CHILDREN’S SOCIAL RELATEDNESS:
AN EMBODIED BRAIN PROCESS
A clinical view of typical development and disorders

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The subject of this book is the infant’s and young child’s social relatedness, including together the relationship between attachment and theory of body, speech-language, and theory-of-mind function, as well as the disorders that can occur.

The authors have sought to integrate the developmental neurosciences and bring them closer to the people who diagnose and treat children with contact, communication and social-relatedness disorders. They are convinced that a developmental neurobiological approach together with careful clinical examination and knowledge of current findings in neuropsychology are indispensable. It has become clear that child psychiatrists and neurologists, as well as child neuropsychologists and psychotherapists, need information about the relationship between brain and behaviour in a practical clinical context.

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The three authors’ focus of interest is developmental psychology, neurology and neuropsychoanalysis.
Neuropsychoanalysis is a rapidly expanding field. It provides information about normal as well as pathological development. Although I have a limited grasp of neurology, I found the reading of this volume extremely rewarding and am sure that both professionals and others working with children or interested in child development will benefit from consulting it.

Why and how do disorders develop? Integration of the different behavioural and biological sciences (fields with over a century of research and experience to draw on) is the key to better understanding babies and young children. As the book explains, the differences between a developing brain and an adult brain need to be understood to make possible prevention and early intervention, the direction I believe mental health care will have to take as interventions later in life are more cumbersome, more costly and often less successful.

The authors argue that we need to ask not only where in the brain a process is located, but also how brain systems interact. They explore subjects like Theory of Body and Theory of Mind and explain how attachment, the earliest form of social relatedness and mentalization becomes possible, how the child learns to understand its own mind and other people’s mental processes. The importance of milieu is taken into account and the reader is shown the extent to which the conscious and unconscious mind is a culturally constructed reality.

Recent discoveries, like the role played by mirror neurons in learning new behaviour, are explained: ‘Infants can appreciate the meaning of complicated expressions in language before their expressive skills are ready.’ The authors show that the mind is basically relational and deal with some of the practical consequences of this, for example that a child will not learn to speak by watching television.

They address issues of interest to professionals, such as how the different forms of autism develop and how they can be diagnosed. They caution against medicating children, arguing that this can happen if a child is labelled as mentally ill without an effort having been made to understand its mind and its environment. This is valuable contribution to our approach to treatment.

The focus is on mind and brain, nature and nurture and their constant interaction from birth onwards. Some quotes: ‘Nature and nurture are not mutually exclusive antagonists but dialectically opposing entities that act in unity.’ ‘The interplay between genes and environment starts just after conception and continues throughout life, shaping the brain.’ ‘Genetic causes of mental illness have been over-emphasized at the expense of paying attention to environmental factors.’ ‘Organisms have a purpose and genes do not.’

As a psychologist and child psychoanalyst with many years of experience, I can warmly recommend this rich ‘encyclopaedia’ to anyone working with children or interested in child development.

Hendrika C. Freud
A guide to the book

**Children's social relatedness: an embodied brain process**
A clinical view of typical development and disorders

**Part I**
The Introduction to the book gives an overview of brain-behaviour relationships and argues that the professionals working with children of the need to know about neuroscientific thinking.

**Part II**
Part I is an introduction to embryology and plasticity, the survival and emotion systems, cognition and the enculturated brain, neurogenetics, and the neural background of developmental disorders.

**Part III**
Part II addresses the principles of development and emergent social relatedness, i.e. attachment. Normal development is discussed. Early speech development and body language are described as are speech disorders and lacking social relatedness, i.e. early autism.

**Part IV**
Part III addresses disorders in the young child after the toddler stage: developmental dysphasia and autism and their differentiation. An important issue is the ability to mentalize, the emergence of a Theory of Mind.

Part IV How are approaches to treatment influenced by the therapist's concepts of the patient's disordered mind and underlying body-brain entity? What treatment should be chosen, why? Who is likely to benefit from a specific treatment. Which treatments are available and what is the rationale for each one?
Foreword

Introduction

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2 Neural bases of cognitive functions C. Njiokiktjien
3 Behavioural genetics B. Korkmaz
4 Models of abnormal brain function and developmental disorders C. Njiokiktjien

Part II: Infant and toddler
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6 Early relatedness: attachment and theory of body C. Njiokiktjien
7 Developmental kinesics, related phenomena of non-verbal interaction, and disorders C. A. Verschoor
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Introduction

**Sections** Intro-1, Intro-2, and Intro-3

1. All three authors are clinicians: Korkmaz and Nijokiikjien are child neurologists interested in developmental science, cognitive neuroscience and psychoanalysis; Verschoor is a child neuropsychologist and occupational therapist interested in motor, speech and emotion development.

2. Our emphasis is on the neurological aspects of development. Embryology is therefore addressed, as is brain plasticity and recovery processes after brain damage and/or developmental disorders (ch. 1). We also discuss theories regarding synaptogenesis (ch. 1.7) as well as those which have attempted to explain integrated and coherent functioning, and concepts such as neuroconstructivism (ch. 4.2.2). As our focus is on social relatedness, attachment, Theory of Body (ch. 6.3) and Theory of Mind (ch. 10), disorders such as autism (ch. 9 & 11), developmental, language disorders (DLD) (ch. 8 & 12) and disorders in body language (ch. 7) are also addressed.

3. The writings of the American neurologist Oliver Sachs, a pioneer in this field, are an example. Less known are books on development by renowned scientists, e.g.: *Diary of a baby* by Daniel N. Stern (1998), and *The philosophical baby – What Children’s Minds Tell Us About Truth, Love, and the Meaning of Life* by Alison Gopnik (2009).

4. Solms has written on consciousness and the neuropsychology of dreams (2005). He often refers to Panksepp (1998), whose research dealt with the neuroscience of emotions and affects. Evolution has led to the human brain, enabling culture and social relatedness a.o. by mentalizing, in order to cooperate and survive as a species (fig. 1). ‘Cogito, ergo sum’ means that the mind is a product of our consciousness; it became a subject of investigation. In ch. 6.4 we address Metzinger’s (2004) thesis that the phenomenal self is not a thing, but a process, a product of brain activity.

**The Objectives of this Book**

**Intro-1.** In writing this book, the authors (note 1) wish to raise interest in developmental neuropsychiatry among the wide range of professional people—child neurologists, psychiatrists and psychologists, clinical geneticists and pediatricians—who diagnose, treat and teach infants, toddlers and young children with developmental neurological, neuropsychiatric and associated behaviour disorders. It is hoped the book will be of use to professionals such as neuropsychologists, occupational, speech-language and motor therapists, and will enable them to view their work within a developmental-neurological framework. This would facilitate teamwork among representatives of the various professions by fostering a common terminology and shared basic assumptions in regard to development per se and developmental problems.

We therefore describe typical development. Many authors see this as a mere phenomenon and/or process, and fail to deal with the related developments in brain functions and the neural changes involved (note 2).

The following are some of the developments in the (clinical) neurosciences and in other fields which have increased the need for a multidisciplinary book:

- **Early development is cascade-like and without modularization.** In typical children early development is characterized by the coming into being of attachment to the caretaker, speech-language, and motor development, including praxis. In children these function domains are not completely separated, nor are they separated from affects, social relatedness and mentalizing abilities (theory of mind) (ch. 10). This is the main reason why a so-called ‘pure disorder’ is rare; in most children with a developmental disorder there is a certain degree of co-morbidity (ch. 4.2.2).

Moreover, early development of attachment, speech-language and motor development is cascade-like: conditions for optimal attachment contribute to learning in other domains, e.g. speech, optimal attachment and speech-language contribute to mentalizing etc. (ch. 8.3.1).

- **Scientists in related and allied disciplines have become interested in neuroscience and there is a wide variance in emphasis in the research being done in different domains, which have remained disconnected from one another. More collaboration and better communication between researchers with different backgrounds, working in different disciplines, is urgently needed in order to develop a converging, interdisciplinary approach.** A review of the available literature on this issue is provided.

- **The publication of books on neurological syndromes for non-scientists has increased interest among the general public (note 3) . after fig.1**

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On the right evolution of the brain in the animal kingdom—from lower to higher vertebrates (mammals/primates)—is shown bottom-up.

All animals with nervous systems have neurons and networks, which follow the same principles [see subscript]. All vertebrates have a central nervous system in which a spinal cord, brainstem, mesencephalon (midbrain), rhinencephalon (limbic brain) and telencephalon can be distinguished.

**NEW IN THE PRIMATE BRAIN** is the increase of the telencephalon and in humans an integration of these brain parts by evolution as well as functionally by enculturation (environmental constraints and education) (Lende & Downey, 2012). The limbic brain contains emotion systems for survival, active from birth [ch.2.1.1].

**NEW IN THE HUMAN BRAIN** is an increase in neocortex, notably prefrontal cortex, with an enormous surface [see ch. 1.5]. Neural networks are organized into neural systems, e. g. the sensory-motor system for actions. Brain hemisphere function is asymmetric: the left side is specialized in formal and sequential function (syntax) for praxis and speech. There is a mirror neuron system for action understanding and prediction.

Action, language, memory and emotion systems as well as (self-)consciousness require a coherent cooperation through neuronal connections (Northoff, 2008; Singer, 2001) of many brain areas in a time course, which von Monakov (1914) called ‘chronogenic synthesis’ [ch. 4.2.10].

A change in inter-area cooperation is a reversal of the preferred direction of information flow from the caudo-rostral bias characteristic of non-human brain dynamics to a rostro-caudal bias characteristic of in the highest association regions of the neocortex, producing the frontal feedback system (Noack, 2012).

Humans can communicate using speech and body language and can understand other people via theory-of-body and theory-of-mind functions—they ‘mentализ’ Actions and language as well as refined emotion systems for mentalization make CULTURE possible. This takes place on the basis of SOCIAL RELATEDNESS, EDUCATION, and COGNITION.
The term ‘neurophilosophy’ wrongly suggests that what is concerned are vague notions and that serious attempts to introduce neuroscience into the study of problems of the mind are marginal to philosophy and noncommittal. Far from being exhaustive, we refer to the work done by the Canadian philosopher Patricia Churchland (2002), the neurologist Antonio Damasio (2010), the philosopher and cognitive scientist Daniel Dennett (2007) the neurobiologist Gerald Edelman (1987, 1992), and Edelman with G Tononi, (2000) in the US; neuroscientist Jean-Pierre Changeux (1985, 2004, 2008), neurologists Lionel Naccache (2009) and Marc Jeannerod (1996) in France; neuroscientist Wolf Singer (2001) and philosopher Thomas Metzinger (2010) in Germany.

Embodied or Embodiment may refer to embodied cognition (or the embodied mind thesis), a position in cognitive science and the philosophy of mind emphasizing the role that the body plays in shaping the mind (Wikipedia, 2012, besides many other meanings in other domains of science and culture). The aspects of the body include the motor system, the perceptual system, the body’s interactions with the environment (situatedness) and the ontological assumptions about the world that are built into the body and the brain (Wikipedia on Embodied Cognition). So, the mind is embodied via the brain and both the embodied mind and the body itself—this is the whole person—are embedded in the environment, which makes the mind basically relational.

In Networks of the Brain Sporns (2011) says that the body forms a dynamic interface between brain and environment. Neural networks evolve while connected to the body’s sensors and effectors, resulting in bodily movement and repositioning of sensors. Functional connectivity in the brain is shaped by sensorimotor activity that occurs as a result of brain–body–environment interaction, an idea of Chiel & Beer (1997).

In fig. 1 we propose a Brain-Body Entity (B-B E) as the source of mind, consciousness, and culture.

The International Neuropsychoanalysis Society has adopted a new approach, based on the questions raised in neurophilosophical publications, which starting in the 1980s, in line with Freud’s way of thinking, questioned the Cartesian approach. The new direction was among others formulated by Nersessian & Solms (1999) and Schore (1997) as well as in a special volume by Bilder & LeFever (1998) (note 4). The focus of these new fields of interests is nearly always adults, and only rarely is child development studied, a gap which the authors of this volume feel should be addressed.

We also feel that the separation between 19th C. neurology and psychiatry, which occurred in Europe around the 1970s, was deleterious for both disciplines, especially where children are concerned. Psychiatrists have become estranged from neuroscience. The division between neurology and psychiatry has been questioned by Kandel (1979, 2005), as well as by Northoff (2008). Northoff suggested that the concept of neuronal integration may bridge the gap between localizationism and holism in the relation between function and brain regions; this has been raised already by Vygotsky [see note 14 in Intro-2]. Kandel (1999) addressed the question whether biology is at all relevant to psychoanalysis. In his opinion this issue is central to the future of psychoanalysis; he attempted to outline the importance of biology for the future of psychoanalysis. Not long after his publication The International Neuropsychoanalysis Society was founded in London, in 2000. Today more and more clinicians are convinced that the neuroscientific approach is vital for the profession, also for child psychiatry (Ouss-Ryngaert & Golse, 2010). These French child psychiatrists address the rationale derived from child development and child psychiatry: to support why and how we should link neuroscience and psychoanalysis.

Although the separation between psychiatry and neurology freed researchers to focus on issues in their individual fields—giving rise to the rapid accumulation of scientific data—it is clear that 21st C. neurology on the one hand and psychiatry, psychology and allied disciplines on the other suffer as a result. Although the previous separation hinders a new collaboration—interdisciplinary approach is once again being adopted in present research.

Since the 1980s, there have been numerous so-called ‘neurophilosophy’ publications (note 5), generally dealing with the connection between ‘body and mind’ or ‘brain and mind’, notably the explanation of what we call ‘consciousness’, as well as the unconscious and its role. The term embodiment in embodied mind and embodied cognition [6] invaded neuroscience (Chiel & Beer, 1997; Clark, 2004; Gallagher, 2008; Pfeifer & Burgard, 2007; Varela et al., 1991) (note 6). Evolution has led to the human brain, enabling culture and social relatedness a.o. by mentalizing, in order to cooperate and survive as a species [fig. 1].
To the best of our knowledge, Tan’s metamorphosis concept (ch. 7) is the only developmental neurological concept of speech-language development with an essential connection to the development of the motor function and affect, with consequences for the treatment of developmental language disorders [details in the chapters 8 and 12].

Grounded cognition has been proposed by among others Wilson (2002) and Gibbs (2006). Praxis and speech and their derivatives as well as related attention, memory and emotions are intrinsically connected to ‘grounded’ cognition, an embodied approach to cognition. According to Barsalou (2008) grounded cognition rejects traditional, Cartesian views that knowledge resides in a semantic memory system separate from the brain’s system for perception, action and introspection, that cognition is computation on amodal symbols in a modular system, independent of the brain’s modular systems for perception, action and introspection.

Instead, grounded cognition proposes that modal simulations, bodily states, and situated action underlie cognition. Some accounts of grounded cognition focus on bodily states, most focus on the role of simulation in cognition, i.e. the re-enactment of perceptual, motor and introspective states, earlier experienced and stored in memory; mental imagery is a typical case of conscious re-enactment. However ‘grounded’ does not mean exclusively ‘embodied’, as cognition often proceeds independently of the body as such.

If people acknowledge that the brain mediates their behaviour and culture as co-operative behaviour in general, they have on top of their theory-of-mind functions a Theory of Brain. The result is another level of their conceptual self (Neisser, ch. 6.4) and social consciousness.

‘Cogito, ergo sum’ suggests that consciousness of the mind in first-person perspective (‘I’ think) is proof of the existence the phenomenal self (‘I’ am), which is, however, a product of embodied brain activity [end of note 4]. Whereas Descartes made a principle difference between thought/mind (res cogitans) and body/brain (res extensa), mind, in his concept not being a subject that one could study scientifically, became a subject of investigation. Freud’s ideal was to show a functional and structural model of the mind; in our time people try to explain what they mean if they state that the mind is enbrained and embodied.

- A new neurological concept of early speech-language development, ‘the metamorphosis concept’, was developed by the Dutch child psychiatrist Xavier Tan (note 7). This concept, uniting speech and motor function and at the same time ontogenetic changes thereof, is in favour of an embodied approach to cognition or ‘grounded’ cognition [note 8]. The present authors, Njiokiktjien and Verschoor, were members of Tan’s team and were involved in his research and the formulation of this concept. Because of Tan’s untimely death, his work has not previously been published in English [ch. 8].

Working with Tan and his ideas and culture was inspiring. It is what stimulated the authors Njiokiktjien and Verschoor to start thinking about neural transformations other than those related to language: The neural transformations involved in performing and understanding actions; infants’ understanding of other people’s actions, for which Njiokiktjien et al. (2012) have proposed the term ‘Theory of Body’ (Tob) [ch. 6.3], and the development of a complex function, ‘Theory of Mind’ (Tom) that follows Tob [ch. 10].

- The mirror neuron system (MNS). The work done by the Italian neurophysiologists Rizzolatti, Gallese and collaborators in Italy and elsewhere in the world was crucial for our understanding of these developmental processes. They discovered the MNS, one of the most important discoveries for behavioural neurology and psychiatry in the 20th Century.

- A Theory of Brain begins to be part of our self-concept as well as our culture (note 9). Following Theory-of-Mind abilities in and between individuals, the conviction of many neuroscientists that the expressions of our embodied mind, namely its behaviour and culture, is largely mediated by brain functions gets a foothold with the general public. Hippocrates (460-379 BC) stated already that the brain is involved with sensation and is the seat of intelligence and Plato (387 BC) teaches that the brain is the seat of mental processes.
Volume changes and gender differences in brain development

1.1 Understanding developmental disorders involves knowing both which cerebral substrates control a specific function and how and when the substrate is formed. In this chapter we describe the key steps in embryonic brain development: neurulation, cell proliferation and cell migration, as well as myelination and synaptogenesis. The outcomes of the first three processes affect morphogenesis, the anatomical shape of the brain. The latter two processes are far subtler in their effect on form (summary in table 1.1).

Although organogenesis, including that of the brain, has been known for over a century, our knowledge of the development of structures and processes such as pathway systems, synaptogenesis and myelination is limited and there is still much about the genetic coding and biochemical control of the developing brain that needs to be discovered. This means that the mechanisms involved in adverse prenatal influences are not always understood.

The early formation of the brain consists of a series of genetically determined events that stand in a temporal-spatial relationship to one another. Embryonic events, such the formation of the corpus callosum, and that of the cortical layers and internal capsule, as well as dozens of other events always occur in the same order in the series of events to which they belong. This predictability is species-specific. The order affects functional development and neural development itself.

When an error occurs in an early stage, the wrong shape remains in the future form and function, e.g. a neurulation disorder or a callosal dysgenesis. Cognitive functions, such as speech and praxis, are more dependent on the brain’s late and slow maturing structures, e.g. the frontal cortical areas and long pathways in the left hemisphere (1.1). Neurodevelopmental disorders therefore can have, depending on type and period of origin, specific behavioural neurological effects. Most studies done in the last thirty years have focused on network structures and functions that are subserved by neural networks and the connections between them. An unresolved question will be addressed in 1.7.3: how do neurons or neuronal networks represent a mental object [6]?

Hemispherectomy, congenital hemisphere damage and perinatal one-sided asphyctic and traumatic damages are the most important clinical ‘models’ that tell us something on the long-term prognosis of damages of one hemisphere and neuroplasticity, which is addressed here.

The human brain is not an organ that has only to grow and mature after birth nor is it ‘empty’ — a tabula rasa —; it is the result of more than a billion years of evolution. Whereas many brain systems are open — notably the neocortices are experience-dependent systems —, it is the result of development and experiences, which are studied today by those who propose neural constructivism.
The vertebrate animals, including primates, have undergone tremendous evolution in their development. The developmental history of a species is known as phylogeny. The development of an individual or individuals in a species is called ontogeny. Within a short time span, phylogeny is partially repeated in ontogeny, a theory of recapitulation originally proposed in 1866 by the German zoologist Ernst Haeckel. In its literal and universal form this theory is rejected. The theory of recapitulation has been influential until our time, e.g. MacLean proposed a trune brain with a reptile-brain layer, which has a core of truth [ch. 4.2].

An example is the homeobox gene EMX-1. When this gene is deleted, mice have callosal agenesis (Rakic & Novakowski, 1981).

(1) Hierarchical processes. Evolutionarily older cell groups are present first and sensory and motor processes, for example, are earlier than cognitive ones. (2) Isochronous development, whereby a brain develops as a whole. (3) A progressive change in the configuration of complex, interrelated networks. Courchesne shows in his review that in reality all three processes take place in different stages. It is clinically important that disorders early in development affect later development. While this is true for morphogenesis as well as function, the latter should be seen in the light of the ‘Kennard principle’ [1.10.3].

In evolution two brain hemispheres are created; in phylogeny of vertebrates they are connected by neural pathways, the most important being the corpus callosum (CC) and the anterior commissure [note 1]. The division of the brain takes place early, during the neurulation stage in the first weeks after conception. Ontogeny is characterized by phenomena that have been studied separately for a century, but whose interrelationship is poorly understood. The interrelationship between the developing structures, such as the size and subsequently the establishment of structures in the anterior-posterior direction and the dorsoventral gradients of the neural tube, are controlled by what are called homeobox genes, part of the HOX family (Sanes et al., 2000). HOX genes, which are expressed along the rostrocaudal neural tube and play a role in segmental formation, control the expression of other genes [note 2] [for these genes, not further addressed here see also brain evolution: ch. 3.5, and genes related to development: ch.9.3.3.1.]

There are organized movements in the early prenatal stages (De Vries, 1982; 1985); the neural structures for body movement and vital functions such as respiratory and autonomic responses as well as the seeking system for foraging are already present at birth. However, cognitive functions, speech and praxis are largely dependent on structures, such as the cerebral cortex and its connections which mature much later.

Sensory and motor systems are myelinated before the end of the 2nd year, but the myelination of networks for complex behaviour lasts much longer [section 1.6].

Table 1-1 gives a schematic overview of the well-known ontogenetic processes. This presents a chronology of global processes but gives no satisfactory answer to the question of cerebral developmental change as a whole. According to Courchesne (1990) there are 3 types of developmental change: Hierarchical processes, isochronous development and progressive change [note 3].

Readers who wish to obtain background information on the topic of pre- and early postnatal development will find the best orientation in:

- the non-specialist literature Changeux, 1985; Cowan, 1979. For (developmental) psychologists, Spreen et al. (1995) and Johnson (1997) and Johnson et al. (2002) present important developmental theories as well as summaries of developmental abnormalities. A detailed description of morphogenesis can be found in Leviton et al. (1984), while one of embryological development can be found in Purves & Lichtman (1985), Sanes et al. (2000) and in surveys (Greenough & Juraska 1986; Innocenti, 1995; Kandel et al., 1995). Dowling (2001) provides excellent general information on brain function. Deviant development processes which start at birth were described by Lemire et al. (1975) and recently by Lagerkrantz et al. (2009). Sporns (2011) informs us extensively about neural networks.

At the end of this chapter we have added figures on adult anatomy in order to orient the non-informed reader.
The neural basis of emotional interaction, attention, memory and time awareness

Charles Njokiktjien

2.0 When we perceive the world and communicate with it—either living or non-living objects—our brain is active. Describing this brain activity is an approach only, keeping in mind Dalla Barba’s (2002) saying that ‘there are no such things as mental representations or memory engrams, because there is no homunculus to interpret them...’ ‘the misleading assumption that time can exist in things’. ‘Time cannot even be found in a “thing” like the brain’.

Although we have long been familiar with the global neuroanatomy of pathways, cortical areas and subcortical nuclei and their functions, neuroscience’s focus on functional networks and functional brain systems, their neurotransmitter and receptor functions and genetic background [ch. 3] is relatively recent. Brain-behaviour relationships are extremely complex. When we speak about neural substrates or neural representations of a certain behaviour, we mean the crucial crossroads in large neuronal networks, each of which has a specific function and works in concert with other networks. What counts are the neuronal connections between them.

Most of what is known about the relation between the brain and behaviour involves what effects a dysfunction of part of the nervous system has in adults. The effects will not necessarily be the same in children, certainly not those under the age of 9. The neural networks mentioned above undergo not only morphological and functional transformations during ontogeny, but that their connectivity changes as well (Sporns, 2011). These changes depend for a large part on environmental variables and personal experiences; the question is: to what extent are these memories fixed?

Here we focus on the neural basis for emotional interaction, which is of crucial importance from birth on. Some thoughts are included on current concepts of perception, and we address the mirror neuron system. Most of what is known about the relation between the brain and behaviour involves what effects a dysfunction of part of the nervous system has in adults. The effects will not necessarily be the same in children, certainly not those under the age of 9. The neural networks mentioned above undergo not only morphological and functional transformations during ontogeny, but that their connectivity changes as well (Sporns, 2011). These changes depend for a large part on environmental variables and personal experiences; the question is: to what extent are these memories fixed?

Praxis and speech as well as the emotions belong to neurobehaviour and are intrinsically connected to ‘grounded’ cognition [see introduction to this volume, Intro 1, note 8]. This is clear in children with developmental disorders, in which dyspraxia, dysphasia, amnesia, attention deficits and autistic behaviours often occur together, a clinical phenomenon known as comorbidity. Attention, memory and time awareness, functions which play an increasingly important role in infancy and childhood, are therefore addressed. As works of reference for functional neuroanatomy we have used Brodal (1981), Freund et al. (2005), Heimar et al. (2008), Kandel et al. (1995), Nieuwenhuys et al. (2008), and Panksepp (1998).
3 Behavioural neurogenetics

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3. 1 The role of inheritance as a determinant of behaviour and the differential response of individuals to the same or similar environmental conditions has long been known to psychiatrists, novelists, ethnologists and animal breeders. The view of the newborn child as a tabula rasa on which parents are free to write has been discredited in developmental psychology for over 40 years (Bell 1968; Fischer & Bidell, 1998; Thelen & Smith 2006). The new focus in behavioural neurogenetics is on understanding the specific underlying neurobiological mechanisms by which genetic and environmental variables interact to produce normal and abnormal behaviour (Sandi, 2008). Behavioural neurogenetics has strong connections with genetic epidemiology, the science that deals with the causes, emergence and distribution of behavioural traits and neuropsychiatric disorders in groups of relatives and populations [note 1]. Surprisingly it has provided some of the strongest evidence in regard to the causal role of the environment in the expression of behavioural traits and psychiatric disorders (Faraone et al., 1999). Nature and nurture are not mutually exclusive antagonists but dialectically opposing entities that are acting in unity.

The major fallacy that characterizes the views of many scholars and lay people is considering genetics as a static rather than a dynamic influence on development, while the interplay between genes and environment starts just after conception and continues throughout life. Gene-environment interaction affects behaviour largely through shaping the brain: genes themselves only have codes for the production of the proteins that make up the macro- and microstructures of the brain and other chemicals that affect brain function; the endocrine and immune systems also have indirect and direct effects on the development of behaviour. Starting from conception, the physical and chemical, and subsequently the psychosocial environmental conditions experienced by the organism then change the structure and circuitry of the brain through plasticity, equipotentiality, etc. so as to ultimately give rise to differences and similarities between individual members of a species.

Behavioural neurogenetics has shown that while the behavioural similarity and variability found in individuals is mainly genetic in origin, a spectrum of potentialities exists; it is only during a neurodevelopmental process, as a consequence of the induction of environmental conditions in a specific historical period and in a specific human society, that some of these are realized and others are not. It has led to a better understanding of the genetics, molecular biology, neurotransmitter systems, and neural circuitry underlying normal and abnormal human behaviour.

Rapid technical advances in the neurosciences—from molecules, cells, and circuits to systems—such as sophisticated brain imaging techniques, as well as the accumulation of neuropsychological and physiological data
made possible more precise determination of brain regions in relation to cognition, emotion, and behaviour. Although most human behaviours cannot be explained by genes alone and many aspects of the brain’s information processing appear not to be genetically determined, variations in the genetic sequences that impact gene function certainly influence these complex brain functions and behavioural phenotypes. Many neurobehavioural disorders (e.g. autism), and almost all behavioural traits have a genetic component {note 2}.

Ongoing advances in behavioural neurogenetics are now making it possible to identify who is at risk of developing neurobehavioural disorders, develop reliable tests for early detection of risk and apply information about biological pathways to the prevention and treatment of disorders, as well as intervention strategies that target an individual’s specific needs {note 3}.

Another major tenet of interest is the genetic basis of brain functions in relation to the developmental evolutionary changes which initiated and secured the differences between human beings and other species, including our close relatives the chimpanzees. It seeks answers to the questions of how new behavioural traits emerge, how they are acquired and how they are transmitted trans species and within a species, and how specific genes and environmental factors give rise to individual differences through shaping neural circuits in the brain. It involves studying the emergence of individual differences and identifying genes for normal and abnormal affect, cognition, and behaviour in correlation with brain development, as well as studying genetic diseases that are manifested by unique cognitive and behavioural phenotypes (Canlı, 2006).

Providing explanations of the uniqueness of humankind as a species and of each individual human being are the two most important theoretical problems behavioural neurogenetics deals with. Human behaviour is a product of a complex interplay between genes and environment. It is a great mistake to overlook the inseparability of environmental and genetic factors in the structure of the behaviour. This will be discussed here from several angles.

Certain assumptions of the present author | The author believes, firstly, that individuality is the highest, most sophisticated end point of evolution. Individuality must not be confused with non-socially related individualism, individual rights and responsibilities, egoism, pragmatism or individual demands for privileges, which are all particularly prominent in modern society. Individuality is the experience of being unique, overriding the feelings connected to being one of a group that share nationality, ethnicity, colour, gender, religion, or being part of an army, institution, etc. It is the human essence of one individual with a unique genotype and unique blend of genetic tendencies as determined by specific socio-environmental conditions. The most practical implication of this assumption is that each individual is

1 The science that studies the relation between behavioural traits and genetics is generally known as personality genetics. An example is studying how polymorphisms in the serotonin transporter gene relate to the personality trait neuroticism (Ebstein, 2006).

2 As in many neuropsychiatric disorders, including autism, no specific genes have yet been isolated, a complex or multifactorial polygenetic aetiology is assumed to cause these disorders. Genetic causes of mental illness have been over-emphasised at the expense of paying attention to environmental factors. Diagnoses of mental illnesses are largely subjective, and even the nosologies in DSM-IV are not widely agreed upon. It is difficult to delineate a definable cluster of heritable traits even for widely used terms like intelligence. None of these traits appear to have a single genetic cause and it is likely they are caused by the interacting effects of multiple genes in combination with lifestyle and environmental factors. Such disorders are more difficult to analyse because they do not follow the classical patterns of inheritance.
The present author also holds that genetically determined traits can be modified by social experience, given suitable environmental conditions, including a sufficient period of duration. Whatever the truest human nature may be, our subjective mental lives are mediated by and expressed via a substrate that is undeniably biological. However all the biological activities associated with mental functions occur in a specific social reality, which, in turn, shapes and alters them in some ways. Criminal and anti-social behaviour is one example of a behavioural tendency that becomes manifest as the consequence of the gene-environment interplay. Understanding the specific interactions between what is social and biological is the first step in then being able to modify risk factors and take measures, and provide services designed to attenuate the criminal propensity in the human society (Nordstrom et al., 2011). It is in fact part of the implicit agenda of the construction of human societies to create a social environment and conditions—which to a large extent replace the natural environment—that will render criminal behaviour useless, and in the long run will divert the function of genes or traits that cause criminal behaviour to something else. Third, views that posit a dichotomy of human nature do not make sense. Human nature is not biologically designed to be egoistical. Ascribing human behaviour to genes is fallacious. The concept of ‘selfish genes’ is disguised teleology; organisms have a purpose while genes do not. It is equally wrong to think of the human mind at birth as a tabula rasa waiting to be filled by social stimuli. It is for these reasons that human nature cannot be changed by oppressive, propagation-oriented educational techniques.

The attempt to trace the continuity of animal behaviour to human behaviour without any consideration of qualitative leaps may lead to mistaken conclusions. Killing is a different behaviour in the context of human society and in the animal world.

Lastly, while it is not possible to say that humans have evolved as the result of a series of chance events, a strictly deterministic view of the human species’ development is equally wrong.

For terms used in genetics we refer readers to table 3-1.
4 Models of abnormal brain function and developmental disorders
Charles Njloktjien

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4.2.10 Anormal convergent activation and discourse cohesion
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4.3 Individuals with a Combination of Deficits and Superior Abilities
4.3.1 An operational definition of intelligence
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4.0 Many aspects of abnormal development, the pathogenesis as well as the aetiology, are poorly understood. Developmental disorders can be caused by gene defects, prenatal poisoning, asphyctic and other traumatic injuries. However, pathogenetic mechanisms differ as well, related to the time of occurrence and the nature of the pathogenic agent as well as the causal inter-relatedness of co-morbid symptoms. It is therefore incorrect to attribute all developmental disorders to the same pathogenetic cause, as they were in the minimal-brain-dysfunction (MBD) hypothesis put forward in the 1960s and 1970s.

Why, in many cases, do certain functions develop deviantly, while in other cases it is overall development that is deviant? What model of impaired development underlies certain deficiency profiles and what are the genetic mechanisms that relate to specific dysfunctions? Does impaired development always have the same basic causes? There are still no conclusive answers to these questions. We need only to think of the controversies surrounding the ‘nerve growth factor’ and the mechanisms involved in how certain neurons find each other to make synaptic contacts [ch. 1.7].

What are actions, thoughts, memories, logical decisions, inner speech and perception in temporal–spatial, neurophysiological and biochemical terms? Something is known about the functioning of neuronal excitation— inhibition, firing frequency, and neurotransmitter activity. The coherent cooperation of neuronal populations in networks—reciprocal inhibition and excitation, re-entrant signalling, oscillating systems, gating, convergence, divergence, temporospatial influence—is, however, less well-understood and even less is known about the integrated functioning of neuronal populations within and between hemispheres—also called the ‘brain code’ (Cook, 1986), or Pribram’s ‘mental hologram’ [ch. 2.2.1]; it refers also to the binding problem (Singer), addressed in 4.2.8.

The scientific focus in the neurology of behaviour and the neural substrates of the mental object’ [c] has shifted from neuronal centres (19th-century localizationism) to distributed networks (20th-century connectionism). However, as the construction of the mental object is different in children than in adults, we hold that it is crucial for understanding developmental disorders to consider how brain function differs in children and adults, how it develops from childhood to adulthood and what obstacles have to be overcome in this process.

Neural constructivism (21st-century)—emphasizing the open structure of many human neural networks, especially the late-developing ones—
4.3.3 Do deficits only seemingly disappear or do functions actually recover?

4.4 **SAVANT SYNDROME: APPEARANCE AND DISAPPEARANCE OF SPECIFIC SUPERIOR ABILITIES**

Fig. 4-3 Drawing by a mentally retarded boy diagnosed as an autistic savant

4.4.1 Hyperlexia

4.5 **CONSEQUENCES FOR TREATMENT**

examines this transformation, in opposition to the static neuropsychological approach [section 4.2.1]. Neural constructivism also challenges the existence of neural modules, at least in children, and questions whether there are modular or pure developmental disorders or deficits \[4.2.2\], arguing that deficits may not reflect a direct causal path from genetic defect(s) to behavioural phenotype [addressed in ch. 3.4.1].

This leads us inevitably to the phenomenon of co-morbidity in children with developmental disorders. The clinical fact that most disorders consist of a prevailing disorder with other co-morbid disorders challenges the existence of neural modules in children.

Therefore, to understand a bit more of the difficult clinical phenomenon of developmental disorders, we highlight a number of subjects, either clinical such as maturational lag, diaschizis and functional recovery, or scientific such as hemispheric balance.
5 Nature, diversity and principles of ontogenetic processes
Charles Njiokiktjien

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5.5.1 Hemisphere specialization (‘dominance’) as a special example of neural substrate development
5.5.2 Focalisation and modularisation
5.5.3 Divergent and convergent activation

5.0 As chapter 6 addresses functional development in early childhood, notably the processes underlying attachment and Theory of Body (TOB), which constitutes the earliest form of social relatedness, this chapter links neural development and functional development and addresses the principles of development and different theoretical approaches to them. Other concepts that are relevant to development, e.g. neuroconstructivism, modularization and the diarchisis principle, are addressed in chapter 4.

Developmental psychology and theories provide the underpinnings for thinking in paediatric behavioural neurology; this knowledge is important for understanding how plasticity contributes to recovery after brain injury [ch. 1.10]. We see human development as a psychobiological phenomenon, and seek to describe the neural mechanisms underlying this phenomenon and the role each one plays.

While development is universally seen as an orderly process, ideas on what it entails have changed during the last half century. Are social-cognitive functions already present in a rudimentary form at birth and do they then subsequently expand (continuity concept) or do they only appear in the course of development (discontinuity concept)?

The neural correlates connected with development, the continuity of developmental processes and the relations between developmental processes are addressed. It is difficult to correlate all the seemingly unconnected processes that occur during development into a ‘grand unified theory’ or define the ‘essence’ of development. Many do not consider this to be theoretically possible and there are conflicting theories on how processes relate to one another. There seems to be no single dominant factor in development.

The relatively large role of the right hemisphere (RH) in childhood compared to that of the left hemisphere (LH) and the changing hemisphere balance is addressed in several chapters. The discovery of the mirror neuron system (MNS) has revolutionized thinking on several key aspects of developmental neuropsychology: imitation, speech acquisition, action understanding and prediction, and empathy are examples. More is also known about crossmodal perception and learning and the role of synaesthesia in the infant.

Psychomotor and affective development are widely studied. An overview of development and functional brain development as well as a good review of current schools of thought can be found elsewhere. Goswami (ed. 2011); Herschkowitz et al. (1997); Johnson et al. (1997); Legerstee (2005); Miller (2002), & the journal Developmental Science are very informative.
6 Early social relatedness: theory of body, attachment and the self

Charles Njokiktjien

6.0 The principles and concepts of development are addressed in chapter 5. This chapter discusses a number of achievements that take place during the infant’s first year: the concept and the neurology of the mother-infant attachment process (Bowlby) including its bodily correlates, i.e. how the understanding of gestures and actions comes into being. A key notion which we address is the understanding of the affectively loaded bodily behaviour of the mother and the accompanying emotional feelings, which Njokiktjien et al. (2012) have called ‘Theory of Body’ (T0B). Throughout the chapter we use ‘mother’ for the caretaker or attachment figure.

The domains of development which play an instrumental role in mother-infant attachment are (1) perception: first synaesthetic, then crossmodal and coherent perception; (2) motor function: axial motor function as well as imitation, grasping, and preverbal gesturing, pointing and hand motor praxis (actions), all connected to early affectivity, which we consider to be domain-general; (3) the preverbal and pre-linguistic and later speech-language stages, which are the cornerstones of ‘Theory-of-Mind’ (TOM) development [addressed in ch. 8 and ch. 10].

Theory of Body (T0B) is a central issue: what is it, when in infancy does it start and what is its role in the attachment process? We also discuss the related concepts of the infant’s body awareness, body schema, body image and body-ownership and the infant’s feelings about them; they are connected with the emerging ‘phenomenal self’. Emotion cognition and early interactions are seen as the core of T0B development. They are the prelude to the ability to mentalize, i.e. the use of a TOM in daily life situations in older children [ch. 10].

Here we will describe the developmental acquisitions necessary for the infant’s social relatedness. These are largely the result of intense interaction with the mother, whose own psychophysiological inner model—mostly unconscious—is imposed on/is transferred to her baby. This also implies a psychoanalytical point of view. Although we use the terms ‘developmental steps’ and ‘developmental stages’, we do not mean to suggest that we are in agreement with the idea that there is discontinuity in development [see note 7 in ch. 5.1, and notes 4 & 5 in ch. 5.4].

The clinically used stages of development, whose duration varies from infant to infant, are listed in tab. 6.1. Development is only grossly visible for the outside world, sometimes only after careful observation. Although variable, there are developmental norms for the many abilities listed above [tab. 6-VII].

Achieving developmental steps, ‘the milestones’, is accompanied by neuro-anatomical and psychophysiological maturation, notably of the long pathways in the brain and the cerebral cortex [ch. 1.6 and 1.7], a process which is not yet fully understood.
When in ontogenesis does a self-system develop?

6.4.2 Stern's stages and other concepts of the self

6.4.3 The self and the role of the brain

Fig. 6-8 Northoff's structural neuroscience approach to understand the human brain and Stern's early stages of self

6.4.4 The relationship of ToB to 'the self' and the infant’s awareness of other 'selves'

6.4.5 ToB as a building block for ToM

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6.6.3 Attachment and sensory-fugal and sensory-petal projections

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Tab. 6-VI The basic neural conditions needed for secure attachment

Fig. 6-11 A summary of the construction of a ToB in the infant's brain

Tab.6-VII The age norms for developmental acquisitions

Here, the emphasis is on early development, when the most remarkable neuronal transformations take place. The view on the development in this stage is generally descriptive. However, we also give our views of the supposed neural correlates of attachment, the early development of emotional feelings, speech development and the body- and hand motor functions.

The discovery of the mirror neuron system (MNS) has far-reaching consequences for theories about early relationships—interpersonal contact—, the attachment process, imitation and learning, speech development, understanding and empathizing with other people and thus identifying with them [5.3.1]. Finally we discuss how attachment as a first model of social relatedness is laid down in the memory systems of the brain, how flexible this model is, the extent to which it can subsequently change, and what may happen if the model is deviant.

A number of developmental psychologists inspired us. They include Bladergroen in the Netherlands (1971), Dornes in Germany (2004) and Legerstee (2005), Stern (1984) and Trevarthen (1979, 1986, 1996; 2001 with Aitken) in the United States. Piaget (1952), Inhelder, (1969) and Vygotsky (1934) were important influences, as were Freud’s (e.g. 1923) and Bowlby’s (1969, 1973) psychoanalytic constructs. The work of Fonagy (2001) was important for showing the connections that exist between attachment theory and classic psychoanalysis. The Leiden research group in the Netherlands with Marinus van IJzendoorn and collaborators has given a special impetus to the understanding of attachment (a.o. McCall et al. 2011).

Neuropsychoanalysis is a new field, pioneered in the work of the psychoanalysts Nersessian & Solms (1999), Solms (2005) and Schore (2003a), neurologist Turnbull (2000), and functional neuro-anatomist Panksepp (1998), among others [see also intro-1, p. 5]. The standard psychoanalytical concepts, including ego and id, that were challenged by neuroscience, notably by ethology, have been reformulated (Solms & Panksepp, 2012) and continue to be vital for our understanding of the workings of the mind.

Another new field of neuroscience is that of the "phenomenal self" (Metzinger, 2003), studied in infants by developmental psychologist and psychiatrist Daniel Stern (1984) and in adults by the German neuropsychiatrist Georg Northoff, who adopts a neurological and structural approach (2008). Not only have Northoff and Panksepp made a strong claim for neurological substrates as the basic structures of the core self, they have also proposed that the core self, made visible as a default-mode neural activity, is an innate relational self. These new fields are significantly influencing theories of child development.
7 Developmental kinesics, related phenomena of non-verbal interaction, and disorders Ank Verschoor

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7.9 ASSESSMENT

Kinesics can however also be impaired in a more instrumental sense by motor dyspraxia or in very rare disorders like Möbius syndrome. Clinical study and assessment of kinesics [section 7.9] can contribute to early differential diagnoses specifically of ASD and developmental dysphasia [ch. 12.xx]. Furthermore, knowledge of the field can enhance the development of treatment.

7.0 In the child’s first years the development of attachment and ‘Theory of Body’ (ToB), joint attention and further social interaction are largely dependent on the perception and adaptation to the expression and timing of others’ messages, conveyed by posture, locomotion, gesticulation, facial movements, gaze, touch and voice.

The expressive motor aspect of nonverbal communication was called kinesics by the anthropologist Ray Birdwhistell (1952, 1970). By analogy the terms vocalics, haptics and proxemics are used for the paralinguistic information conveyed by the voice, the messages implicit in different forms of touch and the use of interpersonal space.

Long before they first use symbolic gestures and words, infants comprehend the meaning of facial expressions and tone of voice and can predict intent from motor acts. Although their expressive skills develop more slowly, infants engage actively in synchronised interpersonal interaction and convey intentions and emotions through axial tone, crying, facial expression, glance and prosodic babbling [addressed in fig. 5-2, ch. 5.2.5 and ch. 6.2.1].

Spoken language develops in this context of nonverbal interaction and, more specifically, it is developmentally closely linked to its motor-gestural counterpart. The first vocalisations, babbling and protowords are co-produced with motor actions. Afterwards, in the child’s second year, many words of the developing lexicon are first expressed in gestural form, and the first two-word utterances consist of word-gesture combinations [addressed in ch. 8.5].

While development of spoken language for social interaction proceeds as the child grows, it does so as part of a nonverbal-vernacular communicative continuum. The pragmatics of language depend on and consist of nonverbal aspects such as correct emotional intonation (emotional prosody), gestural and facial expression (kinesics), the use of interpersonal nearness (proxemics), and adequate interactional synchrony.

The achievement of developmental ‘milestones’ is accompanied by neuro-anatomical and psychophysiological maturation, notably of the cerebral cortex (predominantly in the right hemisphere [ch. 2.5] and the long and late myelinating pathways in the brain [ch. 1.6]. The extent of these changes is not yet fully known. The discovery of the mirror neuron system (MNS) has substantially influenced our views of action/gesture understanding, imitation and learning, speech development and the attachment processes and thus modified how we see children’s identification with other people and their ability to understand others and predict their behaviour. The neural networks involved are discussed in ch. 5.2.2, ch 10.xx and ch.xx.

Disorders affecting the development of nonverbal communicative skills such as kinesics [section 7.8] are seen principally as an aspect of Autism Spectrum Disorders (ASDs), possibly as part of a more global emotional agnosia. {▷}. 

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8 Tan’s metamorphosis concept of speech-language development and early disorders Catharina Anna Verschoor & Charles Njokiktjien

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8.1 In this chapter we address early speech-language acquisition as a developmental process and suggest that it is closely related to the attachment process beginning at birth, as well as to theory-of-body acquisition that starts after the third month [ch. 6.3]. We argue that learning to speak is an interactive and affect-driven process that can only take place in a ‘language bath’ and that it must involve people: a child will not learn to speak by watching television.

In connection with the first thesis, we will argue that although the left hemisphere (L.H) is ultimately dominant for formal language, the earliest learning of both receptive and expressive speech is a bi-hemispheric process: perceiving formal speech sounds is dominated by areas in the L.H, while processing natural speech and emotional meaning is an R.H function. The L.H becomes more active when the child begins to produce syntactic structures (3-word combinations).

These hypotheses are the core of the Dutch neuropsychiatrist, Xavier S. T. Tan’s metamorphosis concept, a neurolinguistic concept which postulates that there is a change in activation balance from R.H to the L.H, brought about by modified joint affect-motor and affect-linguistic paradigms. These paradigms are R.H neural networks in their earliest forms, changing into L.H networks for elaboration of language and praxis. In fact, several research studies seem to support the idea that the R.H plays a dominant role before the three-word-sentence stage.

Many findings point to R.H significance for affect in verbal and non-verbal pragmatics, i.e. affect in body language and speech. First there is R.H dominance for emotions, especially for aspects that are automatic and unisonic and for the preconscious aspect as well. In early childhood these aspects prevail over formal, conceptual and deliberative aspects, processed by the L.H. Furthermore infants learn through a multimodal object-learning process, a R.H function, including the word-sound for an object and its accompanying affect. Lastly, there is extensive research showing early L.H activation for formal speech sounds but prevalent R.H activation for the processing of prosodic and facial information. There is also a higher overall activity level in the R.H in the first three years; recent findings on the normal developmental trajectory of expressive language lateralization suggest that typical adult L.H lateralization emerges from an early bilateral language network.

Deviant speech-language in the early R.H stage may entail a lag in the first year babbling stage, as well as diminished word and speech quality. In toddlers with developmental dysphasia, speech understanding is quite good, but although they are able to utter single words and sometimes a number of words together, they do not progress to sentences.
It may also be difficult to understand their words because of poor speech articulation. Word production and speech articulation are still closely linked in the brain in the Broca area; this is addressed in 8.8.

- For speech and language terminology see ch. 12.0 (introduction).
- Age norms for perception, motor function and speech-language can be found in tab. 6-vii, at the end of chapter 6.
- Treatment principles are addressed in ch. 13.7.
9 Autism spectrum disorders, early signs Barış Korkmaz

9.1 INTRODUCTION

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9.1 Autism is a chronic neuro-developmental disorder present from early childhood which results in deviant social behaviour and social communication. In 1908, the Swiss psychiatrist Eugen Bleuler first used the word ‘autism’, a term etymologically derived from a Greek word Autos (self), to describe self-absorption due to poor social relatedness as one of the pathognomonic symptoms of schizophrenia. Autism means ‘living with self’, i.e. preoccupation with self.

Major problem areas of behaviour include inappropriate sociability, deficits in verbal and non-verbal communication, loss or poverty of imaginative activity, repetitive and restricted activity including stereotyped behaviours, narrow fields of interest, resistance to change, and bizarre, abnormal sensations (Wing, 1988).

Autism was first described by Kanner in 1943 [note 1]. There are currently several different systems for classifying autism spectrum disorders (ASDs) but none of them, including those of the two major systems (the American Psychiatric Association and the World Health Organization), adequately or concisely explains the full spectrum of autistic disorders [note 2]. Recently adopted simplistic unitary approaches have not provided reliable ways of diagnosing autism spectrum and related disorder; and given the nosological problems, misconceptions and terminological confusions in the field of clinical research and practice, significant changes should be expected in the ways DSM and ICD are classified.

The American Psychiatric Association (APA, 2000) [tab. 9-1] and World Health Organization (WHO, 1993) [tab. 9-II] have classified several autism spectrum disorders as pervasive developmental disorders (PDD). Currently there is no biological marker for autism and diagnoses are made on the basis of the clinical picture and the course of the condition over time. This gives rise to uncertainties and disagreements. There is far less certainty about the use of the term pervasive developmental disorder and about the nosological validity of its subcategories. The use of these diagnoses in early childhood has been shown to vary widely and is not considered to be wholly reliable (Charman & Baird, 2002; Cox et al., 1999; Lord et al., 2006; Turner et al., 2006). Childhood autism is also known as autistic disorder, infantile autism, infantile psychosis and Kanner’s syndrome. Atypical autism includes atypical childhood psychosis and mental retardation with autistic features. Other childhood disintegrative disorders include dementia infantilis, disintegrative psychosis, Heller’s syndrome and symbiotic psychosis. PDD-NOS is defined by exclusion (Volkmar, 1998). Paradoxically individuals with PDD-NOS are much less frequently studied although they are much more common than other categories.

In this chapter the terms Autism Spectrum Disorder, ‘ASD’ and autism’ are used interchangeably to refer to the spectrum as a whole.
9.6.5 Sensory abnormalities
9.6.6 Motor problems
9.6.7 Atypical visual exploration and object use
9.6.8 Face recognition deficits
9.6.9 Delays in the perception of physical causality

Tab. 9-VII Symptoms of concern in ASD as listed in related developmental fields

Tab 9-VIII Gesture use and other social communicative behaviour

9.7 STABILITY OF DIAGNOSIS
9.8 CULTURAL ASPECTS OF THE DIAGNOSTIC PROCESS
9.9 EARLY BIOLOGICAL SIGNS THAT MAY INDICATE AUTISM, AND POSSIBLE BIOMARKERS
9.10 SCREENING TESTS, QUESTIONNAIRES AND OTHER METHODS OF DEVELOPMENTAL AND DIAGNOSTIC ASSESSMENT

Tab. 9-IX Some psychological tests used for children with ASD

9.11 DIFFERENTIAL DIAGNOSIS OF AUTISM BEFORE 18 MONTHS
9.11.1 Congenital deafness
9.11.2 Congenital blindness
9.11.3 Social deprivation

Tab. 9-X Differential diagnosis of autism in infancy

9.12 SEVERITY ANALYSIS OF AUTISM SPECTRUM DISORDERS
9.12.1 Typical autism and Asperger Syndrome
9.12.2 Atypical autism (or PDD-NOS) and Asperger Syndrome
9.12.3 High functioning autism and Asperger Syndrome

9.13 FAMILY HISTORY
9.14 ASSOCIATED PHYSICAL HEALTH PROBLEMS
9.15 NEUROLOGICAL EXAMINATION
9.15.1 Head circumference

Tab. 9-XI Aetiology of macrocephaly

[see note 1 in 9.2]. This classification replaces the obscure concept of pervasive developmental disorder not otherwise specified (PDD-NOS), a term that enjoyed little consensus as a diagnostic category. It is therefore reasonable to expect that a new system of classification will adopt a more dimensional framework rather than the current categorical approach to diagnosis.

One of the things that is not yet clear is the causal connection between major defining fields of problems in autism, absent social reciprocity, repetitive behaviour on the one hand and interests and language development on the other.

A recent study indicated the importance of the fractionation of social/communicative and repetitive behaviours or interest symptoms in ASD (Dworzynski et al., 2009).

In DSM-5, problems in social interaction and verbal and non-verbal communication are part of the same category, which means that the inseparability of communication and sociability is recognized. Three symptom domains (social, communication, and repetitive behaviour) become two (social-communication and repetitive behaviours). The behavioural abnormalities characteristic of autism are defined according to biologically defined norms and expectations.

Deficits in socialisation are characterized either by absence of social interaction, deviant social interaction or delayed initiation, usually involving a total or nearly total lack of social reciprocity. In social confrontation impaired use of non-verbal behaviours to regulate interactions is noted in all cases. Delayed, abnormal or absent peer interactions with few or no friendships are seen. Other symptoms include not seeking to share enjoyment and interests and ignoring others’ needs. In addition there are problems in all areas of social cognition, including social judgment.

Although the form of the language may be spared, content is always impaired in autism-related disorders. Deficits in communication include absence, deviance or delay in verbal language, usually without non-verbal compensation (i.e. gestures); impaired expressive language and conversation; severe disturbance in pragmatic language use and semantics; poor vocabulary; stereotyped, perseverating, and repetitive, or idiosyncratic language.

Another category is related to restricted interests and repetitive behaviour. It includes lack of imagination, replaced or compensated for by restricted, stereotyped, and repetitive patterns of behaviour. These may range from delayed imaginative and social imitative play to preoccupation with stereotyped or restricted interests or topics, adherence to routines, rigidity, and perseverative behaviour, stereotyped, repetitive motor mannerisms, and self stimulatory behaviour as well as preoccupation or fascination with parts of items and unusual visual exploration.
9.16 PATHOLOGICAL NEUROANATOMY AND NEUROIMAGING FINDINGS OF AUTISM

9.16.1 Developmental gray-matter/white-matter changes
9.16.2 The cerebellum
9.16.3 Brainstem structures
9.16.4 The limbic system
9.16.5 The amygdala
9.16.6 The hippocampal formation
9.16.7 The anterior cingulate cortex (acc)
9.16.8 The temporal-parietal junction
9.16.9 Corpus callosum (cc)
9.16.10 The frontal and prefrontal lobe
9.16.11 Cerebral cortex
9.16.12 Cerebral cortical folding
9.16.13 Abnormalities in the columnar structure of the neocortex
9.16.14 Connectivity abnormalities
9.16.15 Other brain regions and structures
9.17 MAJOR NEUROBIOLOGICAL THEORIES ON ASD
9.18 MAJOR PSYCHOLOGICAL MECHANISMS UNDERPINNING THE BEHAVIOURAL MANIFESTATIONS OF AUTISM SPECTRUM DISORDERS

1 Leo Kanner, an American child psychiatrist, originally from Vienna, was the first to report on autism as a distinct clinical entity in his publication ‘Autistic Disturbance of Affective Contact’ (Kanner, 1943). The children in the study typically appeared to be self-absorbed, e.g. ‘in a different world of their own’, due to their lack of responsiveness to and interest in the people around them.

A Vienna-born paediatrician, Hans Asperger, reported his findings in 1944 in a publication entitled ‘Autistic Psychopathy’ (‘Die autistischen Psychopathen’ im Kindesalter), which was largely unknown in Anglo-Saxon world until the 1980s. Although autism as a distinct entity is a relatively newly recognized disorder, it has been known for many years (Frith, 1993). In the 1920s, the Russian child psychiatrist and neuropathologist Dr Ewa Sucharewa described children with characteristics similar to those described by Asperger (Wolff, 1996).

Overlapping behaviours (e.g. limited social-emotional reciprocity, limited sharing of interests, and reduced back-and-forth conversation are combined into one reciprocity symptom) (Ozonoff, 2012).

In addition to the criteria of restricted repetitive interests, unusual sensory behaviours that were not included in DSM-IV are considered major subcriteria of diagnosis. The third criterion is that the symptoms of autism must be present in early childhood. This is in contrast with DSM-IV, which required symptoms to be present before 3 years of age (APA, 2010) [tab. 9-111].

As expected, disintegrative disorders due to a neurodegenerative process (Heller syndrome) and Rett disorder are excluded in the recent classification of DSM, as autism was only one of the non-defining symptoms in these disorders.

While disease categories have been grouped together in recent classifications, neurobiological research indicates the presence of several types of ‘autisms’ with different constellations of core symptoms corresponding to different aetiologies and pathogenetic mechanisms (Rapin & Tuchman, 2008).

In line with the recent tendency towards simplification, many of the diagnostic sub-types including Asperger disorder and PDD-NOS have been removed from the classification and the autism spectrum is seen as a broad continuum with variable severity rather than as a cluster of sub-types.

However, the elimination of Asperger syndrome seems premature (Ghaziuddin, 2010) and the revised diagnostic criteria proposed in DSM-5 appear to be less sensitive than the prior criteria, especially for individuals with high functioning autism or AS (Mattila et al., 2011). Autistic traits need to be described taking into account the full range of ways in which they are manifested, in other words starting from individuals with full expression, and seeing them in comparison to family members with ‘some autistic-like characteristics’ constituting the Broader Autism Phenotype, and in terms of similar traits in the general population (Bailey et al., 1998; Bolton et al., 1994; Brugha, 2002; Melzer et al., 2002; Szatmari et al., 1989). Autism denotes not only ‘disorder’ but also a ‘difference’ between individuals. It then becomes doubtful whether all forms of autism, like Asperger syndrome, have a clinical status that requires medical remediation.

From a practical clinical viewpoint, autism has three main clinical prototypes: typical autism (low versus normal/high IQ subtype), Asperger syndrome (AS) and atypical autism. The diagnosis of atypical autism is a provisional diagnosis that can signal a clinical course in different directions – towards either normality with some broad autism phenotype, typical autism or AS. People with AS have many of the same symptoms.
Occasionally it is difficult to make a diagnosis of autism as clinical symptoms suggestive of other disorders may be more prominent. The introduction of operational definitions such as those of the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revised (DSM-IV-TR, [APA, 2000]), although they are not wholly satisfactory, led to greater reliability of diagnosis and relative coherence between different studies. seen in autism, but with ultimately normal superficial language development and IQ in the average range (rarely border) or above. Although DSM- V excludes A as a diagnostic category it is retained throughout this book for several reasons, among others because the present author believes that it will be sooner or later recognized again as a major subtype of autism; some of the changes are attributed to the reverberating splitting and lumping tendencies seen in the disease classifications.

Although the prognosis of functional impairment is a key issue in the clinical management of people with autism and the level of severity of autism is recognized as an important diagnostic category, the relations between symptoms that lead to diagnosis and levels of functional impairment are obscure (Rapee et al., 2012).

The WHO provided a comprehensive framework to classify the functional effects of diseases. It is known as International Classification of Functioning, Disability and Health: Children and Youth Version (WHO, 2007) which organises aspects of impairment across three areas: body (including physical and psychological functions), activity (reflecting the ability to perform tasks) and participation (reflecting the extent to which a child becomes involved in life). Hence, high-functioning autism may be a misnomer. It may at times give rise to more severe distress, low level of quality of life or impairment compared to even low functioning autism, at least in some cases although the implications of the diagnosis are the opposite.
10.1 Theory of Mind: the ability to mentalize

10.1 The dynamically changing complexity of human communication and cooperation, determined largely by the need for food and protection, fostered the development and use of a psychological mechanism that is a crucial component of social consciousness, allowing prediction and interpretation of others’ behaviour.

This construct, which psychologists call Theory of Mind (ToM), is currently used to explain a related set of intellectual abilities that enables us to understand that others have beliefs, intuitions, plans, assumptions, emotions, ambitions, hopes, knowledge, information, desires and intentions and that these may differ from our own. ToM, also known as mindreading, is the awareness of others’ having a mind with varying mental states that cause behaviour, and thus serves to explain and predict it. Mindreading not only enables us to make sense of another person’s current behaviour, it also allows us to imagine a whole set of possible mental states and to predict what the person’s next action might be. It is linked to the development of self-awareness – consciousness of having a self that is distinct from others’ – which includes awareness of awareness and awareness of being a member of a species. This ultimately gives rise to the recognition of sharing similarities and goals as well as dissimilarities and possible conflicts of interests with others.

Human communication starts as interpersonal communication and includes experiences that emerge during interaction with others (Clark, 1996; Goodwin, 2000); at birth this is the mother, but there is soon differentiation into intrapersonal and interpersonal domains as we also have private thoughts, desires etc., and may wish to conceal them, behaving differently in the presence of others. There is no objective way to be sure one knows the contents of another’s consciousness and to access their motivations and desires as causes of their behaviour. Instead the individual constructs a theory for analyzing their impressions of others and self that have been obtained while participating in a range of kinds of human interaction. Reconstructing these impressions from different perspectives according to experiential knowledge is the basis of ToM development.

Advanced forms of ToM are grounded on transforming what are called natural psychological functions into social functions, a process which mainly takes the form of conceptual development (Vygotsky, 1998; 1978). The process is usually mediated by activity led by an adult (parent, teacher etc.), or more competent peers, in the context of a socially meaningful activity (Vygotsky, 1978). ToM ability is ultimately one of the major tools for the emergence of self-development and different selves, as described by a number of researchers, as well as self/other distinction and individuality, a capacity unique to human beings (Liddle & Nettle, 2006). Humans are natural psychologists: we have an insatiable interest in other people, their minds and
10.6 **THEORY OF MIND**
10.7 **SELF DEVELOPMENT, SELF AWARENESS AND THEORY OF OWN MIND**
10.8 **CULTURAL ASPECTS OF TOM**
10.9 **REAL-WORLD CONSEQUENCES OF TOM AND INDIVIDUAL DIFFERENCES IN TOM**
10.9.1 **Tom and moral behaviour**
10.10 **MEASURING TOM IN CHILDREN**
10.11 **SOME CLINICAL CONDITIONS WITH TOM DEFICIT**
10.11.1 **Autism spectrum disorders**
10.11.2 **Learning disabilities (LD)**
10.11.3 **Developmental language disorders**
10.11.4 **Schizophrenia and related disorders**
10.11.5 **Congenital deafness**
10.11.6 **Congenital blindness**
10.11.7 **Personality disorders**
10.11.8 **Attention deficit and hyperactivity disorder (ADHD)**
10.11.9 **Trauma, neglect and abuse**
10.12 **THEORIES OF ‘THEORY OF MIND’**
10.12.1 **‘Theory’ Theory**
10.12.2 **Simulation Theory**
10.12.3 **Modularity Theory**
10.13 **NEUROSCIENCE OF THEORY OF MIND**
10.13.1 **Limbic structures**
10.13.2 **Prefrontal lobe**
10.13.3 **Temporal lobe**
10.13.4 **Mirror Neuron System (MNS)**
10.13.5 **Spindle neurons**
10.14 **EPILOGUE**

**Tab.10-1** Summary of Tom development

Although Tom is largely a human ability, the term was coined by two primatologists, Premack and Woodruff (1978) in a paper entitled ‘Does the Chimpanzee Have a Theory of Mind?’ However, the notion that one’s access to the minds of other people (and also to one’s own mental states) is mediated by an implicitly held theory of the functioning of the human mind was introduced by the philosopher Lewis (1966). Notes 2–4 next page

- behaviour (Doherty, 2009). We therefore continuously make inferences about others’ psychological states. We analyze the impressions others induce in our minds (although these impressions may be false) to construct theories that go beneath the surface, from verbal and non-verbal communications, ‘behavioural’ descriptions of people and actions—Theory of Body—to deeper, more psychologically meaningful understanding and attributions in a causal, explanatory framework. The aim is to understand and predict behaviour on the basis of internal mental states, including motivations, needs, intentions, beliefs, desires, and expectations. Constructing a theory on the basis of predictions, cues and random information makes up the core of folk (or commonsense) psychology [note 3], and also includes elements gathered from casual conversation and gossip. Schooling influences the way the individual constructs their Tom: the more years of schooling one has, the more likely one is to theorise rather than resort to simulation-based schemas or impressions.

Although Tom is inextricably related to categories of mind, many of which were discussed in traditional philosophy and psychology, Western philosophy and science, dominated by pre-determinism, pure objectivity, empiricism, descriptive phenomenology and positivism often find it difficult to accept that one person can know another person’s ‘inner’ meanings solely from their behaviour and words (Austin, 1962; Wittgenstein, 1965; Wittgenstein, 1953; James, 1890). It is likely that research on Tom will continue to be relevant to many issues in the social sciences (e.g. ethics, attitudes etc.) and to be of prime concern to neuroscientists, even if what is now called ‘Tom’ is one day be replaced by another term.


The ontogenetic and phylogenetic aspects of Tom development have been studied in detail but less attention has been given to the historical evolution of the concept from ancient to modern times. Julian Jaynes (1976), one scholar who dealt with the question, found that while the Iliad has few descriptions of introspection, the Odyssey, written later, appears to reflect a view of consciousness similar to our own. This indicates that like all other forms of human consciousness, our Tom ability has evolved over time, shaped both by social conditions and our biological development. The development of traditions, the rule of law, rationality and common sense [note 4] all depend on the Tom level attained by a particular culture and society.

NOTES

1 Although Tom is largely a human ability, the term was coined by two primatologists, Premack and Woodruff (1978) in a paper entitled ‘Does the Chimpanzee Have a Theory of Mind?’ However, the notion that one’s access to the minds of other people (and also to one’s own mental states) is mediated by an implicitly held theory of the functioning of the human mind was introduced by the philosopher Lewis (1966).

2 Notes 2–4 next page
2 Within a general process of development, two qualitatively different lines of development differing in origin, can be distinguished: the elementary processes, which are of biological origin, on the one hand, and the higher psychological functions, of sociocultural origin, on the other. The history of child behaviour is born from the interweaving of these two lines. The history of the development of the higher psychological functions is impossible without a study of their prehistory, their biological roots and their organic disposition. The developmental roots of two fundamental, cultural forms of behaviour arise during infancy: the use of tools and human speech (Vygotsky, 1978: p 46).

3 Folk Psychology (naive psychology) is the set of strategies ordinary people use when they give explanations and make predictions about other people’s actions, based on what the person thinks, knows and expects coupled with what the person wants, intends, or hopes. It is an attempt to discover the mental truth underlying perceivable events by using inferences, deductions, reasoning and generalizations, and involves employing plausible hypotheses and predictions. It thereby provides a causal framework that allows us to explain the emotional reactions that result from these desires, beliefs, preferences, and perceptions, a process which relies on commonsense understandings such as ‘happiness appears as fulfilled desire, frustration as unfulfilled desire; we are surprised when events contradict our firmly held beliefs.’

4 Common sense stemming from a sort of intuitive cognition, gained in part from previous social experience, makes it possible for an individual to arrive at a judgment based on what appears to them to be a sound perception of a situation or facts. Hence it enables the individual to decide what it is right to do on the basis of social norms. Common sense implies the presence of knowing what people in general would agree on; not only what the individual ‘senses’ is a shared understanding but also what they see as knowledge or true belief shared by the other people they know. Obviously ideas that appear to be true by common sense may frequently prove to be false (in fact science is based on experimentation, rather than on what appears to be true).
Autism spectrum disorders, late signs

11.1 Autism is a life-long disability. As the human brain continues to develop until the end of life [note 1], the profile of the clinical symptomatology may change as the individual ages [tab. 11-1]. Although the elaborate changes that occur in the brains of adolescents with autism are not yet fully understood, it is likely that transient regressive events in the brain are part of the normal developmental process. Apart from developmental abnormalities of brain structures through childhood, puberty and adolescence, spontaneous improvement and regressions as well as responses to various therapeutic interventions, all of which correspond to personality development, contribute to subtle or prominent changes in the clinical symptoms of autism. On the other hand, some symptoms of autism persist in the same pattern well beyond childhood and even into adulthood, as is the case for stereotypies. In addition, the difference between age-mates and children with autism in relation to the developmental areas affected becomes clear only at older ages in many developmental fields. It is only after early infancy (8 months or later) that symptoms related to problems in areas such as spoken language, proxemia [c], mental flexibility, psychomotor skills, praxis and imaginative play emerge clearly. Before this age the child has not matured enough to display problems in these fields of development. Particularly the age at which the onset of language performance is expected is critical. In milder cases of autism, preserved formal language can cause delays in making a diagnosis.

Autism typically comprises a constellation of symptoms from different behavioural domains such as sociability [note 2] in addition to problems in verbal and non-verbal communication, imagination, field of interest and activity. Additional diagnostic difficulties emerge as a result of the unclear causal link between these different symptoms, all of which are considered to be ASD [note 3]. It is possible that a contact problem may arise even though the child wants to communicate with others, caused, for example by a lack of expressive language, blindness or deafness, while in other cases the same problem is the result of strong social avoidance behaviour, deficient attachment or a lack of social motivation. All the accompanying symptoms reinforce the core problem, which is deviant contact and sociability. Accompanying symptoms, such as narrow interests, atypical sensory behaviours and stereotypies, tend to be more persistent than problems in social communication and social behaviour, and are more pervasive and disruptive to the life of the child and its parents. During adolescence additional problems of normal puberty further complicate autism.

Epileptic seizures sometimes become the most troubling symptom in autism both for parents and professionals. Since undetected and untreated seizures may hinder the consolidation of attained abilities, it is vital for
there to be cooperation and communication between parents, educators, and physicians so that an early diagnosis can be made.

Secondary behavioural and emotional problems usually appear or come to the foreground in the presence of the inappropriate attitudes of the social environment or they are triggered by adverse circumstances. These problems become sometimes the most distressing problems.

Co-morbid conditions frequently accompany ASD. Intellectual disability is the main complicating co-morbidity of autism. Other common comorbidities include ADHD or secondary behavioural problems such as aggression, which complicate diagnosis and management.

Autistic regression is another important issue in relation to the late symptomatology of autism. Autism is a dysgenetic syndrome in that the pathological processes involved continue long after birth and then reach a plateau. It has recently become clear that many (probably all) cases undergo subtle regressions. Obvious regression is seen in one third of all cases. In some individuals, who later display autistic symptoms, pre-morbid development is completely normal.

In some children the symptom configuration of autism changes when they reach school age and behavioural, learning and emotional problems dominate the clinical picture. A number of autistic children are not diagnosed early and it is only when they are older, usually once they have started school, that bizarre communication and relationship patterns, psychiatric problems and learning disabilities lead to a diagnosis. Some autistic cases such as those with AS, may not have exhibited any developmental language delay in terms formal language. Some children have either mild or subclinical symptoms of autism so that they display observable autistic behaviour only when they are confronted with specific tasks at school age.

School psychologists, teachers and others should recognize the features described below as indications that a child may have an ASD, particularly AS, and if this is suspected a referral pathway needs to be implemented so that specialists can be consulted. Some mild cases of autism escape diagnosis and some may even be diagnosed after death. Milder cases may present with different problems such as problems in peer relations, or attention and learning difficulties (note 4). The mildest cases of AS are sometimes only recognized after an episode of mid-age depression or marital problems and discordances. The concept of ‘broader autism phenotype’ defines the widest boundaries of ASD. Not all researchers accept that AS is a medical disorder (Baron-Cohen, 2000) and some feel that it is instead a personality trait (note 5). Recovery from autism can be expected in some cases but the prognosis is far worse for those who do not have useful language and who have an intellectual disability. Early intervention increases adaptability and improves quality of life.
1 Although neuronal migration ends towards the end of the 5th gestational month, myelination lasts until 36 to 40 months of age (Parazzini et al., 2002). Using MRI, hyperintensities are found as terminal zones of myelination up to the age of 20 (Barkovich, 2005), while changes in synaptic functions occur until the end of life (Evrard et al., 1992; Sarnat, 1998; Lebel et al., 2011). Details are addressed in ch. 1.

2 The various elements and different components of sociability (social relatedness) and problems related to this area are described in detail in chapters 6 and 10.

3 The co-morbidities, such as ADHD, and secondary behavioural problems, such as aggression, which often accompany autism are an additional complication in arriving at a diagnosis.

4 Given the wide range in the constellation of symptoms and in their severity, a diagnosis of ASD must be based on an assessment of multiple domains and take information from a variety of sources into account. Evaluation entails a parent interview and observation of the child’s behaviour in a social-communicative context during play or peer interaction. It is the job of the experienced clinician to integrate information from several sources. No single result (e.g., a borderline score on one instrument and a high score on another) is sufficient. Follow-up is needed before a definitive diagnosis can be made (Lord & Bishop, 2010).

5 Asperger syndrome (AS) is not present as a separate disorder in the DSM-5 but is one of the ASDs. For practical reasons it will be retained in this book. Indeed, what is urgently needed is a comprehensive, coherent theory of ASD, which relates different clinical symptoms and signs of autism in a meaningful frame.

Table 11-1. Major age-related symptoms and signs of autism

<table>
<thead>
<tr>
<th>18 months–3 years</th>
<th>3 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Avoids or ignores other children when they approach or has odd approaches to other children.</td>
<td>• Spoken language is not communicative; does not stick to the norms of turn-taking, fails to show consideration for others.</td>
</tr>
<tr>
<td>• Limited variety of imaginative play and pretence, especially social imagination (does not participate in shared imaginary games)</td>
<td>• Does not play with other children. Parents often say their child plays side-by-side with other children.</td>
</tr>
<tr>
<td>• Failure to initiate simple play with others and to participate in early social games</td>
<td>• Cannot comfort someone else.</td>
</tr>
<tr>
<td>• Preference for solitary play activities.</td>
<td>• Facial expressions are inadequate and do not show anger, happiness, sadness, fear</td>
</tr>
<tr>
<td>• Does not show toys and other objects to others</td>
<td>• No pretend play</td>
</tr>
<tr>
<td>• Play is repetitive and stereotyped (e.g., lining up blocks or toy cars)</td>
<td>• Dislikes changes, e.g., in the physical environment</td>
</tr>
<tr>
<td>• While playing, does not perform actions which involve an interlocutor (e.g., pretending to talk on the phone, playing with dolls)</td>
<td>4 and older</td>
</tr>
<tr>
<td>• Has compulsions and rituals (will only perform actions in a special way or in a certain sequence)</td>
<td>• Understands only the literal meaning of utterances; fails to understand jokes and figurative language.</td>
</tr>
<tr>
<td>• Does not use sentences of at least two words</td>
<td>• Still confuses personal pronouns (I/you)</td>
</tr>
<tr>
<td>• Echolalia (constant literal and pointless repetition)</td>
<td>• Does not have friends, or has few friends and cannot keep them</td>
</tr>
<tr>
<td>• Does not look in the right direction when called, or when someone points to something</td>
<td>• Does not engage in thematic play with others (school, grocery store)</td>
</tr>
<tr>
<td>• Does not spontaneously imitate</td>
<td>• Does not engage in pretend/symbolic play at 5 years of age</td>
</tr>
</tbody>
</table>

Is extremely shy. 

