See also AUDITION; AUDITORY ATTENTION; CONDITIONING AND THE BRAIN; PHONOLOGY, NEURAL BASIS OF; SPEECH PERCEPTION

—Gregg Recanzone

References


Further Readings


Autism

A developmental disorder of the brain, autism exists from birth and persists throughout life. The etiology of the disorder is still unknown, but is believed to be largely genetic, while different organic factors have been implicated in a substantial proportion of cases (for reviews see Ciarnello and Ciarnello 1995; Bailey, Phillips, and Rutter 1996). Autism was identified and labeled by Kanner (1943) and Asperger (1944).

The diagnosis of autism is based on behavioral criteria. The chief criteria as set out in ICD-10 (WHO 1992) and in DSM-IV (APA 1994) include: abnormalities of social interaction, abnormalities of verbal and nonverbal communication, and a restricted repertoire of interests and activities. Behavior suggestive of these impairments can already be discerned in infancy. A recent screening instrument, based on a cognitive account of autism, appears to be remarkably successful at eighteen months, involving failure of gaze monitoring, protodeclarative pointing, and pretend play (Baron-Cohen et al. 1996). These appear to be the first clear behavioral manifestations of the disorder. Contrary to popular belief, failure of bonding or attachment is not a distinguishing characteristic of autism.

The autistic spectrum refers to the wide individual variation of symptoms from mild to severe. Behavior not only varies with age and ability, but is also modified by a multitude of environmental factors. For this reason, one of the major problems with behaviorally defined developmental disorders is how to identify primary, associated, and secondary features. Three highly correlated features, namely characteristic impairments in socialization, communication, and imagination, were identified in a geographically defined population study (Wing and Gould 1979). These impairments appear to persist in development even though their
outward manifestation is subject to change. For example, a socially aloof child may at a later age become socially interested and show “pestering” behavior; a child with initially little speech may become verbose with stilted, pedantic language. The triad of impairments appears to be a common denominator throughout a spectrum of autistic disorders (Wing 1996).

The prevalence of autistic disorder has been studied in a number of different countries, and is between 0.16 and 0.22 percent, taking into account the most recent estimates. Males predominate at approximately 3 to 1, and this ratio becomes more extreme with higher levels of ability. The prevalence of a milder variant of autism, Asperger syndrome, is estimated as between 0.3 and 0.7 percent of the general population on the basis of preliminary findings. These individuals are sometimes thought to be merely eccentric and may not be diagnosed until late childhood or even adulthood. Because they have fluent language and normal, if not superior verbal IQ, they can compensate to some extent for their problems in social communication.

MENTAL RETARDATION, a sign of congenital brain abnormality, is one of the most strongly associated features of autism; IQ is below 70 in about half the cases, and below 80 in three quarters. Epilepsy is present in about a third of individuals, while other neurological and neuropsychological signs are almost always detectable (for reviews see Gillberg and Coleman 1992). Postmortem brain studies have shown a number of abnormalities in cell structure in different parts of the brain, including temporal and parietal lobes, and in particular, limbic structures, as well as the CEREBELLUM. Findings indicate a curtailment of neuronal development at or before thirty weeks of gestation (Bauman and Kemper 1994). No consistent and specific structural or metabolic abnormalities have as yet been revealed, but overall brain volume and weight tend to be increased.

A genetic basis for autism is strongly indicated from twin and family studies favoring a multiplicative multilocus model of inheritance, perhaps involving only a small number of genes (reviewed by Bailey et al. 1995). There is evidence for a broader cognitive phenotype with normal intelligence and varying degrees of social and communication impairments which may be shared by family members. Other disorders of known biological origin, such as fragile X-syndrome, phenylketonuria, tuberous sclerosis, can lead to the clinical picture of autism in conjunction with severe mental retardation (Smalley, Asarnow, and Spence 1988). There is no known medical treatment. However, special education and treatment based on behavior management and modification often have beneficial effects (see chapters in Schopler and Mesibov 1995). Whatever the treatment, the developmental progress of children with autism is quite variable.

