Edited by Christopher Gillberg, Richard Harrington and Hans-Christoph Steinhausen

A Clinician's Handbook of

Child and Adolescent Psychiatry

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A Clinician's Handbook of Child and Adolescent Psychiatry

This authoritative clinical handbook provides a comprehensive overview of the main disorders encountered by child and adolescent psychiatrists in clinical practice, ranging from eating, sleep and affective disorders to substance abuse, gender identity disorder and sexual abuse. The approach is evidence based and emphasis is on good clinical practice and quality control of patient care. In contrast to other books in the field, the authors' intention is not to cover exhaustively all the relevant science, but rather to present in condensed form any research findings that are significant for clinical practice.

For coherence, each chapter is constructed in the same way: introduction, definition and classification, epidemiology, the clinical picture, aetiology, treatment and outcome. The disorders covered are based on the ICD-10 and DSM-IV classifications, and appendices include documents for assessment of intervention planning and evaluation.

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Preface

Child and adolescent psychiatry is a rapidly expanding branch of medicine. Important research gains have been made in the past decades, and there is now a firm knowledge basis for many of the psychiatric disorders with child or adolescent onset. This knowledge has taken some time to filter through to clinical child and adolescent psychiatric services.

The aim of this book is to provide a comprehensive overview of the relevant objectives of child and adolescent psychiatry in clinical practice. The approach is decisively evidence based as far as this is possible with the current status in the field and thus avoids the consideration of various theoretical or even speculative approaches. Rather, the emphasis is on guidance for good clinical practice and help with the quality control of patient care. The various chapters have been written by experts in the respective fields. In contrast to other textbooks, the major objective is not to cover all relevant research findings exhaustively but rather to condense the scientific knowledge that is significant for clinical practice. Thus, the present deskbook deals with clinical disorders only.

The list and order of contents is guided by the ICD-10 and the various chapters also comment on the DSM-IV classification. A clear common structure for the chapters enforces the coherence of the monograph. After an introduction, definition and classification, epidemiology, the clinical picture, aetiology, treatment and outcome are specifically outlined for each disorder. Tables, figures and flowcharts, serve as additional sources of information and highlight special messages. Various appendices include important documents for assessment of intervention planning and evaluation.

The book is intended for child and adolescent psychiatrists, and for other specialists working in, or at the borders of, child mental health, including community and developmental pediatricians, school health doctors, child neurologists and adult and forensic psychiatrists. The language and writing style are such that it should also be a valuable source of clinical guidance for psychologists, social workers, nurses and other paramedical professionals working in the field.

The authorship is international with a strong European and North American orientation. The editors are grateful for the willingness and effectiveness of the authors to adopt the special structure and the intended message of this textbook and to bring in their expertise in order to provide an accurate and comprehensive account of the current state of knowledge of the various child and adolescent psychiatric disorders. Special thanks are also due to the publisher, most notably to Pauline Graham, Jo Bottrill and Mary Sanders for the assistance in producing this book.

Sadly, Richard Harrington died shortly before finalization of the manuscript of this book. His death is a great loss to his family, his colleagues and his friends. Besides his outstanding scientific contributions to child and adolescent psychiatry, he was a most skilful and experienced clinician and a genuine and warmhearted personality. This book will serve as a commemoration to Richard Harrington.

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Brain disorders

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Introduction

All mental functioning, be it normal or abnormal, is mediated by the brain. Thus, no child and adolescent psychiatric disorder can be thought of as not being brain related. However, there is a separate category of disorders in which the structure of the brain itself is disordered or in which the basic neurological functions are altered so that normal mental functioning may not result. This is most obvious in those disorders that result from morphological alterations of the brain structure due to a noxious agent or event, or due to a neurobiological deficit that seriously affects the organization and development of the brain.

Classification of brain disorder in childhood and adolescence is not very satisfactory. The major classes of brain disorders as set out in the ICD-10 are derived mainly from manifestations of disorders in adulthood with insufficient consideration of developmental aspects in childhood and adolescence. Thus, in contrast to most of the remaining chapter in this volume both ICD-10 and DSM-IV are not considered as the relevant framework for classification of brain disorders in childhood and adolescence.

In this chapter the following major brain disorders with a basic neurological alteration of brain structures and functions will be described: injury, infectious disorder, cerebral palsy, epilepsy and brain tumours. In an additional section the concept of minor brain dysfunction syndromes will be discussed. This concept has been very influential in the past and has largely been ignored in the more recent academic debate in child and adolescent psychiatry. Given its remaining relevance in practical child and adolescent psychiatry, an attempt will be made to identify those elements in the concept that reflect the notion fruitfully and validly that there is a continuum of brain-related symptoms between neurologically

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defined structural brain disorders, and the concept of minor brain dysfunction syndromes.

Given the similarities of psychopathological features and mechanisms in the various brain disorders, some repetition cannot be avoided in the present chapter. However, it was decided to have relatively complete descriptions of the various phenomena and issues of each brain disorder so that the busy clinician will get sufficient information from each section of this chapter.

Brain injury

Definition and classification

Traumatic head and brain injury results from an extended force that insults the brain and leads to a transient or persistent impairment of physical, cognitive, behavioural or emotional functions. It may be divided into open and closed head injuries. Open head injury is defined by penetration of the brain, e.g. a depressed skull fracture with underlying cerebral laceration. Usually localized brain damage is involved. Closed head injury is more common, secondary to traffic accidents or to a fall. The resulting damage may be marked by contusions, intracranical hematomes, intraventricular, subarachnoid, subdural and epidermal hemorrhages and contrecoup injuries opposite to the initial impact. Furthermore, diffuse damage of the brain may be a sequel.

A common classification of the severity in the acute stage distinguishes mild, moderate and severe brain injury. The differentiation usually is based on the extent or duration of coma and the posttraumatic amnestic period. Derived from adult traumatology, the Glasgow Coma Scale (GCS) has also been used most frequently in the population of children and adolescents. The GCS measures eye opening, verbal response and hand or leg movement by rating each response on a scale of 1 to 5 and aggregating all three items. Higher scores represent better responsiveness and prognosis.

According to the GCS mild brain injury is defined by a score of 13 or more, posttraumatic amnesia of less than 12 hours or a loss of consciousness for 5 minutes or less. Moderate brain injury is defined by a GCS score of 9 to 12, a post-traumatic amnestic period of 12 to 24 hours, or a loss of consciousness between 5 and 60 minutes (or even more in some studies). Finally, severe brain injury is associated with a GCS score of less than 9, or post-traumatic amnesia lasting more than 24 hours. However, the GCS has been criticized for being often too crude a measure for children. Comparisons of the research literature are hampered by different definitions of the depth and length of coma as a measure of severity. Several studies in children converge in using the above-mentioned GCS total scores as an indicator

of the depth of coma and extending the length of post-traumatic amnesia to 7–14 days in order to define severe brain injury.

Epidemiology

Incidence and prevalence rates

Traumatic brain injury is a very frequent cause of morbidity and mortality in childhood and adolescence. Estimated incidence rates range from 185 per 100 000 children from infancy to age 14 per annum to 295 per 100 000 adolescents and young adults aged 15 to 24 per annum in the United States. With a rate of 550 per 100 000 and per annum, the risk is highest among the 15–19 year olds. Figures around 45 000 children under 16 years with the number of deaths per annum being around 300 have been established in the United Kingdom. British figures document that 10% of children admitted to accident and emergency departments will have a moderate brain injury, and 1 per cent will have a severe injury. As many as 2.5 per cent of children may have sustained a head injury leading to admittance to accident and emergency departments during childhood.

Sex ratios

In terms of prevalence of brain injury, boys outnumber girls 2:1 with a lower rate in young children up to 5 years of age. The gender discrepancy emerges in infancy and is most prominent during school age and adolescence.

Implications for clinical practice

Head and brain injury is a leading cause of mortality and disability in young people and one of the most common causes of chronic brain syndromes in children. In some cases the injury results in transient or even permanent physical and/or cognitive and/or behavioural and emotional deficits. Highly sophisticated professional skills, including expert knowledge in psychopathology, are needed in order to assist the children who are victims of head and brain injury.

Clinical picture

Main features and symptoms

The symptoms due to brain injury vary considerably depending on cause, severity and type of head injury (open vs. closed), additional pre-morbid functioning and age of the child, post-traumatic coping and quality of the psychosocial environment. The various symptoms may be grouped on three functional levels as shown in Table 1.1.

On the neuropsychiatric level the clinical picture differs according to the phase of the disorder.

Table 1.1. Functional sequelae in brain injury

A. Neuropsychiatric
- Acute symptoms
  - Loss of consciousness, agitation, loss of orientation, short attention span
- Transient symptoms
  - Amnesia, slowing of impulse, affective lability, irritability, hallucinations, thought disorder
- Chronic symptoms
  - Psychopathology: agnosia, apraxia, dementia, disinhibition, hyperactivity, attention deficits, personality changes
  - General psychopathology: emotional and conduct disorders, adjustment disorders

B. Neurologic
- Headaches
- Sleep disturbances
- Epilepsy
- Hydrocephalus
- Spasticity
- Movement disorders
- Apallic syndrome

C. Endocrine
- Hormonal disturbances due to
  - Posterior pituitary deficiencies
  - Anterior pituitary deficiencies
  - Hypothalamic pituitary axis dysfunctions

D. Neurocognitive
- Impaired intellectual functions
- Reduced speed of information processing
- Language and communication skills impairment
- Impaired learning and memory
- Attention deficits
- Perceptual deficits
- Executive functions deficits

E. Educational
- Impaired progress in school
- Failing a grade
- Special education provision
- Scholastic skills deficit

F. Psychosocial
- Dysfunctional individual adaptation
- Dysfunctional family adaptation
- Social disintegration

- In the acute phase varying degrees of depth of coma dependent on the severity of the trauma are noticeable with a loss of consciousness even missing in some children. Mild head injury typically is associated only with transient symptoms of dizziness, headaches, confusion and fatigue with no loss of consciousness or a loss of consciousness not exceeding 20 minutes. A diagnosis of delirium may be stated when agitation, loss of orientation and short attention span are evident.
- There may be a transient phase of relatively short-lived psychopathological features due to a post-traumatic impairment of mental functioning including memory, impulse, affects and thought. Minor transient psychopathological syndromes include slowing of impulse, loss of initiative, forgetfulness, emotional lability and irritability. Moderate to severe transient psychopathological syndromes may include prolonged amnesia and even psychotic symptoms, like formal thought disorder, hallucinations and paranoid symptoms.
- In the chronic phase various specific impairments may become evident. In some children localized deficits leading to the syndromes of agnosia, apraxia and aphasia may result from the injury. A persistent intellectual impairment may justify a diagnosis of childhood dementia and a persistent behavioural pattern resembling a frontal lobe syndrome in adults may be emerging. This syndrome of social disinhibition is marked by a lack of impulse control, hyperactivity and attention deficits and may be accompanied by forgetfulness, talkativeness and carelessness. Owing to the persistent change in the child's behavioural features, a post-traumatic personality change may become noticeable.

  Even more common in the chronic phase are symptoms of general psychopathology. With the well-established increased risk of any child psychiatric disorder in brain disorder, there may be an interaction between neurological and behavioural factors in addition to separate paths for the two factors contributing to specific psychopathologies. These interactions may result in oppositional defiant and conduct disorders, anxiety disorders or affective disorders or rather than creating symptoms *de novo* may amplify a pre-existing hyperkinetic disorder. Among the adjustment disorders the possibility of some symptoms or, less common, the full picture of post-traumatic stress disorder should be considered.

In addition to the various neuropsychiatric features, there is a wide range of symptoms on the neurologic level.

- Various post-traumatic headaches resemble other chronic headaches like migraine and tension headaches, cluster, cervicogenic and myofacial pain headaches. In association with dizziness, nausea or vomiting, other problems of the trauma like hematomas, arterial dissection or aneurysm have to be ruled out.
- A sizeable proportion of patients with head injury experience sleep disturbances. The most frequent early symptom after head injury is hypersomnolence. It is

assumed that hypersomnolence results from damage of the reticular formation or the posterior hypothalamus. Insomnia is less common after brain trauma.

- Post-traumatic seizures at the time of the impact do not increase the risk of epilepsy necessarily. However, this is the case for seizures occurring later in recovery. In contrast to adults, children are more prone to develop early seizures and status epilepticus.
- Another potential complication of head injury is hydrocephalus, especially when there is subarachnoial or intraventicular hemorrhage. Hydrocephalus occurs only rarely when there is massive hemorrhage. The typical manifestation is months later.
- Only in the most severe head trauma does spasticity occur. Resulting from an impairment of corticospinal pathways, fine and gross motor co-ordination and dexterity are affected. Spasticity is marked by an increased tone associated with hyperreflexia.
- Appearing only months to years following the injury movement disorders are a relatively rare complication. Most frequently, the symptoms are due to damage of the basal ganglia or the nigrostriatal pathways leading, for instance, to dystonia of a hemiplegic limb or choreoathetosis. Tremor may follow even mild traumatic brain injury.
- Severe brain trauma may result in rare cases in the apallic syndrome characterized by a functional disconnection of the cortex and the brainstem. The main symptom is the so-called coma vigile with the patient having a markedly reduced consciousness without content and activities while being awake and reduced to a few basal autonomous functions.

Also, the endocrine system may be affected by traumatic head injury in various ways.

- The involvement of the posterior pituitary may lead either to excessive retention of free water or to diabetes insipidus with inappropriate or insufficient secretion of antidiuretic hormones.
- Damage to the anterior pituitary may result in dysfunction of various target organs. Thus, short stature may result from deficiencies in growth hormone, hypothyroidism from a deficit of thyroid-stimulating hormone, hypogonadism from deficiencies in follicle-stimulating and luteinizing hormone, and hypoadrenalism from adrenocorticotropic hormone deficits.
- Excessive appetite and also anorexia may be the consequence of post-traumatic hypothalamic dysfunction, and precocious puberty may result from dysfunction of the hypothalamic–pituitary axis.

Further sequelae of head and brain injury are evident on the neurocognitive level.

- Depending on the severity of damage, there is a varying degree of impaired intellectual functions, with a decrement between performance and verbal scale

scores in standardized tests. Non-verbal functioning is more closely correlated to severity of injury than are verbal scores. The former is more dependent on speech, dexterity and problem-solving skills whereas verbal skills rely on retrieval of information that may be overlearnt. The impairment of the general level of functioning may manifest as a loss of skills, a failure to make any progress or a slower rate of learning.

- Another very frequent neuro-cognitive sequel is reduced speed of information processing affecting various other neuro-cognitive functions, i.e. language, learning and memory, attention and perceptual and motor tasks. Functionally, the affected children may be slower in everyday life, including school and leisure time activities, where fast and complex information processing is needed.
- Problems of naming and with expressive and written language and a deficit in verbal fluency will point to language and communication skills impairment. Sometimes the impact of the trauma may also result in transient mutism. Depending on the age of the child, grammar and syntax may be affected in pre-schoolers whereas higher-level language skills may become deficient in older children and in adolescents. Persistent problems may include a lack of prosody, slowed rate of speech or articulation deficits.
- Further problems stem from impaired learning and memory leading to slowed down knowledge and skills acquisition processes, forgetfulness and absent-mindedness that can be seen both in the classroom and at home. Verbal memory deficits may result in failure to recall instructions, or failure to remember can lead to the necessity of repetitions.
- Frequently encountered problems include attention deficits and distractibility. Children with head injuries have problems with both sustained and focused attention and are easily distracted by extraneous events going on around them. Keeping them on task at school or engaging them in activities for a long time at home is not an easy task.
- Among perceptual deficits visual–perceptual motor skills may be affected soon after the injury and these deficits may persist over longer periods in severe injuries. These functional difficulties may be the result of partial loss of visual fields or double vision in a few children.
- Owing to the high vulnerability of the frontal lobes to traumatic brain injuries, executive functions frequently are disturbed. These functions comprise basic attention and orientation, working memory, meta-representation skills, semantic representation skills and a monitoring system. They involve the capacity for self-determination, self-direction, self-control and regulation. Deficits in these areas impair limitations on the child's capacity to adapt appropriately to changes in environmental demands.

