[14,15]
	Genes/ chromosomes	*ARX*, *CDKL5*, *STXBP1*, *SPTAN1*, *IQSEC2*, *TSC1*, *TSC2*, Trisomy 21.
	Inheritance pattern	Family history is rare, typically *de novo* when genetic cause.
	Developmental impact	Developmental progress is typically normal before onset of seizures; developmental delay occurs over time.[1]
	Comorbidities	Down syndrome and tuberous sclerosis are associated with this syndrome.[14] Intellectual disability develops in most.[15] Autism spectrum disorder may develop.[15]
	DRAVET SYNDROME (PREVIOUSLY KNOWN AS SEVERE MYOCLONIC EPILEPSY OF INFANCY)	
	Age at onset	First year of life.[16]
	Seizures	Focal, focal to bilateral tonic-clonic seizures and generalized seizures that are often prolonged and may lead to status epilepticus.[17] Myoclonic and absence seizures may also occur. Febrile seizures are the first seizure in about 60 percent of individuals.[16] Seizures may be triggered by vaccines, fever, flashing lights, or stress. Drug-resistant seizures which are lifelong.[17]
	Management and treatment	Antiseizure medication, but avoid sodium channel blockers,‡ medical cannabis, ketogenic diet may be tried.[18]
	Cause	Genetic.
	Genes	*SCNA1*.[16]
	Inheritance pattern	May be inherited and family history of seizures present in 30 to 50 percent; most are *de novo*.[17]
	Developmental impact	Developmental delay occurs after seizures begin and are typically apparent by age 3.[1,18]
	Comorbidities	Multiple comorbidities including intellectual disabilities, movement disorders, and sleep disorders.[16, 18]

Cont'd.

ETIOLOGY-SPECIFIC EPILEPSY SYNDROMES

KCNQ2-DEE	
Age at onset	Within the first few days of life.[1]
Seizures	Tonic, myoclonic, or focal seizures that are often accompanied by apnea (breathing stops).[1]
Management and treatment	Antiseizure medication such as carbamazepine or oxcarbazepine; seizures are often resistant to typical first-line antiseizure medications[1]
Cause	Genetic.
Genes	*KCNQ2*.[1]
Inheritance pattern	*De novo*.[1]
Developmental impact	Developmental delay is moderate to severe.[1]
Comorbidities	Intellectual disability, movement disorders, autism spectrum disorder, behavior disorders, and sleep disorders.[19]
PYRIDOXINE DEPENDENT (ALDH7A1)-DEE (PD-DEE) AND PYRIDOX(AM)INE-5'-PHOPHATE DEFICIENCY (PNPO)-DEE (P5PD-DEE)	
Age at onset	May occur before birth with intrauterine convulsions or within the first few days of life; about 25 percent present after the neonatal period, up to 3 years; often born prematurely.[1]
Seizures	Myoclonus, focal, epileptic spasms, generalized tonic-clonic; seizures are frequent and often prolonged and may lead to status epilepticus.[1]
Management and treatment	Seizures are often resistant to antiseizure medications but respond well to supplements of pyridoxine or pyridoxal-5' phosphate, which may be needed lifelong. Sometimes leucovorin (a form of folic acid) might be needed.[1,20]
Cause	Metabolic, genetic.[1]
Genes	*ALDH7A1, PLBP, PNPO*.[1]
Inheritance pattern	Inherited.[21]
Developmental impact	Developmental delay.[21]
Comorbidities	Intellectual disabilities range from mild to severe in most individuals.[1]
CDKL5-DEE	
Age at onset	Within the first 6 weeks of life.
Seizures	Tonic, focal, generalized tonic-clonic, epileptic spasms, stereotyped hand movements.[1,22]
Management and treatment	Drug-resistant seizures; sustained benefit has not been shown with antiseizure medication. Ganaxolone, a synthetic neuroactive steroid, is approved by the US Food and Drug Administration (FDA) to treat seizures in individuals who are 2 years or older.

Cont'd.

ETIOLOGY-SPECIFIC EPILEPSY SYNDROMES		
	Cause	Genetic.
	Genes	*CDKL5*; an X-linked gene, seen predominantly in females (4:1).[1]
	Inheritance pattern	Typically *de novo*, may be inherited.[23]
	Developmental impact	Developmental delay is severe, and less than 25 percent of individuals will be able to walk independently or speak single words.[1]
	PCDH19 CLUSTERING EPILEPSY	
	Age at onset	Within the first year of life.
	Seizures	Focal impaired aware (may include screaming), tonic-clonic; seizures may be triggered by fever and often occur in clusters; may lead to status epilepticus. Seizures may resolve in at least 25 percent in adolescence or later.[1]
	Management and treatment	Drug-resistant seizures, often requiring multiple antiseizure medications.[24]
	Cause	Genetic
	Genes	*PCDH19*; an X-linked gene, seen predominantly in females.[1]
	Inheritance pattern	Inherited or *de novo*; approximately half are *de novo*.[1]
	Developmental impact	Developmental delay is common; microcephaly may occur.[1]
	Comorbidities	Intellectual disability, movement disorders, and migraines.[1]
	STURGE-WEBER SYNDROME	
	Age at onset	Condition is present at birth, but seizures typically begin within the first 6 months of life.
	Seizures	Focal, focal to bilateral tonic-clonic, epileptic spasms, status epilepticus; seizures present in 75 to 85 percent of individuals.[1]
	Management and treatment	Antiseizure medication such as carbamazepine, oxcarbazepine, and levetiracetam often provide good seizure control. If drug-resistant seizures develop, epilepsy surgery may be recommended. Ketogenic diet may be recommended.[1, 25] Low-dose aspirin beginning in infancy may be given to prevent stroke-like events.[26]
	Cause	Structural, genetic.
	Genes	*GNAQ*.27
	Inheritance pattern	*De novo*.[27]
	Developmental impact	Developmental delay is common.[26]
	Comorbidities	Angiomas (lesions caused by atypical growth of blood vessels) may be present in the brain, in the eye, and as capillary malformations on the face. When these appear on the face they are referred to as port-wine stains. Stroke-like events resulting in hemiparesis (weakness or partial paralysis on one side of the body) may occur.[26] Learning disorders, psychiatric and behavioral conditions, headaches, and intellectual disability may occur.[1]

Cont'd.

ETIOLOGY-SPECIFIC EPILEPSY SYNDROMES

GELASTIC SEIZURES WITH HYPOTHALAMIC HAMARTOMA	
Age at onset	Within the first year of life in 85 percent of individuals.[1]
Seizures	Gelastic (laughing episodes) or dacrystic (crying episodes) seizures; may also progress over time to focal impaired awareness and generalized seizures.[1]
Management and treatment	Drug-resistant seizures; epilepsy surgery may be an option for treatment.[1]
Cause	Structural (lesions in the hypothalamus§ that are present at birth); genetic.
Genes	About 5 percent are associated with *GL13*.[1]
Inheritance pattern	Typically *de novo*[28]
Developmental impact	Developmental delay may occur.[29]
Comorbidities	Intellectual disability, behavioral disorders, and psychiatric disorders may occur.[29]

* ACTH (adrenocorticotropic hormone) is a medication given over several weeks, often along with antiseizure medications, and is particularly effective in the treatment of infantile spasms.[30]

† A condition in which the individual's head is much smaller than typical and may be caused by lack of brain development and growth.

‡ A type of antiseizure medication that blocks sodium ions from flowing in and out of neurons, inhibiting seizure activity.[31]

§ A structure in the brain that helps to maintain a stable environment in the body by controlling hormones and the autonomic nervous system, including body temperature, blood pressure, hunger, thirst, mood, and sleep.[32]

Table A6.2 Onset in childhood

SELF-LIMITED FOCAL EPILEPSY SYNDROMES		
	SELF-LIMITED EPILEPSY WITH CENTROTEMPORAL SPIKES (SeLECTS) (PREVIOUSLY KNOWN AS ROLANDIC EPILEPSY)	
	Age at onset	3 to 14 years.[33]
	Resolves by	Puberty (age 13); may persist until 18 years.[33]
	Seizures	Brief focal clonic or tonic seizures that involve the throat and/or tongue and one side of the bottom half of the face; may progress to focal to bilateral tonic-clonic;[33] often sporadic.[7]
	Management and treatment	Antiseizure medication; often can be stopped 2 to 4 years after starting. Complete remission is common.[34]
	Cause	Genetic cause is suspected but is not yet well characterized.[33]
	Genes	None identified.
	Inheritance pattern	Family history of epilepsy or febrile seizures may exist, but typically not a family history of SeLECTS.[33]
	Developmental impact	Typical development.[33]
	Comorbidities	Behavior disorders and language impairment may occur but often improve after the epilepsy resolves.[33]
	SELF-LIMITED EPILEPSY WITH AUTONOMIC SEIZURES (SeLEAS) (PREVIOUSLY KNOWN AS PANAYIOTOPOULOS SYNDROME)	
	Age at onset	3 to 6 years.[33]
	Resolves by	1 to 2 years after onset.
	Seizures	Focal autonomic seizures; vomiting is a frequent sign (75 percent of individuals); seizures are infrequent and 25 percent have only a single seizure.[33] Seizures occur with sleep and may be prolonged, lasting more than 30 minutes
	Management and treatment	Antiseizure medication; often able to be stopped within 1 to 2 years. Approximately 20 percent of individuals will develop another epilepsy syndrome.[33]
	Cause	Unknown.
	Genes	None identified.
	Inheritance pattern	Minority have a family history of epilepsy.[35]
	Developmental impact	Typical development.[33]
	Comorbidities	May have mild language impairment that often improves after the epilepsy resolves.[36]

Cont'd.

SELF-LIMITED FOCAL EPILEPSY SYNDROMES		
	CHILDHOOD OCCIPITAL VISUAL EPILEPSY (COVE)	
	Age at onset	8 to 9 years.[33]
	Resolves by	Puberty.[33]
	Seizures	Focal aware or impaired awareness seizures with or without motor signs occur while awake and are often accompanied by sensory phenomena; may be frequent and brief with hallucinations or migraines.[33] Typical absence seizures may also occur.[33] Vision changes, visual hallucinations, eye pain, repetitive eye movements may also occur.[37] Postical headache is common.[33]
	Management and treatment	Antiseizure medication if seizures recur; complete remission of seizures in 50 to 80 percent of individuals with or without antiseizure medication occurs by puberty.[33]
	Cause	Genetic cause is suspected but is not yet well characterized.[33]
	Genes	None identified.
	Inheritance pattern	Family history of febrile seizures or epilepsy in one-third of individuals and family history of migraine in 9 to 16 percent of individuals.[33]
	Developmental impact	Typical development.[33]
	Comorbidities	Typically none associated.[38]
	PHOTOSENSITIVE OCCIPITAL LOBE EPILEPSY (POLE)	
	Age at onset	4 to 17 years.[39]
	Resolves by	Varies.
	Seizures	Focal aware seizures with sensory phenomena; seizures are brief and rarely progress to a focal to bilateral tonic-clonic seizure.[40] Seizures are often triggered by light sources (e.g., TV, video games, flickering sunlight.[41]
	Management and treatment	Antiseizure medication; avoid known triggers.
	Cause	Genetic cause suspected.[42]
	Genes	None identified.
	Inheritance pattern	Family history of the syndrome is present in one-third of individuals.[39]
	Developmental impact	Developmental delays may exist.[40]
	Comorbidities	Typically none associated.

Cont'd.

DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHIES (DEES)

EPILEPSY WITH MYOCLONIC-ATONIC SEIZURES (EMAtS) (PREVIOUSLY KNOWN AS DOOSE SYNDROME; ALSO KNOWN AS EPILEPSY WITH MYOCLONIC-ASTATIC SEIZURES)	
Age at onset	2 to 6 years.[33]
Seizures	Generalized tonic-clonic, myoclonic, myoclonic-atonic, absence; onset of seizures is often abrupt and explosive and may include status epilepticus;[33] may have a history of febrile seizures.[43]
Management and treatment	Ketogenic diet may be successful; antiseizure medication. Drug-resistant seizures that may resolve in about two-thirds of individuals; antiseizure medication can be stopped.[33]
Cause	Genetic.
Genes	*SCN1A, SCN1B, SCN2A, STX1B, SLC6A1, CHD2, SYNGAP1, NEXMIF, KIAA2022, SLC2A1*.[33]
Inheritance pattern	Family history of febrile seizures in 50 percent of individuals.[44]
Developmental impact	Developmental delay or regression may occur, which may improve once seizures resolve.[33]
Comorbidities	Intellectual disability, behavior disorders, sleep disorders, or movement disorders.[33]
LENNOX-GASTAUT SYNDROME	
Age at onset	18 months to 8 years.[33]
Seizures	Tonic, atonic, atypical absence seizures, often subtle, along with multiple seizure types.[45] Ten to 30 percent of individuals have another epilepsy syndrome[45] diagnosed prior to Lennox-Gastaut syndrome, and 80 to 90 percent will continue to have seizures into adulthood.[46]
Management and treatment	No definitive treatment; may try antiseizure medication, steroids, ketogenic diet, medical cannabis, neuromodulation (vagal nerve stimulator),[13] or epilepsy surgery (corpus callosotomy).
Cause	Structural (70 percent), genetic.[33, 45]
Genes	None identified.[45]
Inheritance pattern	Typically occurs without any family history;[33] *de novo* mutation possible.[47]
Developmental impact	Developmental delay is severe and may be present before the onset of seizures.[45]
Comorbidities	Intellectual disability, behavior disorders, sleep disorders, and autism spectrum disorder are common.[48]
LANDAU KLEFFNER SYNDROME (CONSIDERED A SUBTYPE OF EE-SWAS)	
Age at onset	3 to 8 years.[49]
Seizures	Obvious seizures occur in 70 percent of individuals and are often present as absence or focal seizures. Seizures are often self-limiting. Presents with progressive loss of speech and comprehension of speech.
Management and treatment	Antiseizure medication, steroids, epilepsy surgery;[49] seizures may resolve.

Cont'd.

DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHIES (DEES)		
	Cause	Genetic.[49]
	Genes	*GRIN2A*.[50]
	Inheritance pattern	Unknown; family history usually not present.[7]
	Developmental impact	Significant regression in language, and even if seizures resolve language impairment persists in over 80 percent of individuals.[7]
	Comorbidities	Language impairment, behavior disorders, cognitive impairment, ADHD, depression, anxiety, sleep disorders.
	DEVELOPMENTAL EPILEPTIC ENCEPHALOPATHY WITH CONTINUOUS SPIKE-WAVE ACTIVATION IN SLEEP (DEE-SWAS), AND EPILEPTIC ENCEPHALOPATHY WITH SPIKE-WAVE ACTIVATION IN SLEEP (EE-SWAS) (PREVIOUSLY KNOWN AS ELECTRICAL STATUS EPILEPTICUS IN SLEEP [ESES])	
	Age at onset	2 to 12 years.[33]
	Seizures	Focal aware or impaired awareness and focal to bilateral tonic-clonic seizures, absence, atonic; seizure activity occurs during sleep or times of drowsiness; multiple types of seizures may occur or in some individuals, no clinical seizures occur (only electrographic seizures).[33] Seizures typically resolve by puberty.[33,51]
	Management and treatment	Antiseizure medication, steroids, epilepsy surgery, or ketogenic diet.[33,51]
	Cause	Structural, genetic, metabolic, or unknown.[52]
	Genes	*GRIN2A*.[33]
	Inheritance pattern	Family history of seizures in up to 50 percent of individuals;[33] *de novo* mutation possible.[53]
	Developmental impact	Developmental delay is often present before the onset of seizures and continues to progress after the onset. Developmental delay may persist even if seizures resolve, although improvement may occur, particularly when the seizures last less than two years.[33]
	Comorbidities	Cognitive impairment, language impairment, movement disorders, behavioral disorders, autism spectrum disorder, and psychiatric disorders may occur.[33, 52]
	HEMICONVULSION-HEMIPLEGIA-EPILEPSY SYNDROME (HHE)	
	Age at onset	Under 4 years.[33]
	Seizures	Focal clonic status epilepticus. Seizures occur in an acute stage (febrile seizure that progresses to status epilepticus) and a chronic stage (focal seizures, focal to bilateral tonic-clonic). Seizures lead to swelling and atrophy* in half of the brain.[33]
	Management and treatment	Drug-resistant seizures; antiseizure medication, or epilepsy surgery.[33]
	Cause	Unknown.
	Genes	None identified.
	Inheritance pattern	Unknown.