Cognitive explanations of the core features of autism provide a vital interface between brain and behavior. The proposal of a specific neurologically based problem in understanding minds was a significant step in this endeavor. The hypothesis that autistic children lack the intuitive understanding that people have mental states was originally tested with the Sally-Ann false belief paradigm (Baron-Cohen, Leslie, and Frith 1985). This impairment has been confirmed in a number of studies (see chapters in Baron-Cohen, Tager-Flusberg, and Cohen 1993) and has become known as the THEORY OF MIND deficit. Most individuals with autism fail to appreciate the role of mental states in the explanation and prediction of everyday behavior, including deception, joint attention, and those emotional states which depend on monitoring other people’s attitudes, for example pride (Kasari et al. 1993). The brain basis for the critical cognitive ability that enables a theory of mind to develop has begun to be investigated by means of functional brain imaging (Fletcher et al. 1995; Happé et al. 1996). Other explanations of social communication impairments in autism have emphasized a primary emotional deficit in INTERSUBJECTIVITY (Hobson 1993).

The nonsocial features of autism, in particular those encompassed by the diagnostic sign restricted repertoire of interests, are currently tackled by two cognitive theories. The first proposes a deficit in executive functions. These include planning and initiation of action and impulse control, and are thought to depend on intact prefrontal cortex. Evidence for poor performance on many “frontal” tasks in autism is robust (Ozonoff, Pennington, and Rogers 1991; Pennington and Ozonoff 1996). For instance, individuals with autism often fail to inhibit prepotent responses and to shift response categories (Hughes, Russell, and Robbins 1994). Poor performance on these tasks appears to be related to stereotyped and perseverative behavior in everyday life. The site of brain abnormality need not necessarily be in prefrontal cortex, but could be at different points in a distributed system underlying executive functions, for example the dopamine system (Damasio and Maurer 1978).

A second cognitive theory that attempts to address islets of ability and special talents that are present in a significant proportion of autistic individuals is the theory of weak central coherence (Frith and Happé 1994). This theory proposes that the observed performance peaks in tests such as block design and embedded figures, and the savant syndrome, shown for instance in outstanding feats of memory or exceptional drawing, are due to a cognitive processing style that favors segmental over holistic processing. Some evidence exists that people with autism process information in an unusually piecemeal fashion (e.g., start a drawing from an unusual detail). Likewise, they fail to integrate information so as to derive contextually relevant meaning. For instance, when reading aloud “the dog was on a long lead,” they may pronounce the word lead as led.

Clearly, the explanation of autism will only be complete when the necessary causal links have been traced between gene, brain, mind and behavior. This is as yet a task for the future.

See also COGNITIVE DEVELOPMENT; FOLK PSYCHOLOGY; MODULARITY OF MIND; NEUROTRANSMITTERS; PROPOSITIONAL ATTITUDES; SOCIAL COGNITION

—Uta Frith

References


Further Readings


Autocatalysis

See SELF-ORGANIZING SYSTEMS

Automa

An automaton (pl. automata) was originally anything with the power of self-movement, then, more specifically, a machine with the power of self-movement, especially a figure that simulated the motion of living beings. Perhaps the most impressive such automata were those of Jacques de Vaucanson (1709–1782), including a duck that ate and drank with realistic motions of head and throat, produced the sound of quacking, and could pick up cornmeal and swallow digest, and excrete it.

People acting in a mechanical, nonspontaneous way came to be called automata, but this begs the very question for which cognitive science seeks a positive answer: “Is the working of the human mind reducible to information processing embodied in the workings of the human brain?” that is, is human spontaneity and intelligence a purely material phenomenon? René DESCARTES (1596–1650) saw the functioning of nonhuman animals, and much of human function, as being explainable in terms of the automata of his day but drew the line at cognitive function. However, whereas Descartes’s view was based on clockwork and hydraulic automata, most cognitive science is based on a view of automata as “information processing machines” (though there is now a welcome increase of interest in embodied automata).

The present article describes key concepts of information processing automata from 1936 through 1956 (the year of publication of Automata Studies by Shannon and McCarthy), including Turing machines, finite automata, automata for formal languages, McCulloch-Pitts neural networks, and self-reproducing automata.

TURING (1936) and Post (1936) introduced what is now called a Turing machine (TM), consisting of a control box containing a finite program; an indefinitely extendable tape divided lengthwise into squares; and a device for scanning...