Diagnosed as learning disabled at school

Often the causative agent of an early symptom does not disappear, but is instead expressed in a different way. For early symptoms of autism (toddler period) see tab. 9-v.
**The Clinical Core Symptoms of Developmental Dysphasia**

Luria (1977) saw the theory of aphasia as the most difficult domain of neurology, writing that it requires knowledge in several branches of science. In fact, although developmental language disorders (DLD), notably developmental dysphasia (DD), are often seen in child neurological and psychiatric clinics, these disorders are in most cases not always understood by inexperienced practitioners, therapists and psychologists.

We feel that it is important for child psychiatrists to recognize DD/DLD because in many children they lead to a psychopathology and there may be co-morbidity such as dyspraxia, autism or ADHD. DD is important to know about for school psychologists and school doctors as well, because it is one of the main causes of learning disorders. DD has far-reaching consequences.

Our first concerns are the concept of DD, relevant terminology, the clinical core symptoms, and DD subtypes.

DD can occur within the framework of neurological and neuropsychological co-morbidity. Therefore there is a need for a nosological framework to understand what the nature of DD is, how it comes into being, and when it can be expected that the disorder will occur. Acquired aphasias are a form of childhood speech-language disorders, and if acquired early, they often behave as DD and require treatment.

The neurobiological background of DD is our next concern. The neural networks that play a role vary (they may pertain mainly either to the left temporal and frontal lobe or the corpus callosum), and are influenced by maturation. In the clinical setting information about the familial-genetic basis of the disorder as well as the contributing environmental factors needs to be shared with the parents in psycho-education, which is an integral part of the treatment process.

The last part of this chapter is devoted to the diagnostic process. Treatment will be addressed in chapter 13.7.

**Speech and language terminology** | A *phoneme* is the smallest speech sound unit. In ontogeny, infants first hear vowels and very soon afterward begin to produce them as well as consonant-vowel combinations thereafter. *Phonology* is the study of how sounds are organized and used in natural languages. In standard generative phonology, distinctive features are central. A stream of speech is portrayed as linear sequences of discrete sound-segments. Each segment is composed of simultaneously occurring features. *Phonetics* is the basis for phonological analysis, and is the study of speech sounds as (psycho-) physical events, regardless of language. At the age of 6 months infants produce nearly all sounds (universal babbling as consonant-vowel combinations). After this, during the canonical babbling stage infants (7 to 10 months) also listen to themselves and, at around 9 months, make mother- tongue utterances.
After this stage infants, entering Tan’s early verbal stage [ch. 8.5], assemble phonemes into morphemes, the first monosyllabic words. These morphemes can be used to synthesize larger words and plurals according to the morphologic grammar. As soon as toddlers assemble words into 3-word combinations, small sentences, they start using syntactic grammar. Spoken language, however, has to be connected with other knowledge domains and has to be logically used, i.e. it must have meaning (the semantic aspect) and must be spoken with the correct intonation (linguistic prosody). Lexical semantics focuses on individual lexical items (words), whereas propositional/relational semantics focuses on the meaning of whole sentences. The study of spoken language includes its use in a social context, with reciprocity (pragmatics of language), an appropriate emotional intonation (emotional prosody), and adequate body language (kinesics).

The phonological, morphological, syntactic, semantic, prosodic and pragmatic aspects of language are controlled by developing neural substrates and may be deviant during speech-language acquisition. There is a spectrum of developmental language disorders (DLD) which is classified in subtypes [tab. 12-1].

Broca (1861) discovered the brain area which caused what he called ‘aphémie’, a term corrected by Trousseau into ‘aphasie’ (Ryalls, 1984). Aphasia (αφασια) is Greek for speechless. In adults, we speak of aphasia if cerebral damage, usually of the left hemisphere (L.H), has impaired the speech production or the language comprehension of a person whose neuromuscular functions have otherwise remained intact. Extensive literature is available on adult aphasiology (Benson, 1985; Hécaen & Albert, 1978, Davis, 2012).

Childhood aphasia is the disorder that develops (sub) acutely from brain damage at a stage when speech has already been completely or partially acquired (Woods, 1985).

In developmental dysphasia (DD) (Tan, 2004), the most frequently occurring disorder of the DLD spectrum (Rapin & Allen, 1982; 1986), several aspects of speech-language fail to develop in a typical way and can remain dysfunctional throughout life. The term (developmental) dysphasia indicates by definition, like (developmental) dyslexia and dyscalculia, a neurodevelopmental nature and its study belongs to the domain of aphasiology. Terms such as specific language impairment (SLI) are neutral in terms of a neurological pathogenesis and development. The New York child neurologist Isabelle Rapin and the neurolinguist Doris Allan (1982), who studied children with DLD for a number of years, initially used the term developmental dysphasias, but later used DLD, in part in response to the recommendation made by the the DSM’s classification committee (DSM-IV, 1994).
13 Treatment principles C. Njiokiktjien, B. Korkmaz and C.A. Verschoor

SECTIONS, FIGURES AND TABLES

13.0 Although for practical reasons we use the terms ‘mind’, ‘mental’ and ‘body’, what we mean by ‘mind’ is not what Descartes understood as res cogitans, as distinguished from res extensa, i.e. the body, including the brain. In the global description of the mental apparatus/mind that follows, we present a view that is both clinical and (neuro)philosophical.

Brain and body are entirely integrated in each other’s service—what we call the brain-body entity (B-B E)—maintaining psychophysical homeostasis [G'], adapting to the world of conspecifics, and to surrounding culture and nature. Adaptation means that from a first-person perspective, ‘I’ and my mind—a phenomenological self grounded in the B-B E, —uses agency and undergoes and understands agency in the form of action and speech. Humans adapt to and communicate with the world via cognitive instruments such as speech and action: this entails functions such as perception, attention regulation and memory, which belong to the procedural systems whose neuropsychology was first studied and described by Luria.

Agency in human beings is driven by enigmatic internal forces for survival (what Freud called Triebe), which are primitive drives such as (food) seeking, aggression and sexual activity. Furthermore, inborn, automatic basic emotions, such as fear, anger, happiness and sadness (Darwin), and more elaborate ones, such as jealousy motivate agents’ behaviour. These emotions are perceived, felt and expressed by the body, a process collectively called emotion cognition [ch. 2.1].

The survival systems include homeostatic and sociostatic systems. While the former maintain temperature, heartbeat, respiration and metabolism in a state of equilibrium, the latter are related to social interaction, providing balance between the seeking, care, fear and rage systems that enable humans to seek and care for friends and partners but also to keep enemies at a distance [see Panksepp in 2.1.1].

Humans are also able to ‘look inwards’, a reflective mental activity which involves the I. The conscious I (Cogito ergo sum) experiences the world and the emotions associated with its experience, called (emotional) feelings; these are imprinted and unconsciously stored as implicit memory [ch. 2.1]. We are also able to speak about and analyse feelings, as happens during psychotherapy; we can make plans.

All these behaviours, together with many others, are called mind, which is a semantic as well as a cultural construct in the Vygotskyan sense [Introduction to this volume, fig. 1, and note 14, Intro-2]. Human beings are aware of this and can analyse their behaviour: they have (self)consciousness.

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Because the mind is grounded—we could describe it as being embedded) in the B-B E—, it comprises a number of components, i.e. various functions and instruments; thus things can go wrong. There can be non-optimal programming of the brain or even physical damage with the result that the person cannot cope with daily life’s requirements and challenges, especially in the domain of social relatedness. If the I feels that it is suffering from deficiencies, it looks for help.

In this chapter we briefly summarize treatment principles {note}. Therapeutic approaches in general aim to improve the patient’s ability to function, while focussing on social relatedness, which has often been traumatized in one way or another. Sometimes the deficiency is a disorder, such as severe autism (often with severe mental deficiency), a personality disorder, posttraumatic stress disorder, severe attachment disorder, depression or psychosis, and requires treatment. In many more children these disorders are present in moderate forms, the reason why they are called spectrum disorders. In other children treatment aims to improve their procedural instruments for social relatedness, speech and motor functions or the attention regulation and memory sub-functions that are related to them, which also presents on a spectrum of severity. Treatments are meant to improve physical well-being or relieve feelings of ‘unhappiness’; this may concern subtle mood disorders or bodily feelings, indirectly affecting social relatedness {►}.

Therapeutic approaches can be divided into talking cures and body-related non-verbal cures, or a combination; a third therapeutic category affects directly our brain function (e.g. medication or neuro-feedback).

Although we try to give a near-complete list of possible treatments, we do not devote equal attention to all forms of therapy. As early problems in social relatedness and theory of mind, language disorders, and autism are main topics, therapeutic approaches for these problems and disorders, especially the concept of mentalization, have priority.

Primary neurological deficits with ensuing mental problems, which are the cause of adult disorders such as Alzheimer’s disease, Parkinson’s disease, multiple sclerosis and stroke, are not addressed.

In children, most primary neurological deficits, whether as a result of birth trauma, vascular accidents or postnatal head trauma, or with classified or unclassified mental deficiency, and/or with epilepsy, go along with neuro-psychological deficits. They are, however more rare than developmental disorders with a familial-genetic basis, such as ADHD, autism or developmental dysphasia. Mood disorders and psychoses are rare [listed in tab.13-1]. A substantial number of complaints and behavioural problems (or disorders) are caused by mental trauma and neglect in infancy or later {►}.

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13.8 MANAGEMENT AND POTENTIAL BENEFITS OF EARLY INTERACTIVE INTERVENTION IN AUTISM

13.8.1 Educational / behavioural interventions

13.8.2 Treatment of Theory-of-Mind deficits; mentalizing

13.8.3 The effectiveness of treating ToM skills

13.8.4 Pharmacological interventions

13.9 WHY NEURO-CLINICAL THINKING CONTRIBUTES TO TREATMENT: CLINICAL EXAMPLES

All activities in life, in particular therapeutic approaches, involve the systems of the mental apparatus, in other words the B-B E.

It is our conviction that if a (psycho)therapist is asked by a patient for advice, whether other therapeutic approaches may be indicated—and there are ones that would not be harmful—, the therapist should take into consideration their patient’s wish to participate in one or another therapeutic activity, also if this is beyond the therapist’s scope. It is with this in mind that this chapter provides information on the many approaches that can be considered.

► for developmental disorders see ch. 4