"What becomes of autistic children when they grow up?" (Leo Kanner, 1942)

Abstract

An historical context for treatment and four adult patients not previously diagnosed with autism are discussed in order to identify a unique psychiatric contribution to differential diagnosis, management, and therapy at a combined mental retardation - psychiatric unit at Hutchings Psychiatric Center in Syracuse NY in 1979

Introduction

Early reports of combined mental retardation and psychiatric impairment appear between 313-238BC in the Chinese medical writings of Xunzi. One patient is described who suffers from a mental handicap with what appears to be a superimposed stress psychosis (Liu, 1981).

In early 19th Century France, Phillipe Pinel (1745-1826) categorizes four primary mental maladies common in medical institutions: Mania, Melancholia, Dementia and Idiocy. In 1806 he offers the first detailed observations of co-morbid disability in a series of young patients quartered in Bicetre and Salpêtrière. His patients suffer from extremes of intellectual retardation and emotional and behavioral disruption (Pinel, 1798, in Lane, 1976)

Pinel and physician Jean Marc Gaspard Itard (1774-1838) are both influenced by
the sensory-perception educative theories of John Locke (1632-1704) and Etienne Bonnet de Condillac (1714-1780). But Pinel regards Itard's therapeutic efforts with Victor, L'Enfant Savage (the Wild Boy of Aveyron), with profound scepticism. He believes Victor to fall in the class of "Idiot" and hence not educable. In contrast, Itard views a “savage” with the innate human capacity to learn civilized ways through systematic sensory-perceptual-motor and social adaptation retraining. After a long arduous personal training effort with Victor, Itard concedes defeat.

At Salpetriere in 1841, only three years after Itard’s death, Edouard Seguin (1812-1880) commences his own attempt at Victor’s cure (Lane, 1976). Over the next half-century Seguin’s work on numerous patients achieves considerable therapeutic success. Nevertheless, the prestige and therapeutic pessimism of Pinel and his psychiatrist disciple Jean-Étienne Dominique Esquirol (1772-1840) prevail.

A deep professional divide emerges; psychology focused on the impact of cognitive disability in infants and children; and psychiatry, intervening in adults presenting with emotional distress and behavioral disturbance (Masland, 1958). Both professions eschew curative attempts in favor of diagnosis, internment and psycho-social morality programs in large state schools and psychiatric hospitals. By the late 19th century, standardized tests for intelligence favor the psychologists role in the diagnosis and behavioral management of mental retardation. Pediatricians are retained for consultative medical issues. Active psychiatric involvement is minimized or absent. (Potter, 1927).

Early and mid-20th century clinical psychologists adopt refashioned reward/punishment management, in the new “science” of operant-conditioning and special education based upon contemporary learning- During the same period, psychiatry devolves
into two largely separate enterprises, each claiming marriage to medicine: entrepreneurial private practice with emphasis on individual interpretive accounts termed “analysis”; and a public tax-funded speciality promoting public security through social constraints favoring patient isolation and physical restraints including and pharmaceuticals and straight-jackets. (Gardener, 1978).

In 1942 the work of physician-psychiatrist Leo Kanner (1896-1981) on infantile autism sparks psychiatric interest in children with delayed development and psychotic features (Kanner, 1942). After WWII, the projections of psychologist Bruno Bettelheim (1903-1990) with respect to flawed maternal child-raising capture the public imagination and generate a new host of potential causes and remedies for autism (Bettleheim, 1957).

**Unit Treatment Setting**

In 1978 I joined “The Skills For Living" secure unit at Hutchings PC as in-patient team psychiatrist. Other team members included an administrator, psychologist, social worker, and psychiatric nurse, assisted by licensed physical, recreational, and occupational therapists and rotating support staff working both in-hospital and in interim patient housing (half-way house).

This program was initiated in November 1975 as a cooperative venture of the Syracuse Developmental Center (a part of NYS OMRDD; Office of Mental Retardation/Developmental Disability) and Hutchings Psychiatric Center (NYS OMH; Office of Mental Health).

Adult patients exhibiting severe emotional and behavioral disorder in association
with mild mental retardation (IQ range of 50-70) are admitted for up to 3 months for
diagnosis and treatment. Treatment consists in behavioral conditioning, adaptive skills
training with token economy, supportive psychotherapy, community socialization, and
appropriate biological and holistic interventions, with volunteer support from the general
and Syracuse University community, the latter directed by a Hutchings in situ sociologist.
(Many, 1979)

Selected Patients Four young mildly retarded adults, age 18-41, were selected for
psychiatric work-up due to severe episodic long-standing emotional discharge and
misbehavior. Psychiatric-medical-conditions including inborn errors of metabolism,
endocrinopathies, and neurologic conditions emerged in the psychiatric evaluation.

(1) AJ is a 35 year-old female with a pronounced developmental lag present shortly after
birth and episodic psychotic behavior since her early teens.

Observation: intermittent, labile, explosive affect not associated with environmental
cues; attitudinal inattention and negativism, stereotyped movements including whirling,
swaying, and head-rolling; defects in speech and communication improving during singing
and lyric production. Diminished eye contact and general defects in social and peer
relations. No seizures.

Physical exam: large pigmented areas over back and lower spine. Numerous facial
para-nasal spotty erythematous raised small (less than one cm.) lesions.

Cranial X-ray: Diffuse skull calcifications

EEG: normal
Diagnosis: Tuberous Sclerosis/Epiloia; Anxiety/Panic (episodic, moderate-severe)

Treatment: neuroleptic and anxiolytic medication; high dose vitamin B-complex; Behavioral conditioning; supportive social training

Outcome: sporadic improvement in behavior interrupted by episodic emotional and behavioral regression

(2) BL is a 34 year-old white female with marked developmental lag, 17-year history of aggressive un-provoked "attack" behavior, obsessive ideation, and ritual compulsions included extended showering/washing

Observation: intermittent aggression; eye contact elicits jumping, yelling, screaming, followed by dissociated flat-affect. Acute attack with repetitive clawing and slapping of nearby persons, sometimes accompanied by breaking eye-glasses, both her own and nearby staff and peers. Poorly developed language expression, paucity of ideas and expression with marked ideational and semantic repetitions

Physical exam: short stature, truncated fourth digits bilaterally, foreshortening of the trunk and neck and synophrys (heavy eyebrows meeting midline).

Lab testing: increased parathyroid hormone, calcium range abnormalities.

Diagnosis: PseudoPseudoHypoparathyroidism (PPH); Obsession-Perseveration (Organic); Learning Disability

Treatment: Behavioral, Psychosocial, Supportive. (Calcium EDTA, vitamin D for PPH not initiated)

Outcome: partial remission
(3) CM is a 24 years-old white male

**Observation.** Lack of eye contact, social distancing, hoarding, stereotypic “tic” motor patterns primarily in the upper extremities, echoalia (repeat external sounds) and coproalia (spontaneous swearing); sporadic hyper-sensitivity to random stimuli; sporadic aggression (personal attacks).

**Labs:** normal EEG

**Diagnosis:** Tourette Syndrome; Social Anxiety/Aggression

**Treatment:** haloperidol; behavioral conditioning; socialization, supportive psychotherapy

**Outcome:** mild diminution in frequency and intensity of aggression.

(4) DV is a 22 year-old white female

**Observation.** intermittent marked social withdrawal with catatonic features; fluctuating mood; occasional urinary and fecal incontinence; marked speech disturbance; lack of eye contact

**Diagnosis.** Schizophrenia, Catatonic; Capgras Syndrome (delusional disorder with imposter/ misidentification); de Lang syndrome (emotionless attack);

Pseudopseudohypoparathyroidism (PPH)

**Treatment.** psychotropic medication; behavioral-social retraining

**Outcome.** temporary sporadic improvement, not clearly related to interventions

**Clinical Correlates Summary**

The unreliability of developmental histories is well recognized (Cantwell, 1980).
Longitudinal study of retained capacity, intervention effects, and social maturation in mental retardation is limited (Darr, 1951). All of our patients were diagnosed with mental retardation prior to school entry age.

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Three patients presented with early onset medical disorders, namely Tuberous Sclerosis, Pseudopseudohypoparathyroidism (PPH), and Tourette syndrome (Shapiro, 1973). Patient number four had a clear onset of substantial psychiatric disorder in the teen years. She was initially diagnosed as Catatonic subtype of Schizophrenia, with delusions and aggression consistent with Capgras and de Lange syndromes. Lab testing revealed PPH. The coexistence of schizophrenia with autism could not be ruled out.

Motor disturbances were present in all four, repetitions in three who demonstrated both echolalia (repeated echoing of another’s vocalization) and echopraxia (repeated imitation of another’s movements).

General Discussion

Autism is a disturbance with onset before the age of 20 months and the following anomalies:

(1) Perceptual. Hypo or hyper-sensitivity to stimuli such as sudden loud noises,
bruises, cuts, and injections..

(2) **Developmental.** Delay, disruption, spurt and/or lag, generalized disability with special retained abilities.

(3) **Relational.** Episodic aversion, aggression, disrespect, disregard, inattention to parents/peers is common. Physical contact may be avoided, social smile delayed or absent, eye contact infrequent or deviating from social norms.

(4) **Speech and language.** Atonal, arrhythmic, lacking inflection, context-unusual emotional expression including guttural or loud sounds, yells, laughter.

(5) **Motor.** Mannerisms and repetitions: rubbing, banging, flicking, teeth grinding, whirling, rocking, head banging and rolling, hand flapping, trunk oscillations, toe walking, lunging, darting, rocking, swaying, immobility, and posturing (Ornitz, 1976)

Central nervous system (CNS) dysfunction in autism is generally regarded as secondary to deficiencies in higher cortical integration. Disorders as diverse as PKU, Tuberous Sclerosis, Rubella, and Encephalitidies may have a similar presentation (Tanguay, in Syzmanski, 1979).

Autism occurs in persons across a full range of intellect: sub, normal, or above normal. (Kanner, 1943). Concurrent psychiatric impairment with emotional and social/behavioral disability may be exacerbated by selective cognitive defects, confusion, misunderstanding, and/or frustration resulting from impaired manipulation of formal symbols and language (Rutter, 1970) or delayed expression of genetic, constitutional, and developmental variables. Learning disabilities may be a significant unrecognized factor in both autism and psychosis (Wing, 1979).
While normal motor development also includes many motor repetitions also characterized as autistic (Tinbergen, 1974), persistent exaggerated repetitions frequently attend the diagnosis of autism (Freeman, 1981). Diagnosis of autism in adults may be problematic as a result of infrequent review, intermittent or episodic dysfunction, and normative delays in socialization as a result of learned dependency and inexperience in problem solution.

Emotional discharge-display and expectations regarding conversational give-and-take are grounded in social expectation. One signal sign of autistic behavior, eye contact, is a highly culture-specific variable. Acting out (misbehavior, rule-breaking) is a virtual norm in teens and peers due to boredom and frustration facing sexual and social maturation. Maturation is further conditioned on age-related social peer recognition, stigma, class, status, and learning stimuli.

Phasic modulation with respect to the working criteria of autism is under-reported (Ornitz, 1976). In analogy with seizure disturbance this was considered in all patients. Synodic and diurnal frequency variations were charted for potential endocrine mediated hormonal alteration, well acknowledged in PMS (Pre-Menstrual Syndrome). Such evaluation was inconclusive.

A diagnosis of autism in adulthood may go unrecognized due to social development, remission with substantial language, emotional/affective control, and public policy emphasis on the categorical separation of MR and PI. