Neuropsychiatry of Learning Disabilities

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Abstract

The author reviews almost all aspects regarding learning disturbances, disabilities, classification, up date terminology, the neurobiological basis of learning disabilities, the Genetics of learning disabilities, brain measurements, neurobiological basis of neuropsychiatric comorbidity, associated neuropsychiatric disturbances, medical comorbidity, common pitfalls in the neuropsychiatric assessment of patients with developmental disabilities, neuropsychiatric assessment, expanded neurological examination, general physical examination, laboratory studies, neuropsychological testing, neuropsychological testing, and treatment among others aspects. In conclusion: In this review an attempt has been made to look at learning disabilities as a spectrum of syndromes of developmental deficits. Current terminologies have been defined. The neurobiological nature of learning disabilities has been presented with a review of advances in genetics and imaging studies, with particular emphasis on functional neuroimaging. Neuropsychiatric disorders, their neurobiological basis and medical comorbidities have been reviewed as well.

INTRODUCTION

In general pediatric practice, mental retardation (MR) and other neurodevelopmental disabilities are seen often. About 10% of children are learning impaired and as many as 3% of children have MR. 3-6% of children and adolescents suffer the educational and emotional impact of developmental dyslexia. Non-verbal learning disabilities are estimated to occur in up to 6% of school-age children. Prevalence rates for communication disorders range from 1-13% children. Autistic disorders occur at the rate of 2 to 5 cases per 10000 children (Capute and Accardo 1996, Kaplan, Saddock et al 1994).

The evaluation and management of normal and abnormal neuropsychological development in infancy, childhood and adolescence forms an essential component of pediatric neuropsychiatry. In order to establish a scientific neuropsychiatry, developmental diagnosis forms an important aspect of pediatric medicine. This will form the basis of therapeutic care and prevention.

While using developmental approaches in appraisal of a child one should not ignore the environmental influence. The family comprising of parents and siblings, and extended family, or foster family, the socioeconomic status, cultural milieu, schooling and education, presence of illness, trauma: physical and emotional, each play a significant role. But these must always be considered in relation to the organizational integrity of the child's central nervous system. This ultimately determines how and to what extent the child reacts to his or her environment (Knobloch and Pasamanick 1974).

TERMINOLOGY

Mental Retardation is a heterogeneous disorder. Individuals with mental retardation have below-average intellectual functioning and compromised adaptive skills commencing before 18 years of age. The Association for Mental Retardation has chosen “Mental Retardation” as the preferred term. The World Health Organization (WHO) has recommended the term “Mental Subnormality”. This includes two categories: 1. Mental Retardation – subnormal functioning secondary to identifiable underlying pathological causes. 2. Mental Deficiency – I.Q. of less than 70 which is often used as a legal term. The term “Mental Handicap” has been used in India, “Learning Disability (LD)” in the United Kingdoms and “Oligophrenia” in Russia, Scandinavia and other European countries (Kaplan, Saddock et al 1994).

Learning Disorders are diagnosed when the individual's achievement on individually administered, standardized tests in reading, mathematics, or written expression is substantially below that expected for age, schooling, and level of intelligence. The learning problems significantly interfere with academic achievement or activities of daily living that require reading, mathematical, or writing skills (APA 1994).

Dyslexia is one of several distinct learning disabilities. It is a specific language-based disorder of constitutional origin characterized by difficulties in single word decoding, usually reflecting insufficient phonological processing abilities. These difficulties in single word decoding are often unexpected in relation to age and other cognitive and academic abilities; they are not the result of generalized developmental disability or sensory impairment. Dyslexia is manifest by variable difficulty with different forms of language, often including, in addition to problems reading, a conspicuous problem with acquiring proficiency in writing and spelling (Coffey and Brumback 1998).

Nonverbal Learning Disabilities refer to developmental disorders of motor function (developmental coordination disorder), visio-spatial processing, mathematics (dyscalculia), memory, prefrontal executive function, and social-emotional cognition and behavior (Kaplan, Saddock et al 1994, Coffey and Brumback 1998).