The various neurocognitive deficits lead to various problems on the educational level.

- A large proportion of children with head injuries do not make normal progress in school, fail a grade or need special education provision due to less capable performance at school.
- Educational achievement in these children may be hampered by specific scholastic skills deficits in terms of reading backwardness or sometimes even dyslexia, problems with written language and/or deficits in arithmetic skills with dyscalculia in the most severe cases.

Finally, there are various important consequences on the psychosocial level.

- In each instance of brain injury the child and adolescent has to cope with the impairment resulting from the trauma. Thus, a process of individual adaptation has to start that is based on the individual resources and pre-morbid personality features. Depending on these qualities, there may be both risk and protective factors that either contribute to the heightened risk of general psychopathology in brain disordered patients or buffer against it.
- Family resources are of similar importance. A lack of support from the family may stem from dysfunctional adaptation. Usually families, and most specifically parents, undergo a process of adaptation that starts from an initial shock phase with limited understanding and potential for goal-directed actions. An intermittent phase is then marked by various feelings of anxiety, guilt, anger and blame and finally a relatively stable phase of adaptation is reached. Dysfunctional processes of adaptation may be marked by insufficient care of the patient and/or his siblings, increased family disagreements, partnership problems or even break-up of the family. These problems may be even more pronounced if there is grief and loss for another family member who has died in the accident, a parent or sibling suffering from post-traumatic stress disorder as a consequence of witnessing the injury or an injured parent or sibling.
- Beyond the family, the wider psychosocial environment with relatives, friends and peers reacts to the handicapping condition of the child and exerts an influence on the psychosocial adaptation of the affected child. The reactions may vary between isolation and rejection on the one hand and integration and support on the other. The individual child and his family may both be affected by those either negative or positive processes.

Differential diagnosis and comorbidity

The main question of differential diagnosis in brain injury is whether or not a given symptom or syndrome may be delineated directly or only indirectly from the brain disorder. There is little doubt that very early signs of delirium that can be observed, for instance, in the emergency department, i.e. loss of consciousness,

agitation and loss of orientation are related directly to the impact on the brain. However, in the case of lacking information on history, it may not be clear whether there is a closed head injury or an infectious disorder or an intoxication or another noxious agent exerting an influence on the brain. These various causes will have to be ruled out by careful physical examination including laboratory assessments and neuroimaging.

Similarly, transient psychotic features may pose the question whether or not symptoms of schizophrenia may also explain the clinical picture. Owing to the similarity of the main features in the two conditions, differentiation may be difficult in the absence of clear anamnestic data. Most commonly, the transient psychotic features in brain disorders are marked by hallucinations that are closer to reality and less bizarre and strange and by more concrete and trivial paranoid symptoms.

In the absence of a clear history and/or the exclusion of other aetiologies the localized symptoms of agnosia, aphasia and apraxia also deserve careful examinations assisted by neuroimaging in order to rule out causes other than injury, i.e. brain tumours or anomalies of brain vessels.

With the dominant manifestations of general psychopathology, there is less of a question of differential diagnosis or co-morbidity than an attempt to differentiate the various factors that may have contributed both separately and in interaction with the brain injury to the clinical symptoms.

Various sequelae of the brain injury on the neurologic and endocrine level that may lead to the development of orthopaedic disorders (e.g. scoliosis, contractures, arthritis) may be viewed as co-morbid disorders.

Diagnostic instruments and assessment

The main source of diagnosis in brain injury is clinical examination by the use of history taking, observation and neurological assessment. Neuroimaging by the use of computed tomography (CT), magnetic resonance imaging (MRI) and electroencephalogram (EEG) is mandatory in order to get a full and comprehensive picture of the brain injury.

Neuropsychiatric examination of acute symptoms in brain injury will take place only in the emergency room and transient symptoms will also be noticeable most probably in intensive care units, where examination has to be performed in collaboration with neuropeadiatricians and neurosurgeons. Continuous and repeated observations within relatively short intervals of time are needed in order to examine the fluctuating course of symptoms.

The main domain of neuropsychiatric assessment are the chronic sequelae of brain injury that have to be followed over years in some cases with a very protracted course. The majority of symptoms are represented by neuropsychological deficits in the areas of intellectual functioning, speed of information processing, language and

Table 1.2. Areas of assessment, interview questions, and neuropsychological tests (adapted from Middleton 2001)

Area of assessment	Interview questions	Neuropsychological tests
1. Intellectual functioning	Loss of skills	Wechsler Intelligence Scale for Children (WISC-III)
	Failure to make any progress	
	Slower rate of learning	Wechsler Primary and Preschool Scales of Intelligence (WIPPS-R)
2. Speed of information processing	Time to carry out everyday living tasks (e.g. getting dressed)	Peg boards, Coding and Symbols (WISC-III)
Motor speed		
Thinking speed	Grasp what is going on around them (e.g. in general conversation)	
	Process information in unpressed situations	
	Gather and express thoughts	
	Respond to questions or requests	
	Carry out motor tasks	
3. Language and communication skills	Ability to initiate conversations	Standardized Speech and Language Tests
Expressive language	Clarity of speech and intonation	Picture Vocabulary Tests
Receptive language	Word finding	Verbal Scale of WISC-III
Word finding	Grammatical structure of speech	
Written language	Ability to express ideas	
	Ability and flexibility to follow general conversation	
	Understanding of jokes, ambiguity, and abstract concepts	
	Writing, reading and spelling	
4. Learning and memory	Forgetting simple instructions	Children's Memory Scale
Visual and verbal memory	Losing belongings	Test of Memory and Learning
Immediate and delayed memory	Forgetting homework	Wide Range Assessment of Memory and Learning
Recall and recognition	Forgetting plans for the next day	
	Forgetting what happened yesterday	Rivermead Behavioural Memory Test
	Forgetting where things have to be put down in the home	
	Need for many repetitions	

5. Attention	Attention span	Attentional task batteries
Vigilance and attention	Distractibility	Continuous performance test (CPT)
Sustained attention	Deficits in organization of tasks	
Divided and focused attention		
6. Visual-perceptual skills	Trips over or bumps into things	Rey–Osterieth Complex Figure
Spatial awareness	Ignores things on one side of the field of vision	Performance Scale WISC-III
Visuo-construction	Has difficulties with writing, drawing or copying	Tests of Visuo-Motor Integration
Visual organization	Has problems doing puzzles or playing with constructional toys	
Visuo-motor integration	Has poor aims in ball games, etc.	
7. Executive functions	Self-organize (for school, clothes, bedroom)	Wisconsin Card Sorting Task
Verbal fluency	Plan homework and other activities	Stroop Test
Working memory: planning and execution	Keep to plans and not to be distracted by peripheral information	Tower of London
Ability to shift set	Initiate activity	
Ability to inhibit responding to peripheral information	Evaluate own work objectively	
8. Educational achievement	Progress in school	Standardized Achievement Test
Reading	Failing a grade	Standardized Scholastic Skills Tests
Spelling	Special education	
Writing	Scholastic skills	
Arithmetic		

communication skills, learning and memory, attention, visual–perceptual skills and executive functions. As a consequence, educational achievement may be hampered considerably.

Assessment of these neuropsychological deficits needs both exploration and testing. Table 1.2 provides an overview of the area of assessment, probes of interview questions and recommendations for neuropsychological tests. For various functions, recommendations for specific tests have to remain rather general when national standardization is needed for individual assessment.

The process of assessing multiple areas of function over years of follow-up must be regarded in a developmental context. With changes of the child's functions in these areas, problems will not only disappear but also will take some time to emerge, e.g. only after months and years. Thus there may not be only remission of symptoms but also a process where the child partially grows into functional deficits.

Aetiology

The causes of brain injury vary with age. The majority of head injuries in infants are caused by falls or abuse. In older children and adolescents traffic accidents, sports and leisure-time activities account for the majority of injuries. Children and adolescents with pre-existing disorders, in particular hyperkinetic disorder and children from disadvantaged psychosocial environments are at increased risk of head injury.

The severity of damage in open head injury is related to the kinetic energy of the penetrating object. Thus, the shot of a gun or rifle results in more damage than a hit with an object. Open head injuries result in more localized damage than closed head injuries. The pathophysiology of closed head injuries and the severity of the damage are related to the translational and rotational forces involved. Contusions most commonly involve the inferior frontal and temporal lobe surfaces. Rotational forces most often affect the subcortical white matter, the upper brainstem, the superior cerebellar peduncle and the basal ganglia by axonal shearing, leading to diffuse damage in these areas. Shearing effects are also observed by the use of neuroimaging in the hippocampus, corpus callosum and at interfaces between the brain and the dura. Injury of the cell membrane and intracellular swelling leads to cerebral oedema.

The neuropsychiatric sequelae may be mediated via organic pathways or via psychosocial pathways. There is a direct link between brain injury and impaired neurocognitive processing. However, brain injury can also lead to various adverse psychosocial consequences. As stated above, it may be assumed that many common psychiatric disorders in brain-injured children result from an interaction of organic and psychosocial risk factors.

Treatment

Clinical management and treatment setting

Depending on the severity of brain injury, the affected children and adolescents require multidisciplinary intervention. Open head injuries will require neurosurgical interventions and pharmacological measures against the accompanying cerebral oedema. Whereas mild head injuries may not require more than psychiatric consultation, moderate to severe forms of head injuries deserve intensive and often long-standing rehabilitation. Starting with the consultation in the emergency room or intensive care unit, the child and adolescent psychiatrist will be part of an interdisciplinary team consisting of neuropaediatricians, neurosurgeons, orthopaedists, psychologists, physiotherapists, speech and language therapists, occupational therapists, nurses and teachers. Frequently, highly specialized rehabilitation units are required for long-term care of brain-injured children.

Within this interdisciplinary team, the child and adolescent psychiatrist are responsible mainly for the promotion of positive psychosocial adaptation of the patient and his family. The measures of intervention include psychotherapy, behaviour modification, family and team counselling and psychopharmacotherapy.

Psychological interventions

Both the patient and his or her family require professional assistance in their process of adaptation. Supportive psychotherapy may be used to help the child coping with the burden of the disorder. Behavioural interventions including contingency management or skills training may assist the process of redeveloping lost skills or provide intervention schedules for the rehabilitation team in specialized units in order to control for behavioural excesses or deficits. Furthermore, educational achievements have to be recalibrated according to the child's current potential. Accordingly, the school has to be informed and counselled in order to adapt the curriculum to the individual needs of the child.

Owing to the above-mentioned problems in adaptation, the family also will need specific professional attention. Family counselling, including clear and honest information about the patient's present status and the problems of clearly defining prognosis, is helpful and will be appreciated by the family. Usually, the family will find it stressful to live with uncertainty so that at least the discussion of fears and worries with the parents can be useful and supportive. Additional information on getting the necessary extra help in school or about local or national support groups in order to have contact with families in a similar life situation are needed and valued by the parents. Family counselling also will allow an additional focus on mental suffering of other family members, problems with the healthy siblings or within

the parents' partnership so that psychotherapeutic and behavioural interventions directed at these problems may be installed.

Psychopharmacotherapy

Various target symptoms can be altered successfully with psychopharmacological interventions. Table 1.3 provides a list of medications, their possible clinical indications and common and less common side effects. With the exception of the stimulants and typical neuroleptics, the safety and efficacy of these medications have not been tested thoroughly for children. However, stimulants, lithium, anticonvulsants, anxiolytics, selective serotonin-reuptake inhibitors, neuroleptics, β-blockers and α-adrenergic agonists are frequently used in acute or chronic rehabilitation of children with traumatic brain injury.

The treating expert needs to be aware of the fact that children with traumatic brain injury may react differently to certain drugs when compared with children without evidence of brain injury. For instance, the responsiveness to stimulants is less favourable than in attention deficit hyperactivity disorder (ADHD) unrelated to trauma. Children with brain injury may also be more sensitive to untoward side effects so that drugs that usually cause drowsiness only may result in somnolence during the day, cognitive blunting and insomnia at night. Similarly, medication-induced anorexia may affect weight gain and growth negatively.

Monitoring and evaluation of treatment

Since the rehabilitation process in children suffering from moderate to severe brain injury may be long-lasting, constant monitoring of the child's development and progress is essential. Besides expert examination of neuropsychiatric symptoms also behavioural and emotional functioning may be assessed by use of standardized parental checklists like the Child Behaviour Checklist (CBCL) or its companion instrument, the Teacher Rating Form (TRF), or the Strengths and Difficulties Questionnaire (SDQ, see Appendix to the chapter on conduct disorders). With more severe manifestations of brain injury and the accompanying intellectual impairment, more specific checklists developed for handicapped and mentally retarded populations may be more appropriate (see chapter on mental retardation).

One of the cornerstones of evaluation of treatment is repeated neuropsychological testing of various domains that may be affected and have been described above. The areas of assessment and recommended tests have been collected in Table 1.2. Furthermore, drug treatment needs careful monitoring of effects and side effects, and behavioural interventions specifically are apt to evaluation given the fact that clearly defined behavioural symptoms and targets are addressed.

Table 1.3. Medication used to treat brain-injured patients (adapted from Guthrie *et al.*, 1999)

Medication	Possible clinical indication	Common side effects	Less common side effects
Stimulants			
Methylphenidate	Inattention	Anorexia	Tics
Dextroamphetamine	Impulsivity	Insomnia/Irritability	Weight loss
	Hyperactivity	Headache/Tachycardia	Depression
			Hypertension
	Aggression w/above	Tremor, weight gain	Nystagmus,
			Acne
Lithium	Mood lability	Polydipsia, polyuria	Hypothyroidism
Tricyclics			
Amitryptiline	Headache	Sleepiness	Cardiac effects
Anticonvulsants			
Valproic acid	Episodic dyscontrol	Drowsiness	Hepatotoxicity
Carbamazepine	Aggressive outbursts	Weight gain	Blood dyscrasias
Gabapentin	Mood lability		
Selective serotonin-reuptake inhibitors			
Fluoxetine	Depression	Insomnia	Akathisia
Sertraline	Obsessive–compulsive symptoms	Agitation	Disinhibition
Paroxetine	Self-mutilation	Anorexia	
Fluvoxamine	Pathologic laughter/crying	Drowsiness	
		Weight gain	
Anxiolytics			
Lorazepam	Agitation	Drowsiness	Tolerance
Clonazepam	Anxiety	Cognitive slowing	Dependence
		Disinhibition	
		Drowsiness	
		Gastrointestinal complaints	
Neuroleptics			
Chlorpromazine	Psychosis	Hyperprolactenemia	Tardive dyskinesia
Haloperidol	Violent outbursts	Extrapyramidal symptoms	Malignant hyperthermia
Risperidone		Anticholinergic symptoms	Agranulocytosis
Olanzapine		Photosensitivity	
β-Blockers			
Propranolol	Aggression w/sympathetic arousal	Bradycardia	Arrhythmias
		Hypotension	Agranulocytosis
		GI disturbances	Bronchospasm
		Depression	
α-Adrenergic agonists			
Clonidine	Hyperactivity	Sedation	Hypotension
Guafacine	Inattention	Nightmares	Anxiety
	Impulsivity	Restlessness	Hallucinations
		Depression	

Problematic issues

Strong ethical problems may arise in a few cases of prolonged apallic syndromes where continuation of intensive care may be put in question by the parents. These situations require counselling in a highly sensitive way by very skilful and reflective doctors and should be embedded in a setting of continuous and intensive psychological care of the entire family.

Outcome

The short- and long-term outcomes strongly depend on the severity of the head injury. A thorough review of studies in mild head injuries that had been published between 1970 and 1995 found no adverse effects on academic and psychosocial outcome nor on neuropsychological outcome at the more extreme tail of the mild injury distribution.

The outcome with moderate to severe brain injury is less favourable. Educational achievement was impaired with GCS scores <13 and duration of coma >20 min in some follow-up studies. Attention deficits may be observed even in children with better scores. Post-traumatic amnesia >1 week has been established as an important cut-off point for lasting memory problems. Impaired dexterity and visual–motor copying have been observed even $2\frac{1}{2}$ years after severe injuries. Severely injured children have also been found to be less socially integrated.

Despite the relatively unfavourable findings of follow-up studies, it should be noted that, even in the moderate to severe injury groups, there is sizeable individual variation and that outcome is also related to environmental and not only to organic factors so that a close-response relationship between severity of the trauma and long-term sequelae does not fully describe a varying picture embedded in a developmental process.

Infectious disorders

Definition and classification

Infectious disorders are important in child psychiatry because they can (a) directly cause and/or trigger psychiatric symptoms, (b) cause permanent brain lesions that may lead indirectly to such symptoms, and (c) change the symptoms of an already existing child psychiatric disorder. Clinically, the most relevant distinction is that between meningitis and encephalitis with additional subclassification according to the class of the infectious agent, i.e. bacterial or viral subtype. These subgroups may be broken down further by the specific infectious agent, e.g. herpes encephalitis, CMV (cytomegalovirus) encephalitis, or HIV encephalitis.

Epidemiology

Prevalence and incidence rates and sex ratio

Infectious disorders of the brain are quite common in childhood. No overall figures can be given. In accordance with their higher vulnerability boys are affected more frequently than girls. The rate at which infectious disorders of the brain lead to major psychiatric disorders in children and adolescents is unknown.

Clinical implications

The more common manifestations of meningitis and encephalitis can have important psychiatric sequelae and cause handicapping conditions. Furthermore, there are some specific, though rare, associations between certain types of infections and psychiatric disorders, e.g. between streptococcal infections and tic disorders or obsessive compulsive disorder (OCD) and between rubella or herpes with autism, autistic-like conditions or mental retardation.

Clinical picture

Main features and symptoms

The clinical presentation of an infectious brain disorder varies considerably interindividually and depending on agent and timing of infection. The pattern of clinical symptoms in brain infections is shown in Table 1.4 with a differentiation between symptoms in the acute state and chronic sequelae.

- In the acute state the presenting symptoms are caused by local central nervous system (CNS) manifestations. Nearly all patients suffering from either meningitis or encephalitis develop fever, nuchal rigidity and altered mental state. The latter is marked by various intensities of altered consciousness, symptoms of delirium, disorders of impulse and, in some instances, even psychotic features. A wide range of neurological features result from the infection. Generalized seizure activity occurring during the acute state of meningitis is not associated with subsequent development of epilepsy, whereas focal seizures tend to have a worse prognosis.
- The chronic sequelae may include various behavioural symptoms and it is not uncommon for the symptoms constellation to be one of fairly typical hyperkinetic disorder, autism or even depression and schizophreniform psychosis. Frequent sequelae include mental retardation and neurological complications. During the several weeks to months following the infection, physical impairments such as communicating hydrocephalus, gait disturbances, incontinence, deafness and blindness may develop. Even the endocrine and the autonomous systems may be affected. In contrast to bacterial meningitis, neurologic sequelae are rare in viral meningitis, whereas they are very common in viral encephalitis.

Table 1.4. Clinical symptoms of brain infections

Acute state
• Headache, nausea, vomiting, nuchal rigidity, fever • Altered consciousness of varying intensity • Delirium including disorientation, agitation, illusions • Disordered impulse including lethargy and overactivity • Psychotic symptoms including hallucinations and changes in affect • Neurological symptoms including Brudzinski's or Kernig's sign, seizures, cranial nerve dysfunction, sensory impairment, paresis, ataxia • Disturbance of sleep–wake cycles
Chronic sequelae
• Behavioural symptoms – Attention deficits – Disorders of impulse – Changes in affect and emotions – Problems in social relating – Aggression • Cognitive impairment • Epilepsy • Neurological impairment including paresis, hypotonia, movement disorders, sensory impairments, speech and language disorder • Physical symptoms including endocrine disorders (growth delay, precocious puberty), autonomous dysfunctions, disturbed sleep–wake cycles

Various types of infectious diseases may be associated with the development of distinct psychiatric disorders.

- Streptococcal infections can lead to, trigger, or exacerbate symptoms that are inseparable from those shown by individuals suffering from tic disorders including Tourette syndrome (see chapter) and obsessive compulsive disorders (see chapter).
- Congenital infections occasionally lead to brain lesions in areas which are believed to represent susceptibility regions for particular neuropsychiatric syndromes. Cytomegalovirus and rubella may be the underlying cause of, or a major risk factor in, certain cases of autistic disorder.
- Also herpes encephalitis has been shown convincingly to lead occasionally to full-blown autism in individuals who have not had any autistic symptoms prior to onset of the infectious disorder.
- Mycoplasma encephalitis has been reported to be associated with the onset of schizophreniform psychosis that, albeit of a transient nature, has persisted for a year or more after the first symptoms emerged.

- Acute infections, such as infectious mononucleosis, or a influenza-like syndrome with protracted course may lead to the chronic fatigue syndrome with severe and chronic fatigue, depression, inability to concentrate, decreased memory and multiple other complaints.
- The main behavioural and cognitive features of human immunodeficiency virus (HIV) infections in children include deterioration in academic progress, subtle neurocognitive deficits in the areas of attention, motor, memory and perception, social withdrawal and increased emotional lability in the early stages. Often, there is a paucity of facial movements, as is found in diseases of the basal ganglia. Other symptoms include those of ADHD and conduct disorders. In the advanced stages of CNS–HIV psychomotor slowing, apathy and neurologic seizure such ataxia and spasticity in the legs are indicative of encephalopathy.
- Owing to persistence in tissue for long periods, infections with *Borrelia burgdorferi* may lead to chronic Lyme disease. The illness is characterized by three stages. In the first stage, the characteristic erythema migrans (bullseye rash) is visible. In the second stage, 1 to 4 months after the tick exposure, aseptic meningitis is the most common manifestation with associated mild encephalitis and further neurological complications. The third stage, developing years to months after the tick, is marked by tertiary neuroborreliosis with severe neurological sequelae. The most common form of chronic Lyme disease is a subacute encephalopathy with focal CNS deficits, motor disorders, seizures, headaches and various neurocognitive deficits. Children tend to have minor complications of shorter duration.

Differential diagnosis and assessment

Careful history taking, neurological and laboratory assessment of the inflammatory and infectious process including blood and cerebrospinal fluid (CSF) tests and neuroimaging are essential in order to establish the correct diagnosis since mental state abnormalities show large variations and insufficient specificity for various disorders. The clue to the underlying cause is often the acute/subacute onset or some associated physical symptoms including eye, ear, spleen, liver and skin anomalies in congenital infections, and fever, headache and other neurological symptoms in postnatal syndromes.

Aetiology

The pathogenesis of acute bacterial meningitis results from a rapid accumulation of granuloctes in the CSF, which is associated with several inflammatory mediators. The enhanced brain–blood barrier (BBB) permeability leads to a number of pathophysiological sequelae including cerebral oedema, increased intracranical pressure and ventriculitis as potential complications. In later stages decreased blood flow

and vascular impairment secondary to inflammation may lead to cerebral ischemia and infarction.

In viral meningitis the pathogenesis involves hematogenous spread to the CNS. Enteroviruses are the most significant causal agents of this, most commonly in the summer and fall months.

Among more than 100 infectious agents causing acute viral encephalitis herpes simplex type 1 is the most common. Other causally related viruses include enteroviruses, arboviruses, Epstein–Barr virus, CMV and rabies. The direct infection of neuronal cells, predominantly of the grey matter, is the most likely pathogenic mechanism with inflammatory reactions playing an additional role.

HIV produces CNS symptoms by migration of infected macrophages from outside the CNS through the BBB into the brain. In addition, microglia, multinucleated giant cells and various neurotoxic factors with a predominance in the basal ganglia are involved. Most of the children are infected intrauterinally, through the mother.

The underlying cause of the psychiatric disorder is either the infectious agent alone (such as is likely to be the case in autism following herpes encephalitis) or an interaction between the brain lesion caused by the agent and some genetic susceptibility (such as in the case of exacerbation of tics in Tourette syndrome). In the case of herpes encephalitis it appears that the frontal and temporal brain lesions caused by the virus are responsible for the neuropsychological deficits that lead on to the syndromal presentation of autism. In Tourette syndrome, the hypothesis is that antibodies against streptococcal agents cross-react with basal ganglia nerve cell tissue to exacerbate the symptoms of tics and obsessive–compulsive behaviours.

Long-term psychosocial adaptation is dependent on various factors. Besides direct effects resulting from the brain disorder, there are various indirect effects including developmental status, coping capacities and resources of the child, support from parents and siblings and assistance from relatives, peers and school. The process of interaction of these various factors may either be helpful in adjusting to the sequelae of the basic disorder or may even aggravate a burdensome illness.

Treatment

Clinical management and treatment setting

In the acute state most children suffering from infectious disorders of the brain will have to be admitted to a paediatric hospital. Close co-operation between a neuropaediatrician and a liaison child psychiatrist may be helpful. More frequently, the child and adolescent psychiatrist will be responsible for the treatment of the chronic behavioural sequelae of the infection of the brain.

Psychological interventions

Chronic behavioural and cognitive sequelae due to the handicapping conditions and severity deserve intensive rehabilitation including measures of psychotherapy, behaviour modifications, speech and language therapy, physiotherapy and special education. A multidisciplinary team approach is mandatory and sometimes only highly specialized rehabilitation centres can meet the requirements. Both the needs of the patient and the family have to be considered if the mental and physical state of the child has changed dramatically from the premorbid development, and a process of major re-adjustment has to be assisted and guided.

Pharmacotherapy

Acute or subacute infections of the brain should be treated according to guidelines for such infections in collaboration with a doctor with expertise in this area. The definite treatment for bacterial meningitis is early administration of antibiotics. No specific antiviral therapy currently is available for treatment of enteroviruses, the most common causal agents of the small group of viral meningitis. The treatment of choice for viral encephalitis is acyclovir and AZT is indicated in HI infections due to its good CNS penetration.

Psychiatric symptoms may well respond to neuroleptics and stimulants according to the indications for these drugs. Interactions with antiepileptic drugs (AED) have to be considered if the patient is receiving AEDs. These interactions are shown in Table 1.9.

Monitoring and evaluation of treatment

Besides continuous physical assessment including neurological and laboratory assessment, behavioural and emotional changes have to be recorded. Psychopathological features should be described carefully. In addition, behavioural features may be evaluated by various informants including parents, therapists of different kind, and special education teachers. Standardized rating scales and questionnaires may be used (e.g. the Child Behaviour Checklist (CBCL) or the Strengths and Difficulties Questionnaire (SDQ)). Neuropsychological testing of various cognitive functions including general intelligence, speech and language, memory, learning, perception and attention is a major cornerstone of the evaluation of treatment. Specific recommendations for neuropsychological assessment are given in Table 1.2 in the section on brain injury.

Information of patients and parents

Psychoeducation and guidance of patients and parents is of high importance, given the fact that the chronic sequelae of infectious brain disorders may be extremely burdensome. Especially the high mortality rate of meningitis caused by arboviruses

and the increased rate of severe handicaps in the survivors makes intensive counselling and guidance of the parents mandatory. Close co-operation between the child psychiatrist and the rehabilitation team is essential in order to provide accurate information and assistance to the patient and the parents. Reactions of grief and depression should be identified and lead to the installation of appropriate treatment.

Outcome

The outcome of infectious brain disorders varies a lot. Congenital infectious syndromes and herpes encephalitis appear to carry a gloomy prognosis unless treatment can be started early and brain tissue destruction be minimized. Mycoplasma encephalitis may run a protracted course (over 6 to 18 months) but neuropsychiatric symptoms may eventually resolve. The outcome for most cases of viral encephalitis is poor. Treatment of streptococcal disorders in connection with tics eventually may prove to be a fruitful avenue for improving the outcome in some cases of tic disorders, but the evidence to date is too limited to draw any definitive conclusions. Patients with focal epilepsy as a result of bacterial meningitis tend to have a worse prognosis.

Cerebral palsy

Definition and classification

Cerebral palsy is a heterogeneous group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain originating in the early stages of its development. Often, the motor disabilities are accompanied by other clinical manifestations of brain disorders, including epilepsy, learning problems, sensory impairments, and not least, behavioural and emotional problems.

The type and distribution of spasticity forms the basis of subclassification of cerebral palsy. In spastic hemiplegia just one side of the body is affected whereas in spastic diplegia both legs are affected to a severe extent and both arms to a mild extent. Quadriplegia or tetraplegia denotes the severe spastic affection of all four limbs. Less frequently, ataxia, or dyskinesia (athetosis) dominate the motor problems of cerebral palsy.

Epidemiology

Prevalence rates

The prevalence rates for cerebral palsy reported in the world literature since 1990 are quite stable at approximately 0.2–0.25 per cent. The percentages of cerebral palsy

attributed to prenatal risk factor vary between 8 and 43%. This variation may be due to differences in defining exposure to risks. Prevalence rates of the subtypes of cerebral palsy also differ considerably. An English register study of more than 1000 children with cerebral palsy found 35 per cent to have quadriplegia, 31 per cent to have hemiplegia and 22 per cent to have diplegia. A further 5–10 per cent have ataxia (including atactic diplegia) and some variants are considered unclassifiable.

Implications for clinical practice

With 2 in 1000 children and adolescents, cerebral palsy is not a frequent though very handicapping condition and is the single largest course of severe physical disability in childhood.

Clinical picture

Main features and symptoms

Epidemiological and clinical studies of children with cerebral palsy have shown the following characteristics with regard to psychiatric disorders.

- The prevalence of psychiatric problems is higher than in normal children amounting to half and more of the population of children with cerebral palsy. It is increased significantly if two common accompanying factors of cerebral palsy are there, namely, mental retardation and specific reading retardation. This tends to be true also for accompanying seizures. All three factors indicate that there is a relation between organic severity and rate of psychiatric problems.
- In general, there is no distinctive association of cerebral palsy with any type of psychiatric disorder except hyperkinetic disorders. In hemiplegia, with its relatively mild physical disability and little or no intellectual impairment, around half of all affected children have at least one psychiatric disorder. Among these children conduct and emotional disorders each affect one-quarter, hyperkinetic disorders another 10 per cent, and autism around 3 per cent.
- The most common emotional disorders in children with hemiplegia are specific phobias, separation anxiety and generalized anxiety. Some patients also develop depressive disorders. Also, frequently these children show markedly defiant and negativistic behaviour also leading in some cases to marked aggression. A considerably larger number of children with hemiplegia than healthy children show some autistic features. Also in this subgroup IQ, which is associated with degree of neurological impairment, is the best predictor of psychiatric problems. The latter are again more common if specific learning difficulties are present.
- Children with hemiplegia encounter an increased risk of social isolation due to teasing, victimization, marginalization and lack of friendships. These problems

may be due in part to constitutional problems with social understanding and relatedness.
- Psychiatric and behavioural problems are often very persistent and may be seen for many years.

Aetiology

The causes of cerebral palsy are largely unknown. However, some associations are known, namely, between cerebrovascular accidents and hemiplegia, between a variety of generalized brain insults and quadriplegia, and between severe rhesus disease and athetosis. There is a hereditary component in ataxia and prematurity is an important risk factor for various forms of cerebral palsy, most notably for diplegia.

The determinants of psychiatric disorders are multifactorial. Certainly, the underlying brain disorder implies a heightened vulnerability with limited cognitive potential (in many cases) and a lack of personal and psychosocial resources adding to the risk of developing psychiatric problems. Among the latter, social rejection and isolation among peers and within the family are powerful sources of individual maladjustment. In hemiplegia, some degree of bilateral involvement, the presence of seizures and lower IQ have been shown to be associated with a higher rate of psychiatric disorder.

Treatment

The basic condition of cerebral palsy requires competent neurological and orthopaedic treatment with physiotherapy being the major cornerstone. Additional mental retardation and/or sensory impairment may imply that the child with cerebral palsy may not be educated in the mainstream schooling system but, rather, need special education.

With the high probability of developing additional psychiatric problems, there is not only the need for careful monitoring and assessment but also for early intervention or even for the development of preventive approaches. In principle, all sorts of psychological interventions including brief and supportive individual psychotherapy, behavioural interventions, counselling and psycho-education are applicable. Since the families are subjected to the burden of sometimes very distressing and enduring care, a family approach in counselling and guiding is indicated. All these efforts should be started early and be continued over years.

Some of the psychiatric problems such as the frequent hyperkinetic disorders may require standard psychopharmacological treatment. There is a lack of well-controlled studies on the efficacy and effectiveness of drug treatment in children with cerebral palsy. Thus, very careful titration processes and monitoring of effects and side effects are mandatory. If there is co-existent epilepsy, the application of

psychotropic medication may lead to interactions with antiepileptic drugs. Potential interactions are outlined in Table 1.9. However, improved control of seizures also may in some instances improve behaviour and cognitive status. Similarly, stimulant medication can result in improved learning if there is a hyperkinetic disorder, and the application of SSRI can help with depression, obsession and compulsion.

Outcome

Cerebral palsy itself is a chronic condition, marked by persistent physical disability of interindividually varying degree. Concomitant mental retardation and sensory impairment may have an additional impact on the course of the disorder. However, it has to be emphasized that many affected individuals experience a satisfying and productive lifestyle with normal psychosocial adaptation.

There is a very limited body of empirical research on the longitudinal stability of psychiatric problems in children suffering from cerebral palsy. A recent British study of a representative sample of children with hemiplegia found a remarkable stability of psychiatric problems in these children. After a follow-up period of 4 years, 70 per cent of the former psychiatric cases were still fulfilling criteria of psychopathology. In the pre-school years, extending symptoms were predictive of later conduct and hyperactivity problems, and in the school years, hyperactivity was particularly predictive of continuing psychiatric problems. The continuity of psychiatric problems was mainly dependent on the severity and the type of the initial psychiatric problems, whereas neurological, cognitive, demographic and family factors did not contribute much to the persistence of the problems.

Despite the high rate of psychiatric disorders in childhood, hemiplegia does not seem to lead to a particularly raised frequency of adult psychiatric disorders. Some childhood problems may persist as maladaptive personality traits and rather restricted lifestyles with limited social activities and professional careers.

Epilepsy

Definition and classification

Epilepsy is a chronic condition of recurrent unprovoked seizures. The latter can be defined as sudden, involuntary, transient alterations in cerebral function due to abnormal discharge of neurones.

The sub-classification of the various types of epilepsy by the International League Against Epilepsy (ILAE) is based on three factors, namely: (a) clinical seizure manifestations, which define the seizure, (b) electroencephalogram (EEG) ictal patterns, and (c) EEG interictal pattern. According to Table 1.5 either partial or generalized and, in addition, unclassified seizures are differentiated. A partial seizure begins in

Table 1.5. Classification of epilepsy according to the International League Against Epilepsy (ILAE)

I. Partial (focal, local) seizures
Definition: The first clinical and/or EEG changes indicate initial involvement of part of one hemisphere.
Partial seizures are classified on the basis of an impairment of consciousness
A. Simple partial seizures (no impairment of consciousness)
B. Complex partial seizures (with impairment of consciousness)
C. Partial seizures with secondary generalization
II. Generalized seizures
Definition: The first clinical and/or EEG changes indicate initial involvement of both hemispheres.
Consciousness is impaired
A. Absence/atypical absence
B. Myoclonic
C. Clonic
D. Tonic
E. Tonic–clonic
F. Clonic–tonic–clonic
G. Atonic

one area or has a focus, whereas a generalized seizure has an initial bihemispheric involvement both clinically and on EEG. Unclassified seizures include those with insufficient information available and neonatal seizures.

Epidemiology

Prevalence rates

Epilepsy in terms of recurrent seizures affects 0.5–1 per cent of the population and up to 10 per cent of individuals have at least one affable seizure at some time in their life. Three-quarters of all epilepsies originate before 20 years of age. By the same age, 5 per cent of all children and adolescents will have experienced a seizure but fewer than one in five of these will develop epilepsy. Within 5 years of seizure onset, 80 per cent of children will be expected to be seizure free. Approximately 2–5 per cent of children will have febrile seizures.

A recent nationwide British study found a prevalence rate for psychiatric disorders of 37 per cent, with a rate of a 56 per cent for complicated epilepsy and 26 per cent for uncomplicated epilepsy. Scandinavian studies suggest that, if epilepsy is combined with mental retardation, then the risk of psychiatric disorder is higher still.

Implications for clinical practice

Epilepsy is a very common disorder in children and adolescents. The rate of non-recurrent afebrile and febrile seizures without progression into epilepsy is much higher. The majority of children and adolescents with epilepsy will become seizure free. However, a sizeable proportion of patients remain with a severe and handicapping condition. The prevalence rate for accompanying psychiatric disorders is elevated significantly and asks for additional provision of mental health services.

Clinical picture

Main feature and symptoms

A full description of the clinical symptomology of the epilepsies is beyond the scope of this chapter. This section will concentrate on (a) epileptic syndromes with psychiatric manifestations and (b) a more general discussion of the association between epilepsy and mental functioning.

Epileptic syndromes with psychiatric manifestations

Certain epileptic syndromes have significant psychiatric or neurobehavioural problems. These include temporal lobe epilepsy (complex partial seizures), infantile spasms, the Lennox–Gastaut syndrome, the Landau–Kleffner syndrome (LKS), and the syndrome of continuous spike-waves during slow-wave sleep (CSWS) also known as Electric Status Epilepticus during Slow sleep (ESES).

- The large majority of patients with temporal lobe epilepsy marked by odd movements, postures, dreamy states, prolonged absences, automatisms and vocalizations show psychiatric disorders. There are specific associations with hyperkinetic disorders and excessive rage outbursts. The risk for a schizophrenia-like psychosis in later life is elevated. Complex partial seizures are one of the most common epileptic syndromes in autism.
- There is a strong association between infantile spasms characterized by brief jack-knife spasms and hypsarrhythmia on the EEG, on the one hand, and mental retardation on the other since many cases have their origin in tuberous sclerosis or a brain malformation. The most common psychiatric disorders are hyperkinetic disorders and autism.
- In the Lennox–Gastaut syndrome, which presents in young children with frequent myoclonic, tonic and atonic seizures (stare, jerk and fall seizures), the great majority of affected children has severe mental retardation. In prolonged episodes of minor states the child is socially unresponsive, aggressive, less articulate and may show minor twisting of hands and face.
- The Landau–Kleffner syndrome is a rare epileptic syndrome characterized by language regression (usually associated with verbal auditory agnosia, and sometimes

referred to as acquired aphasia) and an abnormal EEG/seizures. Hyperactivity is common as are autistic features. The classical LKS occurs in a previously normal child usually in the 3–7-year-old age range. LKS variants include children without clinical seizures but with abnormal EEGs, children with autism with language regression and abnormal EEGs, and children with congenital aphasias, also called developmental language disorders with epileptiform EEGs.

- The syndrome of CSWS is characterized by spike and wave activity that occurs during at least 85 per cent of slow-wave sleep and commonly is accompanied by infrequent seizures. The affected children had been normally developing prior to onset usually between 6 and 10 years and experience regression in mental, language, and behavioural functions. Main psychiatric manifestations include hyperkinetic features, aggressiveness, affective lability, transient psychosis and regressive behaviour features. LKS and CSWS sometimes are considered to exist on the same spectrum of epileptic behaviour syndromes.

The association between epilepsy and mental functioning

As outlined above in the section on epidemiology, there is a significantly heightened rate of general psychopathology in children suffering from epilepsy. Overall, the most frequently observed psychiatric disorders are hyperkinetic disorders, autism spectrum disorders and mental retardation. There is a clear association between complicated epilepsy, as indicated by additional neurological abnormalities, and further increase of psychiatric morbidity.

The traditional concept of an epileptic personality has been discredited and replaced by more detailed analyses of behaviour problems and personality features. The latter have found that dependency, suicidal behaviour, an external rather than an internal locus of control, lack of self-esteem and feelings of anxiety and fears are more common among children with epilepsy.

The term paradoxical or forced normalization, denotes a specific phenomenon of an abrupt worsening of behaviour in patients with epilepsy when seizures are controlled and the EEG improves. The behaviours do not only include symptoms of psychosis but also dyslexia and affective disorders, histrionic features and hypochondrial symptoms. Usually, paradoxical normalization occurs with a change in anticonvulsant drugs. It is more frequent in adults but may also be observed in children.

Whereas most children with uncomplicated epilepsy show cognitive functioning in the normal range of intelligence, the IQ is lowered in complicated epilepsy. A high proportion of children with epilepsy show mental retardation and, the greater the degree of pro-existing mental retardation, the higher the risk of developing epilepsy. Only in a small number of children intellectual functioning is deteriorating as a result of the disorder and/or deficits in appropriate care.

However, there are several ways in which epilepsy can create learning problems of various size. An extreme is prolonged, brain-damaging status epilepticus. Via state-dependent learning also a non-convulsive status epilepticus accompanied by generalized spike-wave discharges or frequent absence seizures may contribute to disability. Frequent epileptiform discharges without convulsions may also have an impact in terms of so-called transitory cognitive impairment, leading to frustrating experiences, which may affect learning. Similar effects may stem from frequent local or frequent hemispheric discharges, e.g. on the basis of congenital brain malformations.

In addition, there also may be underestimated post-ictal effects in terms of state-dependent learning disability if, in very frequent seizures, there is not sufficient time for the affected child to recover from one seizure before another. The same principle of post-ictal effects applies also to frequent nocturnal seizures or CSWS.

Finally, antiepileptic drug treatment also may contribute to impairment of cognitive functioning. Sedative effects of the wrong type of anticonvulsant may affect learning adversely. Drugs such as phenobarbital, the benzodiazepines and vigabatrin may show a paradoxical effect in children by mimicking the symptoms of hyperkinetic disorders. Children with autism and epilepsy often show particularly adverse behavioural toxicity during treatment with clonazepam and other benzodiazepines.

Unfortunately, research on the cognitive adverse effects of chronic antiepileptic drug treatment in general is plagued by a multitude of methodological shortcomings, so that currently no satisfactory statement can be given. The overall conclusion is that, in relation to cognition, some children benefit significantly, a middle group does not appear to be affected and a third group is made worse by the antiepileptic medication.

A more detailed analyses of the determinants of both cognitive impairment and behavioural problems will be provided in the section on aetiology below.

Differential diagnosis and co-morbidity

From a psychiatric point of view, there is a need to differentiate various disorders from epilepsy as shown in Table 1.6. These disorders are also called non-epileptic paroxysmal events or non-epileptic periodic events and need to be considered in patients with intractable epilepsy or persistently normal EEG. However, it should be kept in mind that many patients with epilepsy have a normal EEG and diagnosis of epilepsy does not require an abnormal EEG. Furthermore, the high rate of psychopathology in epilepsy does also allow the possibility that epilepsy and one of these disorders may coincide in terms of co-morbidity. Medical history and careful description of symptoms are more important than an EEG in diagnosis and differential diagnosis in epilepsy.

Table 1.6. Differential diagnosis of epilepsy

- Pseudoseizures
- Movement disorders
  - Tics and Tourette syndrome
- Sleep disorders
  - Night terrors
  - Somnambulism
  - Hypersomnia
  - Narcolepsy
- Panic attacks
- Rage attacks or episodic dyscontrol
- Migraine
- Munchhausen's syndrome (by proxy)

The following implications derived from differential diagnosis are outlined in Table 1.7.

- Pseudo-seizures may combine with true seizures. Indicators of pseudo-seizures include situations when the child is being observed rather than staying alone, a gradual rather than a sudden onset, uncontrolled flailing paroxysms rather than true clonus, theatrical movements accompanied by loud shouting or screaming, avoidance of painful stimuli and injury during an attack, sudden offset with an immediate return to an alert and responsive state and lack of paroxysmal discharges on the EEG during an episode.
- Tics and especially ocular tics, including Tourette syndrome, may sometimes evoke the question whether myoclonic jerk are present. Again, the lack of paroxysmal discharges in the EEG will allow differential diagnosis.
- Various sleep disorders including night terrors, somnambulism, hypersomnia and narcolepsy will have to be excluded not only by the different phenomenological features but also by the lack of specific epileptiform activity in the EEG.
- Panic attacks with their sudden onset of fear occur usually without alteration of awareness but can be confused with simple partial seizures because the latter can also be associated with a sensation of panic or fear.
- Rage attacks or episodic dyscontrol represent episodes of severe conduct or violent behaviour. Usually, they are associated with some provocation or a precipitating event, although some may develop spontaneously. Rage attacks may be more common in children and adolescents with epilepsy, hyperactivity or personality disorder. During an attack, the EEG does not show epileptiform activity. However, due to the underlying disorder, the inter-ictal EEG may be abnormal.

- Migraine, especially when accompanied by benign paroxysmal vertigo, visual hallucinations, olfactory auras, déjà vu, acute confusional states or transient global amnesia may be diagnosed erroneously as epilepsy. The lack of hypersynchronous epileptic discharges on the EEG, again, will allow distinction.
- Factitious creating or false reporting of symptoms as in Munchhausen's syndrome when the patient originates the symptoms or in Munchhausen's syndrome by proxy, with a parent creating the symptoms in the child, represent very rare instances of differential diagnosis of epilepsy.

Diagnostic instruments and assessment

The main source of diagnosis in epilepsy is careful history taking. Thus, the clinical interview is the main diagnostic instrument. Neuroimaging, especially MRI and EEG may assist the diagnostic process, but may be normal. However, neuroimaging may be very helpful for excluding underlying causes for the seizures, such as a tumour. Observation and description of the event determine classification of the seizure type.

An inter-ictal EEG may show signs of an underlying epileptic tendency, which is present in many patients with epilepsy. However, many patients with epilepsy also show a normal EEG, most particularly between seizures. Thus, a normal EEG does not exclude the diagnosis of epilepsy.

In addition to the basic neurologic examination, the neuropsychiatric assessment is based primarily on the clinical interview and requires expertise in general psychopathology in children and adolescents. Information by caretakers and other educators may be obtained either by interview or standardized checklists, allowing for the screening of a large variety of behavioural abnormalities. The latter include the Child Behaviour Checklist (CBCL) for parents, the Teacher Rating Form (TRF) or the Strengths and Difficulties Questionnaire (SDQ, see Appendix to chapter on conduct disorders). Various more specific checklists for the handicapped are available and may be helpful specifically in identifying problems due to the frequent accompanying mental retardation (see chapter on Mental Retardation).

Both the epilepsy itself and the evaluation of drug treatment in terms of effects and side effects require careful and continuous monitoring. Neuropsychological testing should be used in order to assist this process. Recommendations can be found in Table 1.2, indicating the areas of assessment, related clinical questions and selected neuropsychological tests.

Aetiology

The causes of the epilepsy itself will be discussed only briefly in this chapter. Clearly, hereditary and/or brain-related factors are involved. However, in children and adolescents, as in adults, the largest group of approximately two-thirds represent

idiopathic or cryptogenic cause, 20 per cent have congenital causes, 5 per cent result from trauma, 4 per cent from infection, 2 per cent have neoplastic and 1 per cent show degenerative causes. Age at onset is important insofar as perinatal injury, metabolic disorder or congenital malformations are more common causes in the young age, whereas vascular diseases or tumours are more likely to have an impact in adults. Recently, a genetic source has been found for some types of epilepsy, e.g. an association of juvenile myoclonic epilepsy to chromosome 6 or autosomal dominant nocturnal frontal lobe epilepsy to chromosome 20.

The mechanisms involved in the psychopathology of epilepsy are complicated and need to consider at least three main categories of risk factors and their interaction: (a) brain-related factors – the epilepsy itself and further neurological factors, (b) non-brain-related factors – chronic illness, developmental and subject variables, and (c) treatment-related factors – antiepileptic treatment, surgery and quality of care.

Among the brain-related factors it is, first, the epilepsy itself that has a strong impact on psychopathology. The peri-ictal disturbances of prodome, aura and automatism each present with behavioural phenomena including disturbed mood, feelings of anxiety and symptoms of conduct disorder. In the immediate post-ictal phase confusion, tiredness, irritability, depression and even paranoid states may manifest themselves. Among the inter-ictal disturbances there is the above-mentioned reciprocal phenomenon of behavioural abnormalities and epilepsy that goes along with the paradoxical normalization in the EEG. Other previously mentioned epilepsy-related factors include focal temporal or frontal discharges, frequent absence seizures or non-convulsive status epilepticus that have a direct impact on behaviour by producing various symptoms such as socially withdrawn behaviour or attention deficit disorder.

Further epilepsy-related factors include age at onset and chronicity. The aetiological significance of early onset and long duration of epilepsy may be at least partly due to the third factor of mental retardation because infantile spasms and the Lennox–Gastaut syndrome account largely for this association. High seizure frequency and poor seizure control may be entangled similarly with other factors, such as brain damage, so that the effect on cognitive and behavioural functioning may not be direct. Also the relationship between seizure type and psychopathology and intelligence may be quite inconclusive due to the fact that most studies have concentrated on TLE or primary generalized epilepsies.

In addition to the epilepsy itself, other factors reflecting the associated brain damage or dysfunction play a major role. This may be independent of the epilepsy or be caused by the epilepsy. Injury of the frontal lobe can result in specific neurocognitive dysfunction, or damage to the dominant temporal lobe may result in speech and language disorder. Mediated effects from the brain damage may also

become effective by rendering the child more liable to stresses and strains that may impair the development.

Various non-brain-related factors contribute to the heightened risk of developing psychiatric disorders in epilepsy. As in other chronic illnesses, epilepsy poses a major challenge to the resources of the individual and the family. Deficient coping, parental psychopathology, disturbed family relationships, problem with the healthy siblings or teasing and social isolation outside the family may each have an additional detrimental effect on psychosocial adaptation of the child with epilepsy. The interactive association between epilepsy and these factors is evident in as much as the latter can be both the cause and the consequence of the former.

Finally, treatment-related factors can have an impact on the development of behavioural and cognitive abnormalities. The need for continuous antiepileptic drug treatment renders the child with epilepsy particularly vulnerable to side effects. Among the older and well-established drugs, phenobarbital and benzodiazepines may cause sedation, irritability and disruptive behaviour disturbances mimicking hyperkinetic disorder. Gabapentin can exacerbate pre-existing hyperactivity and learning problems and topiramate has been associated with general slowing of cognition and behaviour. Cognitive and behavioural side effects of polytherapy, the often necessary combination of different drugs may be underestimated and brain surgery may occasionally result in worsening rather than improvement of the behaviour. Deficient quality of care and lack of compliance may also add to abnormal behavioural functioning in the child with epilepsy.

Treatment

Clinical management and treatment setting

The appropriate treatment of children and adolescents with epilepsy requires special expertise. A multidisciplinary team of neuropaediatricians, child and adolescent psychiatrists, and psychologists with specialization in child neuropsychology and behavioural interventions should join forces for the basic care of a patient with epilepsy. Depending on indication, this team should be supplemented by the neurosurgeon.

Support should also come from various experts dealing with varying degrees of disability, i.e. from physiotherapists, speech and language therapists, occupational therapists and special education teachers. Many patients with uncomplicated epilepsy can be treated in ordinary services or practices, whereas the more complicated cases need to be continuously cared for by specialized units in the tertiary sector of services.

Drug treatment plays a major role in the core of epilepsy. Due to the protracted and often chronic course, continuous counselling of the patient and the family and

Table 1.7. Efficacy of standard antiepileptic drugs, according to seizure type (Thiele *et al.*, 1999)

	Seizure type					
Drug	GTC	Absence	Focal/II general	Tonic	Atonic	Myoclonic*
Phenobarbital	+	–	+	+	+	+
Phenytoin	+	–	+	+	–	–
Carbamazepine	+	–	+	–	–	–
Valproic acid	+	+	+	+	+	+
Ethosuximide	–	+	–	–	–	–
Trimethadione	–	+	–	–	–	–
Primidone	+	–	+	+	+	+
Ethotoin	+	–	+	+	–	–
Methsuximide	+	+	+	+	+	–
Benzodiazepines, especially clonazepam	+	+	+	+	+	+
Felbamate	+	+	+	+	+	+
Gabapentin	–	–	+	–	–	–
Lamotrigene	+	+	+	+	+	+
Topiramate	+	+	+	+	+	+
Tiagabine	–	–	+	–	–	–

GTC = generalized tonic clonic seizures; + = efficacious; – = not efficacious.

*Infantile spasms are a certain type of myoclonic, tonic, or mixed type of seizure. Medications used for these include adrenocorticotropic hormone (ACTH), vigabatrin, valproate, benzodiazepines and perhaps topiramate or tiagabine.

various psychosocial interventions are warranted. The specific psychiatric problems of this clientele make both psychological and psychopharmacological treatment mandatory in a large proportion of affected patients A team approach in delivering the various interventions should be followed.

Psychological interventions

Besides prevention of seizures, the major aims of treatment include compliance with treatment strategies, prevention of additional disability due to behavioural problems, psychosocial adaptation to the disorder and social integration of the child. The major forms of intervention in order to meet these goals include individual and family counselling, behaviour modification with the child in his or her natural environments (i.e. home, school, outdoor activities), supportive psychotherapy for emotional and adaptive problems, individual or couple therapy for parents with emotional and/or relationship problems, and family therapy if indicated for the entire family. Besides the child and adolescent psychiatrist, various other therapists may share this complex task.

Table 1.8. Doses, pharmacokinetics and side effects of anticonvulsant drugs (Thiele *et al.*, 1999)

Anticonvulsant	Dose (mg/kg/day)	Therapeutic range		Half-life (hours)	Side effects
Phenobarbital	5	10–30;	15–40	40–75	Sedation, irritability, learning and sleep problems, hyperactivity, ataxia
Primidone	10–25	5–12		5–11	Sedation, nausea, vomiting, diplopia, dizziness, ataxia
Phenytoin	5	10–20		5–18	Gingival hyperplasia, hirsutism, cognitive dysfunction
Carbamazepine	20–30	4–12		8–25	Diplopia, blurred vision, ataxia
Valproate	30–40	50–100		12	Nausea, vomiting, anorexia, weight gain, hepatotoxicity, alopecia, pancreatopathy thrombocytopenia
Ethosuximide	25	50–100		24–36	Nausea, vomiting, anorexia, headaches
Felbamate	45–60	50–100		16–23	Anorexia, weight loss, insomnia, aplastic anaemia, liver failure
Gabapentin	45–60	2–10	(20)	6	Fatigue, weight gain, behavioural problems, disinhibition
Lamotrigene	5–10	2–10	(20)	12–25	Rashes, Stevens–Johnson syndrome, TEN
Topiramate	5–24	–		20	Lethargy, weight loss, anorexia, ataxia, mood disorders
Tiagabine	–	–		5–8	Dizziness, lethargy, anxiety, tumour, concentration problems

–, not established.

Pharmacotherapy

Antiepileptic drug treatment

Only recurring seizures according to the definition of epilepsy or the presence of multiple risk factors for the re-occurrence of seizures are clear indications for antiepileptic drug (AED) treatment. Seizure type, seizure activity and the efficacy and side-effect profile of a drug determine the choice of drug. The efficacy of the various AEDs is documented in Table 1.7.

First-line AEDs include carbamazepine, ethosuximide, phenobarbital, phenytoin, valproic acid and clonazepam. Non-responders to these drugs may be treated with second-line AEDs including chlorazepate, methuximide, acetazolamide and ethotoin. Recently newly developed AEDs include felbamate, gabapentin, lamotrigene, topiramate, tiagabine and vigabatrin. Doses for the most frequently used AEDs, together with pharmacokinetics and side effects are shown in Table 1.8.

Treatment should always be started with a single AED and be titrated slowly. If the single AED is not effective, a second is to be substituted with maintenance of the dosage of the first drug until seizures are controlled and slow discontinuation thereafter. If possible, polytherapy should be avoided due to the risk of drug interactions, more side effects, higher costs and lower compliance.

Alternative therapies include the ketogenic diet, which is composed of high-fat, low-carbohydrate and low-protein food. This intervention has been proved to lead to a significant reduction of seizure activity in children with difficult-to-control seizures. Side effects include weight loss, dehydration, acidosis, abdominal pain and lethargy. Surgical implantation of a vagel nerve stimulator may also be a very effective method of controlling seizures.

Psychopharmacotherapy for associated psychiatric disorders

The reluctance to use psychotropic medication in children with epilepsy is not in the best interests of these children. Indications can be divided broadly into (a) autism spectrum and developmental disorders, (b) disruptive disorders including hyperkinetic disorders and conduct disorders, (c) internalizing disorders including affective and anxiety disorders and (d) psychosis.

Psychotropic medication can have pharmacodynamic and pharmacokinetic interactions with AEDs that need special consideration. The most common pharmacodynamic effect is additive sedation. Pharmacokinetically increased or decreased plasma levels of both substances can result. The most common known and theoretic interactions between psychotropics and AEDs are shown in Table 1.9. Starting with low doses, adding slowly, and monitoring plasma levels and toxicity will contribute to safe use of psychotropics in children with epilepsy and control for lowering of the seizure threshold, which can result from psychotropics.

Treatment of the main psychiatric disorders in children with epilepsy, with an emphasis on psychotropic medication, will now be summarized.

- Autism spectrum disorders or severe developmental disorders predominantly require behavioural interventions and special education. However, in some instances (e.g. severe obsessionality interfering with functioning) neuroleptics or even serotonin re-uptake inhibitors (SRI) with cautious dose titration may be indicated.
- Hyperkinetic disorder (attention deficit hyperactivity disorders) can be treated effectively and safely with stimulants. The traditional concern that stimulants could result in more seizures is no longer valid. However, for children still having seizures while receiving AED, more caution is warranted. If stimulants fail, clonidine or guanfacine can be used cautiously. Tricyclic antidepressants have a pronounced effect on seizure threshold and should thus be avoided or

Table 1.9. Pharmacodynamic interactions of psychotropic medications with antiepileptic drugs (adapted from Thiele *et al.*, 1999)

Psychotropic class	Specific compounds	Interactions
Stimulants	Methylphenidate, dextroamphetamine, adderall	Use cautiously with active seizure disorders; methylphenidate infrequently raises phenytoin, primidone and phenobarbitol levels Increased risk of hepatotoxicity when combined with hepatically metabolized AEDs
α-Adrenergic agonists	Clonidine, guanfacine	Variable effects on seizure threshold, usually benign
Antidepressants		
Selective serotonin re-uptake inhibitors	Fluoxetine	Increased carbamazepine, valproate, phenytoin, mephenytoin, ethosuxicimide, trimethadione, benzodiazepine and methsuximide levels; decreased fluoxetine levels with phenobarbitol, carbamazepine
	Paroxetine	No interactions found
	Fluvoxamine	Increased carbamazepine, phenytoin, mephenytoin, benzodiazepine, methsuximide levels
	Sertraline	Increased lamotrigine due to inhibition of glucoronidation; increased phenytoin and diazepam levels
	Citalopram	Decreased citalopram levels with carbamazepine, phenobarbitol, primidone
Unique compounds	Nefazadone	Increased carbamazepine, ethosuximide, trimethadione and benzodiazepine levels; decreased nefazadone levels with carbamazepine phenobarbitol, primidone
	Trazodone	Decreased trazodone levels with phenobarbitol, carbamazepine, primidone, case report of phenytoin toxicity with trazodone
	Mirtazepine, venlafaxine, bupropion, buspirone	Decreased levels with phenobarbitol, carbamazepine, primidone. Decreased buspirone plasma levels with phenytoin and carbamazepine
Tricyclics	Imipramine	Increase in carbamazepine levels; decreased imipramine levels with phenobarbitol, carbamazepine, primidone
	Nortriptyline	Decreased nortriptyline levels with phenobarbital; increased levels with valproate
	Desipramine	Decreased desipramine levels with phenobarbitol, carbamazepine
	Amitriptyline, clomipramine	Decreased tricyclic antidepressant levels with phenobarbitol, carbamazepine, primidone; increased tricyclic antidepressant levels with valproate
Mood stabilizers	Lithium	Case reports of neurotoxicity at therapeutic lithium levels with carbamazepine co-administration

(*cont.*)

Table 1.9. (*cont.*)

Psychotropic class	Specific compounds	Interactions
	Valproate	Increases levels of lamotrigine, phenobarbitol, diazepam, lorazepam, carbamazepine epoxide, primidone; displaces phenytoin and carbamazepine from protein binding sites; phenytoin decreases and felbamate increases valproate levels; uncommonly can produce absence seizures when combined with clonazepam
	Carbamazepine	Carbamazepine decreases levels of valproate, clonazepam, ethosuximide, lamotrigine, topiramate, tiagabine, primidone, antidepressants, neuroleptics; in combination with felbamate or lamotrigine increased carbamazepine epoxide and variable carbamazepine level effects; reports of ataxia and blurred vision when combined with lamotrigine; increased risk of neurotoxicity with lithium; decreased carbamazepine levels with phenobarbitol, phenytoin, primidone; variable effects on phenytoin levels with carbamazepine
Benzodiazepines	Clonazepam	Increased levels of primidone. Variable effects on phenytoin and carbamazepine levels; additive sedation with other sedating agents
	Alprazolam, lorazepam, zolpidem	Additive sedation with other sedating agents
	Chlorpromazine	Increased phenytoin and valproate levels, theoretical increase in lamotrigine levels
Atypical neuroleptics	Risperidone	Increase in extrapyramamidal symptoms with phenytoin
	Olanzepine	No interactions found
	Clozapine	Decrease in clozapine with carbamazepine, phenobarbitol; case reports of asterixis induced by clozapine with carbamazepine and lithium; case reports of delirium when benzodiazepines added
	Quetiapine	Potential changes in plasma level with phenytoin, carbamazepine, phenobarbitol, primidone due to CYP 3A4 interactions
Classic neuroleptics	Haloperidol	About 50% decreased haloperidol levels with carbamazepine, increased carbamazepine levels with haloperidol
	Loxapine	Increased carbamazepine epoxide levels with loxapine
	Thioridazine	Decreased levels with phenobarbitol; phenobarbitol levels variably affected; rare variable effect on phenytoin levels

reserved for patients for whom other treatments have failed. If frequent epileptiform discharges are responsible for poor attention or overactivity, the appropriate treatment should be effective antiepileptic medication.

- Intermittent explosive disorders or episodic dyscontrol syndromes can also be a direct manifestation of seizure discharge. Ictal violence is likely to be non-directed and stereotyped, whereas organized violent behaviour is directed at specific targets. A patient may also be violent during post-ictal confusion.
- Non-ictal violence is associated more frequently with mental retardation and psychosis. The use of atypical neuroleptics such as risperidone, olanzapine or quetiapine can be effective for intermittent explosive disorder. However, given the often long-standing administration of these drugs, there is a concern of side effects including tardive dyskinesia.
- Depression and anxiety disorders are common among children with epilepsy. If psychological interventions are not feasible or have failed, antidepressant medication may be initiated. In line with current approaches in the general population, the selective serotonin re-uptake inhibitor (SSRI) represent the drugs of first choice (see chapter on Affective Disorders). If the patient does not respond to the first SSRI, a second should be tried. Furthermore, augmentation by use of other drugs is possible. Buproprione is to be avoided in epileptic children because it increases the number of seizures. Possible interactions between SSRI and AED are listed in Table 1.9.
- Panic affects or other anxiety disorders can also be treated briefly with benzodiazepines. However, the development of tolerance and the risk of withdrawal symptoms, including increased seizures, limit the administration of benzodiazepines for the short term until SSRI take effect. Also, the behavioural toxicity of benzodiazepines in children with brain disorders needs to be kept in mind.
- Mood instability can be seen as a symptom of bipolar disorder, severe ADHD or intermittent explosive disorder. Mood stabilizers such as carbamazepine or valproic acid, or the recently developed AEDs, can have significant effects including better tolerance of stimulant treatment of ADHD. Among the mood stabilizers lithium is most proconvulsive, whereas the other mentioned drugs can have significant interactions with other AEDs (see Table 1.9).
- Psychotic symptoms and illogical thinking are more common in children than previously believed. First-line medication is represented by the atypical neuroleptics risperidone, olanzapine and quetiapine. Clozapine should be avoided because of its pronounced proconvulsive effects or be reserved for psychoses that have been refractory to other drugs.

Monitoring and evaluation of treatment

The essential role that AED treatment plays in symptom control, and the common indications for the administration of psychotropic drugs, makes continuous

monitoring of effects and side effects mandatory. Laboratory checks need to control for plasma levels and toxicity. In addition, careful observation of symptoms, especially of seizures, is essential for the titration process of AED treatment.

Any intervention for psychiatric problems, be it psychological or pharmacological, needs to be evaluated for its outcome. Both the clinical interview, and the administration of standardized questionnaires and checklists, serve these purposes. Examples for checklists were given above in the section on diagnostic instrument and assessment.

Problematic issues

There is a sizeable proportion of medically refractory epilepsy, which has been recently estimated to include 10–20 per cent of new patients per year with seizures. These patients could be candidates for epilepsy surgery. Presurgical examination has to determine whether or not there is an epileptogenic zone in the brain that is apt for cortical resection. Invasive monitoring has to exclude, via critical stimulation, that no so-called eloquent cortex tissue is removed that would leave the patient with an unacceptable neurological handicap. Focal resection would only be appropriate if the epileptogenic zone is not in eloquent cortex.

In severe cases with a large epileptogenic zone, localization to one hemisphere, and a contralateral neurological deficit, hemispherectomy can be considered. If the epileptogenic zone cannot be identified and the patient suffers from multifocal seizures, especially with generalized seizures that lead to drop attacks, a corpus callosotomy, leading to a disconnection of the two hemispheres, can be performed. The corpus callosotomy is only a palliative procedure, whereas both focal cortical resection and hemispherectomy are curative.

Recent evaluations of surgical treatment of epilepsy in children, adolescents and adults have provided favourable results. Good outcome was particularly associated with the resection of tumours, temporal lobe surgery before the age of 15 years and less than 10 years' duration of epilepsy. Psychosis is no longer considered to be an absolute contraindication to neurosurgery. Some children, e.g. those suffering from the rare Landau–Kleffner syndrome improve dramatically in their learning ability after surgery. In temporal lobectomy the complete removal of abnormal tissue containing the epileptogenic zone should lead to better outcome. With early onset of seizures, there is a good chance that brain plasticity will have ensured that most useful cognitive functions reside in the normal tissue.

Outcome

The general outcome of epilepsy varies with the type of seizures. Infantile spasms mainly occur as a result of pre-existing brain damage and therefore carry a poor prognosis. The same also applies to the Lennox–Gastaut syndrome. In contrast,

absence seizures respond well to adequate anticonvulsive treatment and do not lead to handicapping long-term developments.

Also temporary lobe epilepsy (TLE) has a better long-term prognosis than once considered. This statement applies particularly for the simple partial seizures rather than for the complex practical seizures in which the persistence into adulthood is associated with poor social outcome. Most children with TLE become adults without psychiatric disorder. Risk factors for an unfavourable outcome include low IQ, early onset of epilepsy, high frequency of seizures, a left-sided EEG focus, hyperkinetic disorders, rage attacks and a history of special education.

Prognosis for intellectual function is good in uncomplicated epilepsies where there is only one seizure type, which responds promptly to a simple anticonvulsant regimen. This applies especially for onset of epilepsy later in childhood or in adolescence.

Brain tumours

Definition and classification

The major brain tumours in childhood comprise medulloblastoma, astrocytoma, brainstem glioma and ependymoma. Other less frequent brain tumours in childhood include mesodermal tumours (e.g. meningioma) and tumours of the pituitary (e.g. craniopharyngioma). In children, over two-thirds of the primary brain tumours arise below the tentorium, whereas in adults tumours tend to arise above the tentorium. Less than 45 per cent of brain tumours are malignant with an exceptionally poor prognosis, such as high-grade glioma, ependymoma, high risk primitive neuroctodermal tumours (an inclusive term for medulloblastoma and other histologically similar CNS tumours) and brain tumours in infants.

Epidemiology

Incidence rates

Recent statistics from the United States have calculated an annual incidence rate of 22 cases per million. Similar rates have also been estimated in other countries. Astrocytoma make up 45 per cent of the relative incidence rate, followed by medulloblastoma with 20 per cent, and brainstem glioma and ependymoma each with 10 per cent.

Implications for clinical practice

Brain tumours in childhood are relatively rare. However, both the malignancy of certain types of tumours, and the high impact on quality of life of others in long-term

survivors, imply high demands on care and expertise. Only in highly specialized teams of the tertiary services will child and adolescent psychiatrists see sufficient numbers of patients to build up special expertise. However, many surviving patients will return to the community and will require child psychiatric care and treatment there.

Clinical picture

Main features and symptoms

The determinants of clinical symptoms in brain tumours are age at onset, location of the tumour, rate of growth and the presence of metastatic disease. In school-age children clinical syndromes mainly depend on the neuroanatomic location (i.e. infratentorial vs. supratentorial) and whether or not they result in raised intracranial pressure.

- The posterior fossa or intratentorial compartment is the most common neuroanatomic location for brain tumours in children. Frequent expansion of growth results in a variety of neurological symptoms. A full description of these symptoms is beyond the scope of this chapter. Suffice it to say that the neurological sequelae of cerebellar astrocytomas, ependymomas or brainstem tumours include a variety of severely handicapping symptoms, such as hydrocephalus, hemiparesis, vomiting, headache, ataxia, diplopia, dysphagia and dysarthria to name just a few major symptoms.
- The supratentorial tumours originate either from the dicencephalon or from the cerebral hemispheres. Craniopharyngiomas or germ cell tumours may result in endocrine dysfunction with growth failure, precocious puberty, obesity, hypothermia and may cause hydrocephalus and raised intracranial pressure. Tumours arising in the cerebral hemispheres usually are associated with seizures of focal neurological deficits. Most of these tumours are astrocytomas and mixed glial tumours.
- Psychiatric symptoms sometimes may precede gross neurological abnormalities. A change in behaviour or personality and cognitive deficiencies may be the primary manifestation of a frontal lobe tumour. Difficulty in performing simple, repetitive procedures in terms of apraxia may be noticeable before major neurological symptoms are apparent. Cerebellar tumours may present with lethargy and irritability and tumours causing endocrine dysfunction such as craniopharyngioma may be accompanied by affective lability, depression, anorexia, disorders of impulse and sleep disturbances. Also in brainstem tumours such as diffuse pontine glioma behavioural symptoms including attention deficits, irritability and even personality changes may be present as very first indicators. Depending on the location, cerebral tumours may be associated by visual hallucinations if

the occipital lobe is affected, and agnosia and apraxia if the locus is the parietal and temporal lobe. Due to the raised intracranical pressure, cerebral tumours frequently produce disordered consciousness.

- As a result both of the tumour itself and treatment intervention, various cognitive impairments may result. In medulloblastoma long-term effects of neurocognitive deficits in attention, memory, language comprehension and acquisition, and academic achievement depend on age of diagnosis, type and duration of symptoms, tumour extent, cranial radiotherapy (CRT) and duration of survival. CRT generally results in cognitive impairment, i.e. in deficits in fine motor, visuo-motor, visuo-spatial and memory functions and is associated with a drop in IQ and needs for special assistance in school.
- In addition to disease-related factors, psychosocial adaptation is challenged by a variety of further risk factors. Hospitalization, multiple painful and medical procedures, alteration of physical appearance, isolation from peers, and the wide range of possible deficits in senses, mobility, emotional and behavioural functioning may overstress the coping resources of the child. Also, the family may be overwhelmed by the multitude of psychological stresses as evidenced by the initial shock of the diagnosis, the uncertainty of prognosis, the stress of decision making, the disruption of family life and the disruption of normal social activities that almost inevitably result from the severe disorder of the child. Thus, social isolation, alienation and disruption may result both for the child and for the family and may coincide with, or lead to, various behavioural abnormalities.
- Finally, additional medical therapy involving anti-epileptic drugs (AED) and steroids can have an impact on both cognitive and behavioural functioning. AEDs can alter attention and may cause mood changes. Steroids with higher doses can result in irritability, excitation, insomnia and also psychosis, most particularly in the postoperative phase, but also later on.

Differential diagnosis and co-morbidity

With the rather unspecific first behavioural signs, many manifestations of brain tumours may be mistaken as abnormalities resulting from other than brain-related factors. Recurrent vomiting, anorexia or chronic weight loss may imply gastroenterologic dysfunction. Affective lability, frequent mood swings or depression might, at first sight, raise the question as to whether a primary affective disorder is present. Tumours of cerebral hemispheres with presenting hallucinations might also imply the differential diagnosis of schizophrenic psychosis. Various neurological syndromes (e.g. aphasia) and endocrine disorders should be considered as consequences of brain tumours rather than as co-morbidities. Differential diagnosis rests mainly on a complete neurological examination and neuroimaging of the

brain, preferentially by MRI. For ordinary child and adolescent psychiatric practice, it is essential to exclude a primary brain tumour in various symptoms of psychopathology.

Diagnostic instruments and assessment

The psychiatric contribution to the interdisciplinary examination of brain tumours in children is primarily based on observation and exploration of the various symptoms of psychopathology across a long-term period. Symptoms will be varying not only as a function of tumour location and raised intracranical pressure but will also alter across time due to tumour development, intervention effects, developmental effects and environmental conditions.

In long-term survivors, and with the aim of evaluating medical interventions, neuropsychological testing is of great importance. The often progressive deficits over time in intellectual functioning and learning make cognitive assessment mandatory at regular intervals after therapy in order to institute appropriate interventions. Various functions in the area of intelligence, language, motor, attention, perception and executive functions need to be assessed. Some guidance with regard to clinically related questioning and recommendations for suitable tests is given in Table 1.2.

The question of behavioural and emotional functioning can also be assessed by standardized questionnaires and checklists like the Child Behaviour Checklist (CBCL) for parents, the Teacher Rating Form (TRF), or the Strengths and Difficulties Questionnaire (SDQ, see the Appendix to chapter on conduct disorders). Questionnaires for the assessment of quality of life may also provide valuable insights into the stresses and strains of the child and the family. These instruments assess domains such as activity and mobility, psychological well-being, social integration, comfort or pain, achievement and others.

Aetiology

The large majority of brain tumours in children arise without any obvious predisposing condition. However, there are a few hereditary syndromes that can lead to central or peripheral nervous tumours. They include neurofibromatosis-1, which is an autosomal dominant disorder with a mutation on the long arm on chromosome 17, neurofibromatosis-2, which is due to a mutation on the long arm of chromosome 22, and tuberous sclerosis, which is an autosomal dominant disorder.

As in other brain disorders, the aetiology of psychiatric symptoms in brain tumour is multidetermined. The various factors include tumour-related risks (e.g. tumour location, raise of intracranial pressure), treatment-related risks (e.g. CRT, AED, steroids), general disease-related risks (e.g. chronicity, progressive nature,

hospitalization), individual risks (e.g. developmental factors, coping deficits) and environmental risks (e.g. lack of family support, social isolation). Deficits in psychosocial adaptation and psychiatric symptoms may vary inter-individually depending on the interaction of these risk factors.

Treatment

Clinical management and treatment setting

The principles of therapy of brain tumours include radical surgical resection wherever possible, assisted by cranial radiotherapy and chemotherapy when indicated. The broad spectrum of early and late effects, including various neurological deficits, hormonal deficiencies as well as cognitive and behavioural dysfunction, requires an extended and highly specialized clinical team. In particular, experts in neurosurgery, anaesthesia, neuro-oncology, neuropsychology, psychiatry, rehabilitation and social work are needed to provide the necessary specialized care. Tertiary service centres are needed for the provision of these programmes.

Psychological interventions

The need for psychiatric assistance may be articulated repeatedly in the course of care for children with brain tumours. In the first 48 hours after operation, pain syndromes, mood disturbances, withdrawal and suicidal ideation require assessment and intervention. During hospitalization and after discharge, many of these problems may persist and may be supplemented by additional symptoms such as phobias, anxiety and anger.

During the various phases of clinical treatment, psychological preparation for various examinations and surgery is necessary. Expertise in behavioural techniques, imagery or hypnosis can be used in order to prepare the child for the management of pain, anxiety, needle phobias as well as nausea and vomiting associated with chemotherapy. The recovery process of the child will be assisted greatly by these interventions.

Any pre-morbidly existing or post-diagnosis developed co-morbid psychiatric problem can also be addressed by various interventions of the child and adolescent psychiatrist including brief and supportive psychotherapy, crisis intervention, behaviour modification or long-term care and counselling. The family should receive similar guidance and assistance during the various phases of adaptation with an initial shock phase and later on in developing stable adaptation processes.

Further goals include the professional reflection of feelings and reactions of the rehabilitation team. Members of the team that cares for children with malignant disorders may themselves develop feelings of depression, anxiety and burn-out

so that team counselling is another important task of the experienced child and adolescent psychiatrist. Finally, he or she is also an important mediator and counsellor to the school system in establishing appropriate expectations for achievement and academic progress.

Psychopharmacotherapy

Indications for drug treatment include the use of SSRI and mood stabilizers with affective disorders and of atypical neuroleptics with psychotic symptoms and severe aggressive outbursts. Stimulants may not only be helpful for the treatment of ADHD symptoms but also in the so-called post-radiation syndrome, which can last for as long as 6 months after radiotherapy and is characterized by increased somnolence and fatigue and by marked attention deficits.

In general, medications with use in brain-injured patients can be administered similarly in children with brain tumours. An outline is given in Table 1.3. In the case of co-existing epilepsy and treatment with AEDs, the same caveat regarding interactions between AEDs and psychotropic medication applies that has been articulated in the section on the treatment of epilepsy and co-morbid psychiatric disorders (see Table 1.9).

Monitoring and evaluation of treatment

Careful observation of the patient for any change of the target symptom of intervention, and continuous monitoring of any side effect including toxicity are mandatory for any implementation of drug treatment. Neuropsychological testing may also be helpful in order to detect subtle changes in cognitive impairment. In some instances it may be difficult to differentiate between intervention effects by pharmacotherapy or CRT or direct effects of the tumour.

Psychological interventions may be evaluated by use of structured questionnaires for the assessment of personality features, behavioural and emotional abnormalities, quality of life and satisfaction of the client, be it the patient, the family or the rehabilitation team.

Problematic issues

Both the fatality of a brain tumour and treatment failure represent major problems in the care of children with brain tumours. Malignant tumours can only be insufficiently cured. Thus counselling the child and his family in the process of dying is one of the most difficult tasks in psychological care. It requires a most sensitive, reflective and honest guidance by the professional in order to assist the patient and his family in a process of mourning and grief that may start early in the disease process and continue after the death of the child.

Treatment failure can result from the inability to achieve local control either because of surgical inaccessibility or because of lack of therapeutic intent at the initial surgical resection. Often, radical surgical resection could be achieved only at the cost of neurological morbidity. Only new technologies that increase the ability to more safely resect brain or more effective medical therapies will solve this dilemma.

Outcome

Long-term survival after developing a brain tumour in childhood is different for the various types of tumours. The overall 5-year survival rate is 70 per cent in medulloblastoma of standard risk and 40% in high risk medulloblastoma. Low grade astrocytoma has the highest 5-year overall survival rate with 90% of the affected children, whereas it is only 30% in high grade astrocytoma. The figure is lowest for brainstem glioma (7%) and amounts to 50% for ependymoma. The rates for progression-free survival is lower than the reported overall figures.

As described above, long-term survivors show a high rate of cognitive impairments and limited educational experiences. Psychological maladjustment is prominent and has been shown empirically to depend on low socio-economic standards, young mothers and single-parent families, younger age of the patient, physical stigmatization, functional impairment and low IQ.

Syndromes attributed to minimal brain dysfunction

Definition and classification

The concept of minimal brain damage (MBD) emerged in the early twentieth century, years after George Still described a syndrome that he linked to 'moral dyscontrol' and that comprised attention deficits, impulsivity, motor restlessness, clumsiness and various types of learning problems. It became rooted firmly after an epidemic of encephalitis around 1920 had left many previously purportedly well-functioning children affected by similar symptoms. In the late 1940s and the 1950s it became one of the most common 'diagnoses' in child psychiatry. However, criticism against the concept mounted in the 1960s when it became clear that not all children with the problems referred to had 'minimal' brain damage, but widespread brain dysfunction, and that others had no documented brain damage at all. The meaning of MBD then changed to minimal brain dysfunction. In the early 1980s, two influential papers by Sir Michael Rutter – highlightling again the criticism from the 1960s and adding that children with various kinds of proven minimal brain damage did not show necessarily any of the symptoms believed to be diagnostic of MBD – seemed to put the concept to rest. It soon became superseded by other labels and combinations of labels such as attention deficit disorder, hyperkinetic disorder, deficits in attention, motor control and perception syndrome (DAMP), and

specific developmental disorders (including developmental coordination disorder or DCD).

The research literature has had little to say about MBD in the past 20 years. However, in clinical practice, the term has remained in many centres, usually as an umbrella term for various combinations of problems in the areas of attention, activity control, impulsivity, motor control, perception and learning (including speech and language).

A survey of all the many synonyms, acronyms and partly overlapping concepts used in this field is beyond the scope of this chapter. However, a list of some of the most common diagnostic labels is appropriate (Table 1.10). The Table provides a brief introduction to the symptom profiles encountered in children who have been given the, sometimes incorrect, label of MBD over the past 50 years.

Epidemiology

Prevalence rates

The prevalence rates for 'MBD' have varied from a few per cent to as many as one in five children, even though most estimates have been below 10 per cent of all children at school age. The figure has varied most of all with the definition used. Unfortunately, many of the studies in the field have not used operationalized criteria at all. One of the very few studies actually to have used strict criteria found a minimum rate of 4 per cent in the 1970s in Sweden, with one in four of these being regarded as severe. That study referred to 7 year-old children with the combination of the problem now subsumed under the label of DAMP (meaning the combination of AD(H)D and DCD). Boys are about three times more often affected than girls.

Implications for clinical practice

The MBD label is still used sometimes in clinical practice to describe children who usually have ADD or ADHD in combination with various kinds of developmental and learning disorders. In Scandinavia, such combinations are diagnosed frequently under the descriptive label of DAMP. This 'condition' affects a few to several per cent of all school-age children.

Clinical picture

Main features and symptoms

Epidemiological and clinical studies of children with DAMP have shown the following characteristics with regard to psychiatric disorders.

- The prevalence of psychiatric problems is very much higher than in children without DAMP amounting to two-thirds of the population of children with the syndrome. It is increased significantly if DAMP is combined with psychosocial

Table 1.10. Syndromes attributed to minimal brain dysfunction

Diagnostic label	Comments
MBD	Once referred to minimal brain damage, since *c.* 1960 to minimal brain dysfunction. Almost universally used label in child psychiatry up until *c.* 1980. Still in clinical use in many countries. Usually refers to various combinations of attention and motor/learning problems. Inappropriate in that it implies brain dysfunction on phenotypical grounds and in its use of the word 'minimal'
ADD (Attention deficit disorder)	DSM-III-label (APA 1980). Still in use in the US, usually to refer to the inattentive subtype of AD/HD (see below). Semantically confusing (should be 'deficit' *or* 'disorder', not both). Diagnostic criteria in the DSM-III loose and subjective. Pervasiveness not required. With or without motor/learning problems
ADHD (Attention deficit hyperactivity disorder)	DSM-III-R-label (APA 1987). Does not account for cases without clear hyperactivity. Pervasiveness required for cases classified as 'severe'. With or without motor/learning problems
AD/HD (Attention-deficit/hyperactivity disorder, predominantly inattentive, predominantly hyperactive–impulsive, or combined subtypes)	DSM-IV-label (APA 1994). Accounts for cases without clear hyperactivity, but does not make it clear how the two predominant subtypes are inter-related (if at all). Requires a measure of pervasiveness. With or without motor/learning problems
DAMP (Deficits in attention, motor control and perception)	Accepted term in Nordic countries. Umbrella concept covering various combinations of attentional/activity problems in conjunction with motor control and perceptual problems. Recently harmonized with DSM-IV to mean AD/HD with DCD (see below). Probably the concept that resembles George Still's original descriptions most closely
Hyperkinetic disorder	ICD-10-label (WHO, 1993). Mostly used in the UK. Refers to a syndrome that must include all of inattention, impulsivity and hyperactivity. In the past, this diagnosis was often made only if there were no major oppositional or conduct problems. The syndrome was then considered very rare. As used currently it is believed to be not very rare, that oppositional and conduct problems are extremely common, and that motor/learning problems are the rule in severe cases
Clumsy child syndrome	UK concept. Highlights only one aspect of what is usually a multifaceted syndrome
MPD (Motor perception dysfunction)	Nordic concept. Attention problems, autistic features and other specific learning problems are common in this group
DCD (Developmental coordination disorder)	DSM-IV-label (APA 1994). Synonymous with Nordic concept of MPD
MND (Minor neurological dysfunction)	Sometimes used to describe summary score for minimal motor/neurological problems or 'soft neurological signs'
MCD (Minimal cerebral dysfunction)	Rarely used term. Usually refers to overriding concept of cerebral dysfunctions rather than to any specific clinical syndrome
Organic brain syndrome	Central European concept. Highlights certain behavioural features, but essentially similar to MBD-concept
OBD (Organic brain dysfunction)	Used particularly by European groups highlighting the importance of neonatal reflexes in the pathogenesis of attentional and motor/learning problems

adversity. However, the link with autism spectrum problems (see below) is associated with the severity of the symptoms of DAMP and not at all with psychosocial conditions.

- In general, there is no distinctive association of DAMP with any type of psychiatric disorder except for ADHD (which is usually included in the DAMP/MBD diagnosis *per se*), autism spectrum disorders, depression and conduct disorder. In DAMP, with its relatively mild physical disability and little or no intellectual impairment, around two-thirds of affected children have at least one psychiatric disorder. Autism spectrum disorders (including full-blown Asperger syndrome) occur in more than half of all those with severe DAMP (children with severe DAMP usually have all of the following: attention deficits/hyperactivity, fine motor problems, gross motor problems, perceptual dysfunction and speech and language deficits). It is not uncommon for a child to present with DAMP around the age of 3 or 4 years and then to be 're-diagnosed' some years later as having an autism spectrum disorder. Conversely, some children are suspected of suffering from an autism spectrum disorder at a very young age and then develop the typical symptoms of DAMP (attention deficits and motor clumsiness and an uneven pattern of abilities in the field of learning). Depression is present in about one-third of all children with DAMP, regardless of degree of severity. The highest rate appears to be around the age of 8–12 years, and may be associated in many cases with feelings of always being unsuccessful at school and in peer relationships. Oppositional defiant problems are present in about 35–40 per cent of all pre-school children with DAMP. When present, it is usually symptomatic from a very young age and does not develop later in childhood. Conduct disorder and later antisocial personality disorder develop in about half of these cases with severe oppositional and defiant behaviours.
- The most common emotional disorders in children with DAMP are specific phobias, separation anxiety and generalized anxiety. Some patients also develop depressive disorders (see above). Frequently, these children with DAMP and emotional disorders also show markedly defiant and negativistic behaviour, leading in some cases also to marked violent behaviours.
- Children with DAMP encounter an increased risk of social isolation due to teasing, victimization, marginalization and lack of friendships. These problems may, in part, be due to constitutional problems with social understanding and relatedness. In severe cases, autism spectrum disorders may be present (see above).
- Psychiatric and behavioural problems are often very persistent and may be seen for many years. Over the years, the additional ('co-morbid') psychiatric problems are often those that contribute to the very poor psychosocial outcome seen in so many cases with DAMP. As children with DAMP grow up, many will meet criteria for one or more of the so-called 'personality disorders'. However, it is unclear

whether it is helpful or not to make 'additional' diagnoses of these conditions in people with DAMP, given that they provide little in the way of explaining the underlying problems or in terms of suggesting avenues for appropriate interventions.

Aetiology

The causes of DAMP are not known in any detail, but familiality is extremely common and there is a high rate of associated pre- and perinatal problems. Most of the available indirect evidence (such as that from the study of attention deficits and hyperactivity, which are universal phenomena in DAMP) suggests that the familiality is due to genetic factors. The meaning of the high rate of pre- and perinatal problems is less clear. These problems could be a reflection of an underlying genetic abnormality in the child, an index of psychosocial adversity (which is also common in the DAMP syndrome) or a factor indicating a risk for structural brain damage. The few studies that have looked at these factors in any detail suggest that the DAMP syndrome is often caused by genetic factors, either alone or in combination with some degree of brain damage. Psychosocial factors themselves appear not to contribute to the presentation of the DAMP syndrome as such but may be strongly associated with co-morbid conditions such as oppositional defiant disorder and conduct problems.

Treatment

The basic condition of DAMP cannot be cured at the present time. Nevertheless, attention deficits and hyperactivity are amenable to the same kind of interventions that are helpful in treating ADHD (see chapter on hyperkinetic disorders). Many families of children with DAMP benefit greatly from having the diagnosis made and explained to them in some detail. The children will usually derive considerable benefit from having their teacher gaining a better understanding of the child's specific needs in terms of the educational setting. Training of working memory is probably important in many cases. A training programme for the motor deficits that are part and parcel of the DAMP syndrome is sometimes a key intervention that may have to be drawn up by a physiotherapist or by an occupational therapist. Physical education teachers usually have to be informed in detail about the child's motor control problems. Some children with DAMP need the additional input of speech–language therapists. Those who do not seem to develop in a hopeful manner in spite of these kinds of interventions may be candidates for treatment with a stimulant or another drug effective in the treatment of ADHD. Associated psychiatric problems usually need the same approach when it comes to interventions as is useful for children with the particular psychiatric problem without DAMP.

With the high probability of developing additional psychiatric problems, there is not only the need for careful monitoring and assessment but also for early intervention or even for the development of preventive approaches. In principle, all sorts of psychological interventions are applicable. A family approach in counselling and guidance is always indicated. All these efforts should be started early and often be continued over many years.

Outcome

DAMP/MBD itself is a chronic condition marked by persistent psychosocial disability of varying degree. Probably no more than one in three does well in terms of education, vocational skills, daily living skills, psychosocial interactions and independent living in their mid-twenties. A limited database suggests that diagnosis in childhood, and interventions as outlined in the foregoing (particularly psychoeducational approaches), may contribute significantly to a better outcome. Concomitant psychiatric disorders of various kinds – perhaps particularly autism spectrum disorders and conduct disorder – contribute to a worse outcome. Learning disability/mental retardation and severe executive function deficits may have an additional impact on the course of the disorder. However, it has to be emphasized that some affected individuals experience a satisfying and productive life with normal psychosocial adaptation.

There is a very limited body of empirical research on the longitudinal stability of psychiatric and academic problems in children suffering from DAMP. One longitudinal prospective study from Göteborg, Sweden, suggested that, if untreated, DAMP may be associated with a poor psychosocial outcome in more than 50 per cent of the cases. 'Poor outcome' in that study referred to the presence in early adult age of one or more (usually two or more) of the following: full-time sick pension, severe criminality, severe drug or alcohol abuse, persistent major psychiatric disorder (such as bipolar disorder and schizophrenia), or the persistence of a disabling autism spectrum disorder. No more than a few per cent of all cases had had a successful academic career.

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2

Substance use disorders

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Introduction

Substance use and abuse by children and adolescents remain critical problems for modern developed countries. Although tolerance and public policy for substance use vary among Western countries, the use of psychoactive substances and other harmful of abuse is common among adolescents. The use of such psychoactive agents can lead to a variety of negative consequences for youth. The risk for substance use and abuse, the acquisition of use behaviours, and development into substance use disorders and interventions for such problems should be considered in a comprehensive manner that considers neurobiology, development and the adolescent's environmental ecology.

This chapter presents essential background information for the clinician in order to understand the presentation, risk, prevention and treatment of adolescents with substance use problems. While written primarily from the perspective of clinical and research experience in the United States, the processes of addictive behaviours are similar for all adolescents. Modifications for assessment and treatment should be made for cultural and ethnic differences when relevant.

Definition and classification

According to the *International Classification of Disease*, 10th edition (ICD-10), the pathological use of substances is classified under the heading of 'Mental and behavioural disorders due to psychoactive substance use'. In addition to the clinical conditions of acute intoxication with or without various complications, withdrawal states, psychotic disorders and amnestic disorders; the primary diagnoses are 'harmful use' and 'dependence syndrome', which correspond closely to the *Diagnostic and Statistical Manual of Mental Disorders – IV*, 4th edition (DSM-IV) diagnoses of

A Clinician's Handbook of Child and Adolescent Psychiatry, ed. Christopher Gillberg, Richard Harrington and Hans-Christoph Steinhausen. Published by Cambridge University Press.

'substance abuse' and 'substance dependence'. The term 'substance use disorders' encompasses both substance abuse and substance dependence, under the DSM-IV category of substance-related disorders. Substance use disorders are defined for alcohol, amphetamine or amphetamine-like, caffeine, cannabis, cocaine, hallucinogens, inhalants, nicotine, opioids, phencyclidine (or phencyclidine-like) and sedative-hypnotic- or anxiolytics. Although these parameters were written to apply to both children and adolescents, adolescents are referred to most frequently due to the less frequent occurrence of SUDs in prepubertal children.

In ICD-10, 'harmful use' is defined as a pattern of psychoactive substance use that is causing damage to health. The fact that use or a pattern of use is disapproved of by another person or by culture, or may have led to socially negative consequences such as arrest (e.g. for use or possession) or family arguments is not in itself evidence of harmful use. For the ICD-10 Dependence Syndrome, the central descriptive characteristic is the often strong desire to take psychoactive substances. The syndrome also may include a physiological withdrawal state, evidence of tolerance and persisting use despite overt negative consequences.

The diagnosis of DSM-IV 'substance abuse' requires evidence of a maladaptive pattern of substance use with clinically significant levels of impairment or distress. Substance abuse is characterized by a maladaptive pattern of substance use. Recurrent use in adolescents who abuse substances results in an inability to meet major role obligations, leading to impaired functioning in one or more major areas of their life, and an increase in the likelihood of legal problems due to possession, risk-taking behaviour and exposure to hazardous situations. Substance dependence requires a substantial degree of involvement with a substance as evidenced by the adolescent meeting at least three criteria, including such symptoms as withdrawal, tolerance and loss of control over use. Adolescents commonly exhibit tolerance (i.e. requiring increasing amounts of a substance to achieve the same effect), which is one criterion for dependence, but show withdrawal or other symptoms of physiological dependence much less frequently. Tolerance, in the case of adolescent alcohol use, appears to have a low specificity for a diagnosis of alcohol dependence among adolescents, while withdrawal symptoms and medical problems as a consequence of use are much less common than in adults. However, increasing research points to a high prevalence of withdrawal symptoms in adolescents with cannabis and opiate use disorders. Preoccupation with use is often demonstrated by giving up previously important activities, increasing the time spent in activities related to substance use, and using more frequently or for longer amounts of time than planned. The adolescent may use despite the continued existence or worsening of problems caused by substance use. For adolescents, it is important to include criteria such as alcohol-related blackouts, craving and impulsive sexual behaviour

when determining if criteria are met. Polysubstance use by adolescents appears to be the rule rather than the exception; therefore, adolescents often present with multiple SUD diagnoses.

Although these diagnoses of substance abuse and substance dependence assist clinicians in identifying adolescents with pathological patterns of substance use, the ICD-10 and DSM-IV criteria, developed for adults, have not been established as applicable to adolescents. For adolescents these diagnoses probably define a heterogeneous population and communicate insufficient information in terms of natural history and treatment response. While ICD-10 and DSM-IV remain the guides for determining substance-use-related pathology in adolescents, it is important to recognize the frequent differences between the most common manifestations of the diagnoses of substance abuse and dependence in adolescents vs. adults. While substance use is a necessary prelude to abuse or dependence, and regular use further increases the risk for SUDs, substance use *per se* is not sufficient for a diagnosis of abuse or dependence. Through the remainder of this chapter, I will use the term substance use disorders (SUDs).