Cont'd.

DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHIES (DEES)	**Developmental impact**	Developmental delay may occur and may be present before the first seizure.[33]
	Comorbidities	Hemiparesis (half of the body is paralyzed) may result; in the majority of individuals, this is permanent; in 20 percent this resolves within a year.[33] Language impairment may occur and typically resolves within two months.[33] disability may occur.[33]
	RASMUSSEN SYNDROME	
	Age at onset	1 to 10 years;[54] in 10 percent of individuals, onset is in adolescence or adulthood.[33]
	Seizures	Focal aware or impaired aware motor, focal atonic, focal to bilateral tonic-clonic; seizures increase in frequency and severity over time.[33,54]
	Management and treatment	Drug-resistant seizures; antiseizure medication,[55] steroids, immunologic therapies, or epilepsy surgery.[54]
	Cause	Unknown.
	Genes	None identified.
	Inheritance pattern	Unknown.
	Developmental impact	Developmental delay is common.[55]
	Comorbidities	Progressive weakness on one side of the body; cognitive, memory and language impairment.[56]
GENERALIZED EPILEPSY SYNDROMES	**CHILDHOOD ABSENCE EPILEPSY**	
	Age at onset	2 to 13 years;[57] seizures may resolve by age 10 to 14.
	Seizures	Absence seizures; occur multiple times per day and last less than 20 seconds; may be provoked by hyperventilation; history of febrile seizures in 10 to 15 percent.[57] may later develop juvenile myoclonic epilepsy or juvenile absence epilepsy.
	Management and treatment	Antiseizure medication.
	Cause	Presumed genetic cause; idiopathic generalized epilepsy.
	Genes	None identified.
	Inheritance pattern	Family history of absence seizures or other generalized seizures present in about one-third of individuals.[58]
	Developmental impact	Developmental delays may occur.[59]
	Comorbidities	ADHD, learning impairment, behavior disorders, or psychiatric disorders may occur.[59–61]
	EPILEPSY WITH MYOCLONIC ABSENCES	
	Age at onset	2 to 12 years.[62]
	Seizures	Myoclonic absences, generalized tonic-clonic, atonic; seizures are frequent (several times a day) and last 10 to 60 seconds; may be triggered by hyperventilation.[62,63] Seizures persist into adulthood in more than 50 percent of individuals.[62]

Cont'd.

GENERALIZED EPILEPSY SYNDROMES		
	Management and treatment	Drug-resistant seizures; antiseizure medications.[64]
	Cause	Genetic, structural.
	Genes	None identified.
	Inheritance pattern	Family history of seizures present in 20 percent of individuals.[33]
	Developmental impact	Developmental delays may occur.
	Comorbidities	Intellectual disability;[33] learning impairment is present in 70 percent.[62]
	EPILEPSY WITH EYELID MYOCLONIAS (PREVIOUSLY KNOWN AS JEAVONS SYNDROME)	
	Age at onset	2 to 14 years.[65]
	Seizures	Myoclonic jerks in the eyelids that last 1 to 3 seconds and are repetitive; absence seizures, generalized tonic-clonic.[33] Seizures triggered by eye closure, sunlight, or flashing lights.[65,66] Seizures often persist lifelong.[33]
	Management and treatment	Drug-resistant seizures; antiseizure medication.[33] Ketogenic diet may help as well as the use of blue lenses.[66]
	Cause	Genetic.
	Genes	None identified.
	Inheritance pattern	Family history of seizures or epilepsy in 40 to 80 percent of individuals.[65]
	Developmental impact	Typical development.[65]
	Comorbidities	Mild learning impairment and attention problems may occur.[67]

* Atrophy refers to the shrinking or degeneration of tissues. Brain (cerebral) atrophy specifically is the loss of neurons and their communicating networks, often accompanied by a loss in the overall brain volume.

Table A6.3 Onset in adolescence and adulthood

FOCAL EPILEPSY SYNDROMES	**AUTOSOMAL DOMINANT EPILEPSY WITH AUDITORY FEATURES**	
	Age at onset	10 to 30 years.[68]
	Seizures	Focal seizures with sensory phenomena (mostly sounds), aphasia; seizures are mild and infrequent. mainly occurring at night.[68]
	Management and treatment	Antiseizure medication; seizures well controlled.[68]
	Cause	Genetic.
	Gene	*LGI1, RELN.*[69]
	Inheritance pattern	Inherited; family history typically present.[69]
	Developmental impact	Typical development.[69]
	Comorbidities	Typically none associated.
	OTHER FAMILIAL TEMPORAL LOBE EPILEPSIES	
	Age at onset	Older than 10 years.[70]
	Seizures	Focal aware or impaired awareness with sensory phenomena (visual or sound hallucinations); seizures are often mild and infrequent; focal to bilateral tonic-clonic seizures in two-thirds of individuals.[70]
	Management and treatment	Antiseizure medication; seizures well controlled.[70]
	Cause	Genetic.
	Gene	Rarely *DEPDC5*.[70]
	Inheritance pattern	Inherited; family history in 60 percent of individuals.[70]
	Developmental impact	Typical development.
	Comorbidities	Typically none associated.
GENERALIZED EPILEPSY SYNDROMES	**JUVENILE ABSENCE EPILEPSY**	
	Age at onset	8 to 20 years.[71]
	Seizures	Absence, generalized tonic-clonic, myoclonic jerks.[71,72] May evolve into juvenile myoclonic epilepsy.[72]
	Management and treatment	Antiseizure medication; required lifelong.[71]
	Cause	Presumed genetic cause; idiopathic generalized epilepsy (IGE).
	Gene	None identified.
	Inheritance pattern	Family history of seizures or epilepsy in 42 percent.[72]

Cont'd.

GENERALIZED EPILEPSY SYNDROMES	**Developmental impact**	Typical development.[71]
	Comorbidities	ADHD, learning impairment.73
	JUVENILE MYOCLONIC EPILEPSY	
	Age at onset	8 to 40 years.[74]
	Seizures	Myoclonic, generalized tonic-clonic, absence; seizures often occur within 1 to 2 hours of waking. Seizures may be triggered by sleep deprivation, stress, or flashing lights.[74] May evolve from childhood absence epilepsy.[74]
	Management and treatment	Antiseizure medication.
	Cause	Presumed genetic cause; idiopathic generalized epilepsy (IGE).
	Gene	None identified.
	Inheritance pattern	Family history occasionally present.[75]
	Developmental impact	Typical development.
	Comorbidities	ADHD, learning impairment.[61, 73]
	EPILEPSY WITH GENERALIZED TONIC-CLONIC SEIZURES ALONE	
	Age at onset	5 to 40 years.[76]
	Seizures	Generalized tonic-clonic; seizures occur within 1 to 2 hours of awakening; may be triggered by sleep deprivation.[76] History febrile seizures may be present.
	Management and treatment	Antiseizure medication; required lifelong.[76]
	Cause	Presumed genetic cause; idiopathic generalized epilepsy (IGE).
	Gene	None identified.
	Inheritance pattern	Family history of epilepsy may be present.76
	Developmental impact	Typical development.
	Comorbidities	Typically none associated.

Table A6.4 Onset at a variable age

FAMILIAL FOCAL EPILEPSY WITH VARIABLE FOCI	
Age at onset	1 to 52 years.[77]
Seizures	Focal, focal to bilateral tonic-clonic seizures
Management and treatment	Antiseizure medication; seizures well controlled.[77]
Cause	Genetic, genetic-structural.
Gene	*DEPDC5, NPRL2, NPRL3, TSC1, TSC2.*[77]
Inheritance pattern	Family history required for diagnosis.[77]
Developmental impact	Typical development; may have mild developmental impact.
Comorbidities	May have mild intellectual disability, autism spectrum disorders, and behavior disorders.
REFLEX EPILEPSY (EPILEPSY WITH READING-INDUCED SEIZURES)	
Age at onset	10 to 46 years.
Seizures	Seizures occur in response to particular stimuli (e.g., reading) rather than spontaneously. Myoclonic jerks, generalized tonic-clonic; reflex seizures.
Management and treatment	Antiseizure medication; limit reading.
Cause	Genetic.
Gene	None identified.
Inheritance pattern	Family history in 20 to 40 percent of individuals.[78]
Developmental impact	Typical development.
Comorbidities	Typically none associated.
PROGRESSIVE MYOCLONUS EPILEPSY	
Age at onset	2 to 50 years.[55]
Seizures	Myoclonic, generalized tonic-clonic.
Management and treatment	Drug-resistant seizures; antiseizure medication.
Cause	Variable; genetic and metabolic.
Gene	Depends on the cause.
Inheritance pattern	Depends on the cause.
Developmental impact	Developmental delay is common and progressive.[55]
Comorbidities	Poor long-term prognosis.[79] Motor impairment, intellectual disability.[79]

Cont'd.

SLEEP-RELATED HYPERMOTOR (HYPERKINETIC) EPILEPSY (SHE) (PREVIOUSLY KNOWN AS NOCTURNAL FRONTAL LOBE EPILEPSY)	
Age at onset	1 to 60 years of age.[80]
Seizures	Focal, motor; seizures are brief (typically about 30 seconds) and occur during sleep.[55, 80]
Management and treatment	Antiseizure medications or epilepsy surgery; 30 percent of individuals have drug-resistant seizures.[55, 80]
Cause	Structural, genetic.
Gene	*KCNT1, DEPDC5, CHRNA4, CHRNB2, CHRNA2.*[80]
Inheritance pattern	Inherited, may be *de novo*.[55]
Developmental impact	Typical development.
Comorbidities	Intellectual disability, sleep disorders, or behavioral disorders may occur.[80]
MESIAL TEMPORAL LOBE EPILEPSY WITH HIPPOCAMPAL SCLEROSIS (MTLE-HS)	
Age at onset	Variable; most common in adolescents and young adults.[55]
Seizures	Focal aware or impaired awareness, tonic-clonic; autonomic signs and symptoms, automatisms, behavioral arrest, sensory phenomena.[55] May develop after prolonged febrile seizures. Seizures may be drug-resistant.[81]
Management and treatment	Antiseizure medication or epilepsy surgery; epilepsy surgery may provide full remission of this syndrome.[55]
Cause	Structural.
Gene	None identified.
Inheritance pattern	None.
Developmental impact	Typical development.
Comorbidities	Psychiatric disorders, cognitive and memory impairment.[55]
FEBRILE INFECTION-RELATED EPILEPSY SYNDROME (FIRES)	
Age at onset	2 to 17 years of age.[82]
Seizures	Focal impaired awareness, focal to bilateral tonic-clonic, status epilepticus; seizures occur 1 to 14 days after an illness with a fever.[82] Often leads to brain atrophy.[7] Seizures may be drug resistant.
Management and treatment	Antiseizure medication; often requires medically induced coma.[82]
Cause	Unknown.
Gene	None identified.
Inheritance pattern	None.
Developmental impact	Developmental delay is common.[82]
Comorbidities	Intellectual disability, motor impairment, and behavior disorders. A poor prognosis and death occurs in 10 percent of individuals.[33]

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