Communication Disorders are developmental speech and language disorders which include expressive language disorders, mixed receptive-expressive language disorders, phonological disorder, stuttering and other unspecified communication disorders (Kaplan, Saddock et al 1994).

Pervasive Developmental Disorders (PDD) are characterized by severe and pervasive impairment in several areas of development: reciprocal social interaction skills, communication skills, or the presence of stereotyped behavior, interests and activities. The impairments are qualitatively distinct and deviant relative to the individual's developmental level or mental age. The conditions include Autistic Disorder, Rett's Disorder, Childhood Disintegrative Disorder, Asperger's Disorder and other unspecified pervasive developmental disorders (APA 1994).

NEUROBIOLOGICAL BASIS OF LEARNING DISABILITIES

The developing brain undergoes multiple changes. The “wiring” of the brain involves the formation of trillions of connections between the neurons of different parts of the brain. Both nature and nurture play important roles in the development of this complex system. The precision of this development process can sometimes indeed go wrong. Causative factors in learning disabilities include genetic which can be chromosomal or inherited conditions; intrauterine factors like maternal malnutrition, exposure to irradiation, TORCH infections, substance abuse; disorders of cerebral dysgenesis and inborn errors of metabolism; perinatal causes like placental insufficiency, prematurity, complications of labor and delivery; postnatal causes like CNS damage due to trauma, infections, malnutrition, resistance to thyroid hormones, abuse, neglect, toxin exposure, uncontrolled seizures and neurodegenerative disorders (Kaplan, Saddock et al 1994, Coffey and Brumback 1998).

THE GENETICS OF LEARNING DISABILITIES

Twin studies, sibling analysis and family pedigree analysis have shown a genetic basis for learning disabilities. For example, twin studies have shown that if one twin has reading disability, the probability of its occurrence in the other twin is 68% for monozygotic twins and 40% for dizygotic twins. Familial transmission is known to occur. For example, if there is family history of reading disabilities the probability of its occurrence is significantly increased. The relatives of children with learning disorders have a relatively high incidence of expressive language disorder. In many syndromes which are associated with learning disabilities, like Down syndrome, Fragile X syndrome, Williams syndrome, Prader-Willi Syndrome the chromosomal abnormality is known. Clinical studies and reports suggest that the nonautistic members of the families share various language and other cognitive problems with the autistic person but have them in a less severe form (Kirchhoff M, Gerdes T et al 2005, Fiedorowicz C 1999).

BRAIN MEASUREMENTS

Many sophisticated techniques are now available to measure the structures and also the functions of the brain. Neuroimaging techniques include computed tomography (CT) scan, magnetic resonance imaging (MRI), positron emission tomography (PET), regional cerebral blood flow (rCBF), single photon emission computed tomography (SPECT), functional magnetic resonance imaging (fMRI). Electrophysiological methods include electroencephalography (EEG), event related potentials (ERP) and averaged evoked potentials (AEP). Neuropsychological assessments evaluate brain/behavior relationships. Neuroanatomical studies include autopsy studies.

Reading disabilities form the most common and frequently identified type of learning disability. Recent important research has focused on reading disorders. Neuroimaging studies and postmortem findings have indicated that there are asymmetries in the normal brain. These asymmetries are expected variations and are considered normal. About 70%

of normal brains show left > right asymmetry in the posterior region and left < right asymmetry in the anterior region. In subjects with learning disabilities this asymmetry is not found (Hynd GW, Semrud-Clikeman M 1990).

Studies on normal subjects have found that the planum temporale in the left hemisphere is typically larger than its fellow in the right hemisphere. In subjects with reading disability they are found to be of the same size. CT scans of the occipital lobe have shown asymmetry of the occipital poles in normal subjects and symmetry in subjects with learning disability. MRI studies have shown that subjects without LD have a leftward asymmetry in the angular gyrus of parietal lobe, whereas this is absent in subjects with LD. Electrophysiological studies show that there is less electrical activity in the parietal lobe in subjects with LD compared to subjects without LD (Fiedorowicz C 1999).