Epidemiology

Prevalence and incidence rates

In the United States, substances such as opiates, LSD, inhalants and steroids have shown periodic epidemics among youth in the past several decades. In 2000, nearly a quarter of eighth graders report having taken an alcoholic beverage in the past 30 days and half of twelfth graders report having done so. A third of twelfth graders report having been drunk in the past 30 days. Almost 12 per cent of eighth graders and almost a quarter of high school seniors report having used any illicit drug in the preceding 30 days. In community studies, lifetime diagnosis of DSM-IV alcohol abuse ranges from 0.4 per cent to 9.6 per cent. Lifetime diagnosis of alcohol dependence range from 0.6 per cent to 4.3 per cent in the Oregon Adolescent Depression Project. The lifetime prevalence of drug abuse or dependence has ranged from 3.3 per cent in 15-year olds to 9.8 per cent in 17 to 19-year olds. Other than alcohol, 54 per cent of 12 graders and almost 27 per cent (26.8%) of eighth graders report ever having used any illicit drug. Marijuana is the most widely used of the illicit drugs with about a third of high-school students indicating some use in the preceding 12-month period. Six per cent of high-school seniors report daily marijuana use.

Overall, school surveys show that the lifetime prevalence of illicit drug use is highest in the USA and Australia (>40%) and high in Canada (>35%), but less than 20–25 per cent in Europe. In the late 1990s, the lifetime prevalence of illicit

drug use rose 40% in Europe, while an increase in the USA over the 1991–7 period was followed by a decrease during 1997–2001. The increase in Europe appears to be the result of a doubling of prevalence rates in Eastern Europe. There are important differences between European countries. For example, in 1999, the lifetime prevalence of all illicit drug use was 36 per cent and 12 per cent for use of drugs other than cannabis in the United Kingdom (UK). During the same period, in Scandinavian countries, the lifetime prevalence ranged from 9–13 per cent for all illicit drugs and 3–6 per cent for drugs other than cannabis. The vast majority of students in Europe have drunk alcohol at least once in their lives, ranging from 61 per cent in Turkey to 95 per cent in the Czech and Slovak Republics and Denmark. The proportion of students reporting having drunk alcohol more than 40 times include Denmark at 49 per cent and the UK at 42 per cent. Binge drinking within the past 30 days was reported by 58 per cent of students in Denmark, 51 per cent in Finland, 48 per cent in the UK and 46 per cent in Iceland. Generally, teenagers in a group of northern countries reported the highest rates of heavy drinking and intoxication (drunkenness). Teenagers in southern Europe reported much lower levels of such behaviours and experiences.

As of 2000, cigarette smoking among adolescents in the USA has continued a decline from a peak in the mid-1990s. An almost 50 per cent increase in the rate of smoking among younger adolescents (eighth and tenth graders) in the early 1990s. In 2000, 14.6 per cent of eighth graders reported current smoking (defined as smoking at least once in the preceding 30 days) while 23.9 per cent of tenth graders and 31.4 per cent of twelfth graders reported current cigarette use. Approximately a quarter (24.7 per cent) of students nationwide reported having smoked a cigarette before the age of 13 years.

Sex ratios and ethnicity

For all substances, males use more than females, although the gap appears to be closing during the past two decades. Similarly, the gap is closing between lower levels of substance use in rural areas and higher levels in urban areas. This smaller gap may be due to increasing accessibility to all youth whether or not they live in urban or rural areas.

In the USA, white and Hispanic students report a lifetime prevalence of about 82 per cent for alcohol use as compared with 74 per cent for African-American youth. Heavy episodic use prevalence rates are 35, 37.7 and 18.8 per cent for white, Hispanic and African American students, respectively.

African-American students are more likely to report both lifetime and current use of marijuana than white students. In other countries similar ethnic differences in the rates of substance use and problems associated with use may be present.

Implications for clinical practice

Substance use by adolescents is not distributed randomly among adolescents of different genders, ethnicity or other demographic characteristics. Clinicians need to be aware of both demographic and other high-risk characteristics in order to target the most vulnerable populations of adolescents for prevention and treatment interventions. The specific characteristics of an adolescent may also shape the types of interventions or emphasis within an intervention that are used. For example, urban or female adolescents may have special needs distinctive from the intervention needs of rural or male adolescents.

Clinical picture

Main features and symptoms

Patients who present with substance use, and frequently with resulting intoxication, often manifest significant levels of acute change in mood, cognition and behaviour. The manifestations of substance use and intoxication vary with the type of substance(s) used, the amount used during a given time period, the setting and context of use and a host of characteristics of the individual such as experience with the substance, expectations, and the presence or absence of other psychopathology.

- Behavioural changes may include disinhibition, lethargy, hyperactivity or agitation, somnolence and hypervigilance.
- Changes in cognition may include impaired concentration, changes in attention span, and perceptual and overt disturbances in thinking, such as delusion.
- Mood changes can range from depression to euphoria.
- A hallmark of SUDs in adolescents is impairment in psychosocial and academic functioning. Impairment can include family conflict or dysfunction, interpersonal conflict and academic failure.
- Associated characteristics include deviant and risk-taking behaviour, and comorbid psychiatric disorders such as conduct, attention-deficit/hyperactivity, mood and anxiety disorders.

Almost all psychoactive substances, including those available to adults such as alcohol and nicotine, are illegal for adolescents to obtain, possess and use although the legal age for use varies among developed countries. Certain of the negative consequences of substance use for adolescents follow from the illegal nature of, rather than from the actual, use.

Medical consequences

- Accidents such as motor vehicle accidents and trauma in adolescents commonly occur under the influence of substances. This includes driving motor vehicles and even bicycles and skateboards while intoxicated. Alcohol is estimated to be

involved in as many as 40 per cent of adolescent drownings. Adolescents who use and abuse substances are more likely to participate in other risk-taking behaviours such as driving a motor vehicle while under the influence. Adolescent victims and perpetrators of violence, both with and without weapons, are commonly under the influence or have histories of SUDs.

Substance use is often associated with other high-risk behaviours. Early and promiscuous sexual behaviour is common among adolescents with the early onset of substance use and abuse. Among currently sexually active students nationwide, about a quarter had used alcohol or drugs at last sexual intercourse. Overall, male students were significantly more likely than female students to have used alcohol or drugs at last sexual intercourse. The impairment in judgement, and the increase in impulsivity, often produced by an intoxicated state, may result in unprotected and/or unsafe sexual activity and subsequent pregnancy and/or sexual transmitted diseases. Unsafe sexual practices are also common among these populations of adolescents.

- While not all adolescents who use or even abuse substances are considered to be at high-risk status for Human Immunodeficiency Virus (HIV) infection, substance use by an adolescent should prompt a thorough inquiry into specific HIV risk factors and consideration of appropriate testing. Although adolescents constitute a small percentage of the total number of individuals with HIV disease, the often long latency between HIV infection and the onset of symptoms mean that many acquire HIV as adolescents. Intravenous (IV) drug and crack cocaine abusers are among the subgroups of adolescents with the highest risk for HIV.
- Medical complications of substance use are uncommon among adolescents. Long-term sequelae such as liver disease and memory problems from chronic substance use may not become apparent until well into adulthood. Those adolescents who frequently use very high quantities of substance use or who are using acutely toxic substances may be an exception. For example, inhalants such as aromatic or halogenated hydrocarbons or other organic solvents may produce varying levels of neurotoxicity (including acute and chronic encephalopathy) and, on occasion, cardiac arrest secondary to arrhythmia even during the initial episodes of use.

Differential diagnosis and co-morbidity

Given the varying tolerance of various countries and cultures, it is not surprising that the mere use of substances by adolescents might be seen as abnormal or pathological or normative by others. Clinicians need to consider the effect of substance use on the adolescent and his or her family before giving a diagnosis.

Many adolescents with SUDs also have co-existing psychiatric conditions that cannot be described adequately within a single DSM-IV diagnostic category. Some

Table 2.1. Differential diagnoses and co-morbid disorders

Disruptive behaviour disorders
• Conduct disorder
• Oppositional defiant disorder
• Attention-deficit hyperactivity disorder
Mood disorders
• Major depressive disorder: single episode vs. recurrent
• Bipolar disorder
• Dysrthymic disorder
• Cyclothymia
Anxiety disorders
• Social phobia
• Post-traumatic stress disorder
• Generalized anxiety disorder
• Panic disorder
Other disorders
• Schizophrenia
• Bulimia nervosa
Personality disorders

conditions, such as disruptive behaviour disorders (e.g. oppositional defiant disorder, conduct disorder and attention-deficit/hyperactivity disorder, ADHD) and mood disorders (e.g. major depressive disorder and bipolar disorder), co-exist with adolescent SUDs more often than not (see Table 2.1). Knowledge of the assessment and treatment of these disorders is essential for the adequate management of SUDs. In view of the frequency of co-morbidity in adolescents, the clinician should consider, but not be limited by, the practice parameters applicable to both the substance use and the co-morbid psychiatric disorder. However, the optimal treatment of adolescents with SUD and psychiatric co-morbidity involves an integration of treatment modalities rather than merely concurrent or consecutive treatment with specific modalities for either SUD or psychiatric disorder(s).

Significant rates of adolescents with co-existing SUD and psychiatric disorders (disruptive behaviour disorders, mood disorders and anxiety disorders) are reported in both clinical and general populations. Furthermore, the co-morbidity of psychiatric disorders, particularly conduct disorder and, to a lesser extent, major depressive disorder, may have an effect on the alcohol and substance use and related problems both at baseline and at follow-up and impair an adolescent's ability to

engage effectively in treatment. Evidence suggests that depression increases the rate and rapidity of relapse.

Disruptive behaviour disorders are the most common psychiatric disorders diagnosed in adolescents with SUDs. Conduct disorder, including the component of aggression, usually precedes and accompanies adolescent SUDs. Clinical populations of adolescents with SUDs manifest rates of conduct disorder ranging from 50 to almost 80 per cent. Although attention deficit hyperactivity disorder (ADHD) is often present in substance using and abusing youth, the observed association may be due to the high level of co-morbidity between conduct disorder and ADHD. The early onset of conduct disorder, aggressive behaviour, in the presence of attention deficit hyperactivity disorder, may increase the risk for later substance abuse still further. Aggressive behaviours are present in many adolescents who have substance use disorders. The direct pharmacological effects resulting in aggression may be exacerbated further by the presence of pre-existing psychopathology (e.g. ADHD, mood disorders), the use of multiple agents simultaneously and the frequent, relative inexperience of the adolescent substance user. For example, a novice user may be more likely than an experienced user to react to specific psychoactive effects with anxiety. An inexperienced drinker may drink too rapidly, thus causing a rapid rise in blood alcohol level and thus further impairment in judgement and behavioural controls.

Mood disorders, particularly depression, frequently have onsets both preceding and consequent to the onset of substance use and SUDs in adolescents. The prevalence of depressive disorders in these studies of clinical populations ranged from 24 to more than 50 per cent. Substance use disorders among adolescents are also a risk factor for suicidal behaviours, including ideation, attempts and completed suicide. Possible mechanisms for these relationships include acute and chronic effects of psychoactive substances. Adolescents who commit suicide frequently have used alcohol or other substances at the time of suicide. The acute substance use episode may produce transient but intense dysphoric states, disinhibition, impaired judgement and increased level of impulsivity or may exacerbate pre-existing psychopathology, including depression or anxiety disorders. A number of studies of clinical populations show high rates of anxiety disorders among youth with SUDs. In clinical populations of adolescents with SUDs, the prevalence of anxiety disorder ranged from 7 per cent to over 40 per cent. The chronology of the onset of comorbid anxiety and SUDs is variable, depending on the specific anxiety disorder. Social phobia usually precedes abuse while panic and generalized anxiety disorder may more often follow the onset of abuse. Adolescents with SUDs often have a past history or current manifestation of post-traumatic stress disorder (PTSD). Bulimia nervosa is also associated frequently with adolescents having substance use disorders. SUDs

are very common among individuals, especially young and chronically impaired, who are diagnosed with schizophrenia.

Diagnostic instruments

Clinicians often use a variety of rating scales or standardized interviews to assess SUDs and associated problems (see Appendix 2.1). Screening instruments include The Drug Use Screening Inventory – Adolescents (DUSI-A) and the Problem Oriented Screening Instrument for Teenagers (POSIT), which self-administered instruments that tap relevant functional domains of substance use, psychiatric symptoms, school and vocational functioning, family functioning, social competency and peer relations, leisure and recreation, medical problems. More detailed interview instruments tapping the same domains include Adolescent Drug Abuse Diagnosis (ADAD), the Adolescent Problem Severity Index (APSI), the Severity Index (T-ASI), and the Comprehensive Adolescent Severity Inventory for Adolescents (CASI-A). Several brief self-report instruments provide measures of alcohol and substance use severity and consequences in addition to quantity and frequency of use. The Alcohol Dependence Scale (ADS) is a self-report measure of severity of alcohol dependence over the past year. The Rutgers Alcohol Problems Inventory (RAPI) consists of 23 items that assess negative consequences due to alcohol use typically experienced by adolescents. Other comprehensive instruments that measure substance use problem severity as well as a range of psychosocial constructs include the Global Appraisal of Individual Needs (GAIN). The Minnesota Chemical Dependency Adolescent Assessment Package (MCDAAP) includes a structured diagnostic interview, the Adolescent Diagnostic Interview (ADI), a brief screening tool, the Personal Experience Screening Questionnaire and Personal Experience Inventory (PEI), a comprehensive assessment instrument that covers all substances and related problems. It provides a list of critical items that suggests areas in need of immediate attention by the treatment provider and summarizes problems relevant for planning the level of treatment intervention.

Assessment

In evaluating the child or adolescent, the first goal is to determine if a problem in using one or more psychoactive substances exists, what effects substance use has on various domains of the adolescent's psychosocial functioning, and whether such a problem fits diagnostic criteria for substance abuse or dependence. To be considered a problem, substance use must produce some level of dysfunction in one or more domains of the adolescent's life. These domains include substance use and associated problems, psychiatric/behavioural, family, school/vocational, recreational/leisure and medical.

Because of the covert nature of substance use, optimal assessment often requires information from a variety of sources including the adolescent, parents (or other caregivers), other family members, school, any involved social agencies and previous treatment records. Elements of the parent/caregiver interview include the reasons for referral, parental concerns, and details, when known, about the presenting problems (onset, duration, consequences and impairment/dysfunction). Parents may have limited knowledge of their child's substance use patterns, although whenever possible collateral informants are desired. The interview should enquire about the extent of the parent's knowledge of their child's use patterns and negative consequences. Aggressive and suicidal behaviour are common among adolescents with substance use disorders. A treatment history should consist of enquiry into past mental health and SUD interventions, including response to treatment and attitudes about past interventions.

- A comprehensive developmental, social and medical history is a part of any complete assessment involving adolescents. Particularly important is a review of HIV risk factors, including sexual and other high-risk behaviours. Inquiry into these variables can often provide important information into pre-morbid functioning and risk factors for the development of SUDs. The parent/guardian should be able to provide information about a family history of SUDs, family functioning, stressors and supports as well as community resources and risks. Because family involvement forms the cornerstone of many empirically proven treatments, optimal family assessment includes observing how family members, including siblings, interact with one another.
- The interview of the adolescent is much more problematic due to the common desire of the adolescent to deny or minimize problems with substance use and related behaviours as well as the consequences of the substance use. The attitude of the clinician should be non-judgemental and flexible in the order of the interview elements. The clinician may wish to start with less potential anxiety-provoking elements such as enquiry into school, peers and family and delay questions about substance use and other deviant behaviours until later in the interview. The clinician may attempt to 'normalize' use in an attempt to get an accurate report. While using words and concepts appropriate to the adolescent's cognitive, linguistic and emotional development, the clinician should avoid jargon and attempts to appear more like a peer than adult. In clarifying the purpose of the interview/assessment, the clinician should ask about the adolescent's perception of the reason for referral and attitude about the assessment.
- Detailed assessment of the adolescent's substance use behaviour is an essential element of the interview. Enquiry into patterns of use includes information about the age of onset and progression of use for specific substances, quality, frequency and variability of use and the types of agents used. The clinician should ask