More recently functional brain imaging techniques are proving to be very useful in understanding functional activation patterns characterizing neuropsychiatric syndromes. Here the brain activity is measured using PET, rCBF, SPECT or fMRI while the subject is engaged in a mental task like reading. Levels of brain activity or activation can be examined through regional cerebral blood flow or regional cerebral glucose metabolism. Results of functional imaging studies in learning disabled subjects suggest that the functional abnormalities are not limited to specific regions, but distributed over the cortex. Any specific cognitive activity as for example reading has a widely distributed neural network connectivity. Hence deficits in any part of the network may yield the symptoms of a specific learning disability including some of the unique behavioral-emotional characteristics that often accompany such disabilities.

Neuropsychological assessments include a variety of tests of abilities and functions in the domains of cognitive/intellectual, language, visual-perceptual, academic, motor, sensory, and emotional/behavioral. A correlation is then drawn between a profile of strengths and weaknesses and known brain functions. Deficiencies in language/verbal learning, reading, written language, verbal reasoning, verbal memory, arithmetic computation, and processing speed have been associated with left hemisphere dysfunction. Deficiencies in spatial function, nonverbal reasoning, nonverbal cues, social skills, and social/emotional information have been associated with right hemisphere dysfunction. Structural and functional abnormalities in the medial geniculate nuclei have been associated with phonological processing deficits which have been identified as the primary difficulty in subjects with language and reading disabilities (Coffey and Brumback 1998).

Young infants depend to a large extent on right hemisphere circuits in their interactions with their caretakers. In order to function well they must be able to perceive the mother's responses. During the first three years of life, there is a preponderance of cerebral blood flow in the right hemisphere. In the fourth year this preponderance shifts to the left posterior hemisphere. This shift correlates with the shift from an early emphasis on visiospatial abilities to an emphasis on language and sensorimotor activity later in childhood. Any damage or dysfunction of the ‘wiring' or neural circuitry or disruption of sequential acquisition of social information may lead to non-acquisition of normal emotional gestural output. The infant's feedback comes from the social responses from those around her/him. She/he must be able to perceive and interpret those signals. Acquired brain lesions can affect social-emotional perception and behaviors in adults as well as in children. But lesions very early in development occur during a time of considerable neural plasticity. Therefore there is considerable scope for functions to be regained. But to what extent early lesions damage ‘phyletic memory stores' i.e, inborn, species-specific memories, and to what extent such damage, given the potential for plasticity, impact later development, is not clear. What is apparent is that many of the developmental syndromes seem to result from genetically programmed cerebral dysgenesis (Ahn and Jeong et al 2006, Coffey and Brumback 1998).

Future research questions would relate to what mechanisms underlie the genetically programmed affections of developmental deficits. As data are generated from the human genome project, it may become increasingly possible to relate gene defects to specific neurocognitive syndromes. These disorders appear to be polygenic and so it is likely that the definition of specific phenotypes will be facilitated by understanding the genetic lesion. The multifactorial mechanisms controlling neuronal migration, neurotransmission, gene expression, etc., may become more comprehensible with advances in this field of molecular neuroscience.

NEUROBIOLOGICAL BASIS OF NEUROPSYCHIATRIC COMORBIDITY IN LD

The presence of brain dysfunction in childhood is associated with a greater risk for the development of neuropsychiatric

disorders much more than with physical handicaps. The effects may be direct or indirect. The effects can persist and impede the long-term adjustment of the child. There are many mechanisms by which brain dysfunction may lead to psychopathology. The relative merit of each is not certain but they are certainly not mutually exclusive in their contributions. The mechanisms include the following (Coffey and Brumback 1998):

1. Behavioral disruption that arises directly from abnormal brain activity
2. Heightened exposure to failure, frustration, and social stigma because of associated disabilities
3. The possible effects of brain damage or anomalous neurodevelopment on subsequent temperament and personality development
4. Adverse family reactions, ranging from overprotection to scapegoating
5. The individual's own reaction to being handicapped and its effect on his or her actual capacity to cope and compete
6. Possible adverse effects of the treatment themselves (e.g., lack of and/or poor treatment) that may restrict normal activities and socialization

NEUROPSYCHIATRIC DISTURBANCES IN LD

Difficulties in psychosocial adjustment appear to be the major social-emotional manifestations of learning disabilities (Pearl 1992). Children with LD experience less acceptance, lower popularity, more peer rejection, and increased neglect by peers than do normally achieving children or low-achieving peers (LaGreca et al 1988). In a study, special education teachers indicated that nearly 40% of their students required social skills training and the difficulties tend to increase with age (Baum et al 1988). The social skill deficits that begin in childhood persist into adulthood (Polloway et al 1984).

In addition to low self-esteem, social skills deficits and general psychosocial adjustment difficulties, many children with LD experience more serious psychopathology or seek psychiatric help. In one of the most comprehensive long-term follow-up study of children with LD, it was found that at about age 18 years the LD group with brain impairments reported less satisfaction in their lives. They were viewed as more impulsive, showed increased social-emotional disturbances and antisocial behaviors. In a second follow-up at around age 25 years LD adolescents experienced more maladjustments and reported more depression, acting-out tendencies, social insensitivity, obsessions and compulsions, phobias, social withdrawal and disorganized thoughts. Gender differences were also noted with more affective or mood disruptions in females and problems with behavioral dyscontrol and thought organization in males (Spreen 1988).

It has been estimated that 30-70% of children with LD will experience ongoing comorbid symptoms of attention-deficit/hyperactivity disorder (ADHD) as they enter into adulthood (Bellak and Black 1992). Both LD and ADHD have a high degree of comorbidity with other neuropsychiatric disorders like depression, conduct disorder, anxiety disorder, substance abuse, Tourette syndrome, tic disorders and other stereotypic movement disorders, and sleep disorders. Schizophrenia may have a prevalence of 3% in individuals with MR compared to 0.8% in the general population. Bipolar illness has a 2 to 3-fold greater prevalence in the cognitively impaired than in the general population. Self-injurious behaviors require treatment in 3-15% particularly in the severe range of MR. MR with comorbid epilepsy confers up to 56% risk of comorbid psychiatric illness (Isle of Wight Study). Studies have shown that a large number of individuals with LD seek mental health services. A survey showed that at least 50% of adults with LD wanted and/or needed psychiatric services for more severe emotional problems (Chesler 1982). These facts stress the need for screening, early detection, recognition, comprehensive assessments and early intervention or even incorporate prevention strategies and thus improve the quality of life of the affected individuals.

MEDICAL COMORBIDITY IN LD

The prevalence of general medical conditions is increased especially in the more severely cognitively impaired. In up to 50% of individuals with MR, occult visual and auditory deficits occur. 1 in 5 individuals with MR have cerebral palsy (CP). Up to 20% of individuals suffer from seizures. Gastrointestinal complications can occur including feeding difficulties, excessive drooling, reflex esophagitis and constipation. Genitourinary problems include urinary incontinence and poor menstrual hygiene. There is a significant increase in the rates of transmissible diseases like sexually transmitted diseases (STDs), hepatitis B, and Helicobacter pylori infection in people with MR. The life expectancy for the mildly cognitively impaired is not different from that of the general population. Individuals

with severe to profound cognitive impairment experience a decreased life expectancy. This is related to the etiology or associated neurological disorders like epilepsy. Neurological dysfunctions life immobility, oral motor incoordination, dysphagia and aspiration pose greater risk for premature death. Neurological dysfunction with other organ system anomalies can further shorten the lifespan of the individual. Respiratory disease is the most common cause of death among individuals with profound disability (Volkmar and Lewis 1996).

COMMON PITFALLS IN THE NEUROPSYCHIATRIC ASSESSMENT OF PATIENTS WITH DEVELOPMENTAL DISABILITIES

We have noted earlier that 30-70% of individuals with developmental disabilities also suffer from neuropsychiatric disorders. Referrals for neuropsychiatric evaluation are often requested for “behavioral” symptoms like hyperactivity, disorganized behavior, aggression, destructiveness or self-injurious behaviors. Neuropsychiatric assessments rely heavily on verbal reports from carers and significant others and observations. Developmentally disabled children are unable to communicate or clearly define the nature of their discomfort and can present with “behavioral” symptoms. The clinician has to observe and infer the common ground between temperamental variation, developmental levels, adaptability and vulnerability to change. A theory of mind has to be constructed based on this modified database. This can sometimes generate anxiety, uncertainty and confusion (Coffey and Brumback 1998).

Diagnostic overshadowing: This refers to attributing symptoms to the developmental disability regardless of the cause. E.g., Aggressive behavior attributed to LD. It is necessary to evaluate each symptom as such for effective management.

Diagnostic presumption: It is presumed that the symptom is a result of complication of learning disability. E.g., acute confusion in Down syndrome may be attributed to dementia, rather than another treatable cause. It is therefore essential to systematically rule out all components of the differential diagnosis.

Premature pharmaculation: This means prescribing medication for an isolated behavioral symptom. E.g., prescribing an antipsychotic drug for aggression without examining other options. It is important to evaluate the pattern of symptoms before arriving at a diagnostic formulation and a treatment plan.

Organicity trap: This involves attributing all neuropsychiatric symptoms to unspecified ‘organic' disorder. It is possible to avoid falling into this trap by defining the specific nature of the neuropsychiatric dysfunction. E.g., prefrontal executive dysfunction, obsessive compulsive disorder, panic disorder, etc.

Diagnostic overgeneralization: Here a behavior is treated as a “symptom”. This requires thorough evaluation of the functioning of the individual. A significant change in the behavior or appearance of new behaviors should be looked for.

Diagnostic undergeneralization: The common error is to use an isolated symptom as equivalent to a diagnosis. E.g., an Attentional problem equals ADHD. A complete and formal neuropsychiatric diagnostic evaluation is essential.

The art of developmental neuropsychiatry often lies in merging strategies from different conceptual models like the behavioral-learning model, psychosocial model and medical-syndromic model and providing comprehensive assessments and management strategies.

NEUROPSYCHIATRIC ASSESSMENT

In addition to obtaining a standard neuropsychiatric history, it is often necessary to refer the child for a pediatric medical evaluation. Many individuals cannot clearly indicate the nature of their discomfort. Irritability, aggressive or self-injurious behaviors can be seen in otitis, dental pain, urinary tract infection, migraine headaches or gastritis. Hypothyroidism and epilepsy are often associated with MR and appropriate treatment may improve function. It may be necessary to modify the interview technique to accommodate for communication deficits. It is very important to obtain as much information as possible from parents, caregivers and teachers about past and present functioning of the child in his or her social environment. The mental status examination is a critical component of the evaluation. The body language is read carefully. The clinician may also make use of standardized and comprehensive instruments for diagnostic assessment and also rating scales (Volkmar and Lewis 1996).

EXPANDED NEUROLOGICAL EXAMINATION

This includes assessments of head growth, muscle tone, strength and coordination, deep tendon reflexes, primitive reflexes, pathological reflexes, ataxia, unusual motor

slowing, focal and lateralized deficits, abnormal movements like tics, dystonia and athetosis. Cerebellar function and reaching movements through space can be assessed. In children with early lesions standard sensorimotor abnormalities may not be necessarily present even when there is a lateralized lesion. Motor skills should be evaluated across a number of areas like conceptual level (praxis), comprehension of pantomimes and emblems (e.g., thumbs-up sign), finger movement skills (rapidity, ability to isolate individual finger movements, ability to prehend). An evaluation should be made for subtle or soft signs. There is no single examination for eliciting subtle signs, but a number of overlapping examinations are performed such as, lateral preferences, stressed gaits, gait/steadiness, sustentation postures/stations, finger to nose, tongue protrusion, maintaining eye closure, balance, hopping, and timed coordination. Assessments should be made for visual impairments like refractive errors, strabismus, amblyopia, cataract, abnormal retinal pigmentation and cortical blindness. Hearing defects should be looked for. Sensations have to be tested as many subjects have hypo- or hyper-sensitivity or altered sensations. A cognitive screen using an instrument like the modified mini mental state examination should be performed. Lastly, tests for prefrontal executive functions should be performed (Coffey and Brumback 1998, Kaplan, Saddock et al 1994).

GENERAL PHYSICAL EXAMINATION

This includes the vital parameters of pulse, temperature and respiratory rate. The height, weight and blood pressure are recorded. Dysmorphic features both gross and subtle and congenital anomalies should be looked for. Cutaneous findings of etiological interest include hypo- or hyper-pigmented macules, fibromas and irregular pigmentations. Major organ system anomalies may direct further investigations.

LABORATORY STUDIES

No laboratory investigation is routine in evaluating a case of LD. The clinician determines the type and extent of investigations to be performed based on the history and examination. DNA analysis for FraX (Fragile X) promoter region should be ordered for all prepubertal males with MR, especially if autistic features are present. Karyotyping of 500 band resolution level should be considered for all children with MR. Chromosomal abnormalities are seen in as many as 50% of children with MR. Sex chromosome aneuploidy is seen in up to 5% of children with mild MR or learning disability. Fluorescence in situ hybridization (FISH) probe is ordered when clinically indicated in the following: Prader-Willi/Angelman syndrome, Smith-Magenis syndrome, CATCH 22, Williams syndrome, Wolf-Hirschhorn syndrome, Cri Du Chat syndrome, Langer-Giedion syndrome, Miller-Dieker syndrome. Tests for inborn errors of metabolism are ordered as clinically indicated. Thyroid function test and creatine kinase are requested if indicated. Brain imaging should be performed in any child with neurological findings and facial dysmorphism. MRI is generally preferred over CT scan, because MRI has greater resolution and enhanced detection of abnormalities in the progression and timing of myelination and demyelination, and heterotopic gray matter. Skeletal films may assist in growth assessment, syndrome characterization and phenotypic description. EEG is done if clinically warranted. Audiometry and auditory evoked potentials are performed in the context of audiological assessments. Visual evoked potential may be needed to detect cortical blindness (Kirchhoff and Gerdes et al 2005, Capute and Accardo 1996).

NEUROPSYCHOLOGICAL TESTING

Standardized tests are administered by a clinical psychologist. Intellectual functioning can be measured yielding an intelligence quotient (IQ). Commonly used tests are Bayley Scales of Infant Development, Weschsler Preschool and Primary Scale of Intelligence – Revised, Weschsler Intelligence Scale for Children – IV, Weschsler Adult Intelligence Scale, Binet-Kamat Intelligence Scale, Peabody Picture Vocabulary Test which can overcome the language barrier for the children with communication difficulties. Copying geometric figures, Goodenough Draw-a-Person Test, Kohs Block Design and geometric puzzles may be used as screening tests for visual-motor coordination. Assessments of adaptive functioning can be done with Vineland Adaptive Behavior Scales. Specific tests to detect specific learning disabilities are available. The Bender Visiomotor Gestalt Test and Benton Visual Retention Test can be used for detecting brain damage. Lobe functions can be determined by neuropsychiatric test batteries like the Luria-Nebraska Neuropsychological Battery. Assessments for communication and language disorders are performed by the speech and language pathologist (Kaplan, Saddock et al 1994).

PHYSIOTHERAPY AND OCCUPATIONAL THERAPY ASSESSMENTS

The nature and degree of sensory and motor deficits need to be ascertained and appropriately rehabilitated to improve

and maintain the patient's functioning. Assessments also have to be done to suitably manipulate the environment through prostheses and other modifications to enhance the functionality and independence of the patient.

SOCIAL ASSESSMENTS

Ideally a social worker would be involved in assessing the patient's family and social situation, social support, meals, physical safety and security, home environment, financial resource and planning, recreation, vocational opportunities and provide necessary advice and support.

TREATMENT

All the facts and diagnostic studies are assembled and reviewed. A set of diagnoses and diagnostic formulations are generated. It is important to take into account the child's areas of strengths and areas of weaknesses. The strengths have to be capitalized on and the weaknesses have to be supported or strengthened. An individualized treatment plan is developed. Treatment should be multimodal and judiciously utilize medical intervention, psychopharmacological treatment, behavioral management, physiotherapy, psychotherapy, counseling, remedial teaching or vocational training, Montessorie technique in preschoolers especially with developmental coordination disorder, group therapy, family education, counseling and therapy, psychosocial management, environmental prostheses, all to improve and maintain optimum functioning, limitation of disability and rehabilitation. Parent support groups and advocacy organizations have a very important role to play. A multidisciplinary approach has to be taken to provide the most effective and efficient therapeutic care. The team would ideally comprise of developmental pediatrician, neuropsychiatrist, psychologist, remedial teachers, physiotherapist, occupational therapist, speech and language therapist, social workers, nurse counselor/therapists. Other professionals may need to be consulted depending on the nature of the problem.

Psychopharmacological preparations have to be used very carefully. The dosages have to be titrated carefully, since the window between therapeutic effect and overdosage may be narrow. The already compromised brain is sensitive to the adverse effects of these drugs. Some of the drug treatments that have proved useful are lithium, naltrexone, carbamazepine, valproic acid, clonidine for controlling aggression and self-injurious behavior; risperidone, haloperidol, chlorpromazine for stereotypical motor movements; propranolol, buspirone for explosive rage behavior; methylphenidate, clonidine for ADHD. Certain medications can produce behavioral side effects e.g., H2 blockers causing irritability, decreased concentration, psychosis; diuretics causing depression, emotionality; Phenobarbital causing hyperactivity, depression of cognition, depression and emotionality. When these occur, alternative choices with no neuropsychiatric side effects can be used (Coffey and Brumback 1998, Kaplan, Saddock et al 1994).

Food fads, vitamin and mineral therapies, nutritional supplements, although have gained popularity, their efficacy has not been well established from clinical trials (Volkmar and Lewis 1996).

MEDICO-LEGAL ISSUES

- The majority of individuals with LD including those in the range of mild MR, are capable of making appropriate legal decisions when adequately and appropriately informed of the decision outcomes.
- Some individuals may not be capable of comprehending the long-term implications of the medical or legal matter at hand. In such cases, the decision is best made by a member of the biological family; the family member must obtain guardianship status for power of attorney over these matters. If a family member is unavailable to serve as guardian, then a guardian ad litum can be assigned by the court for assistance in such legal and medical matters.
- Complex decisions, particularly those involving issues of sterilization, are perhaps best handled by a committee with the assistance of the ethics committee of the involved medical institution.
- Failure to identify a genetic cause of MR with risks to other family members or risks to the patient for future medical complications is a potential medical/legal pitfall.

CONCLUSION

In this review an attempt has been made to look at learning disabilities as a spectrum of syndromes of developmental deficits. Current terminologies have been defined. The neurobiological nature of learning disabilities has been presented with a review of advances in genetics and imaging studies, with particular emphasis on functional

neuroimaging. Neuropsychiatric disorders, their neurobiological basis and medical comorbidities have been reviewed. The review of neuropsychiatric assessments deliberately begins with a discussion on the pitfalls highlighting the special difficulties encountered and the need for care and caution. Physical examination with an emphasis on the expanded neurological examination has been presented. Relevant laboratory tests are briefly reviewed. The importance of psychological testing along with assessments of communication abilities, activities of daily living and social needs have to be emphasized as they form an important basis for planning appropriate intervention. This is followed by a brief review of the treatment with its emphasis on a multimodal and multidisciplinary approach. The review concludes with a brief note on common medico-legal issues. The long term nature of the conditions requires a life span view. The ever changing nature of the problems in the learning disabled child places an increasing emphasis on the field of growth and development. Therefore there is a need here to focus on the life cycle of the child using normal progressions of this cycle as a basic frame of reference. This enables us to view the child as a unique individual with unique abilities and potentials, and integrate all aspects of her/his development.

References

1. Ahn KJ, Jeong HK, Choi HS: DYRK1A BAC transgenic mice show altered synaptic plasticity with learning and memory defects. Neurobiol Dis 2006 Jan 30.
2. American Psychiatric association (APA): Diagnostic and stastical Manual of Mental Disorders (DSM-IV). American Psychiatric Association. Washington, DC. 1994.
3. Baum DD, Duffelmeyer F, Geelan M: Resource teacher perceptions of the prevalence of social dysfunction among students with learning disabilities. Journal of Learning Disabilities 21: 380-381, 1988.
4. Bellak L, Black RB: Attention-Deficit Hyperactivity Disorder in Adults. Clin Ther 14: 138-147, 1992.
5. Capute AJ, Accardo PJ: Developmental Disabilities in Infancy and Childhood. Vol 1 and 2. Baltimore: Paul H Brookes; 1996: 1-619 and 1-521.
6. Chesler B: ACLD Vocational Committee survey on LD adults. ACLD Newsbrief 145, 1982.
7. Coffey CE, Brumback RA: Textbook of Pediatric Neuropsychiatry. Washington, DC: American Psychiatric Press, Inc. 1998.
8. Fiedorowicz C: Neurobiological Basis of Learning Disabilities: An Overview. Linking Research to Practice: Second Canadian Forum Proceedings Report. Canadian Child Care Federation. November 1999.
9. Hynd GW, Semrud-Clikeman M, Lorys AR, et al: Brain Morphology in Developmental Dyslexia and attention Deficit/Hyperactivity Disorder. Arch Neurol 47:919-926, 1990.
10. Kaplan HI, Sadock BJ, Grebb JA: Kaplan and Sadock's Comprehensive Textbook of Psychiatry. Seventh Edition. Maryland: Williams & Wilkins. 1994.
11. Kirchhoff M, Gerdes T, Brunebjerg S: Investigation of patients with mental retardation and dysmorphic features using comparative genomic hybridization and subtelomeric multiplex ligation dependent probe amplification. Am J Med Genet A 2005 Dec 15; 139(3): 231-3.
12. Knobloch H, Pasamanick B: Gesell and Amatruda's Developmental Diagnosis: The Evaluation and Management of Normal and Abnormal Neuropsychological Development in Infancy and early Childhood. Third Edition. Maryland: Harper & Row Publishers 1974.
13. LaGreca AK, Stone WL, Halpern DA: LD status and achievement: confounding variables in the study of children' social and behavioral functioning? Paper presented at the annual meeting of the International Academy for Research in Learning Disabilities (IARLD), Los Angeles, CA, February 1988.
14. Pearl R: Psychosocial characteristics of learning disabled students, in Learning Disabilities: Nature, Theory, and Treatment. Edited by Gaddes W, Edgehill D. New York, Springer-Verlag, 1992.
15. Polloway EA, Smith JD, Patton JR: Learning Disabilities: an adult development perspective. Learning Disability Quarterly 7: 179-186, 1984.
16. Rutter M: Issues and prospects in developmental neuropsychiatry, in Developmental Neuropsychiatry. Edited by Rutter M. New York, Guilford, 1983.
17. Spreen O: Learning Disabled Children Growing Up: A Follow-Up Into Adulthood. New York: Oxford University Press, 1988.
18. Volkmar FR, Lewis M: Mental Retardation: Child and Adolescent Psychiatric Clinics of North America. Philadelphia: WB Saunders Company 1996; 5: 769-993.

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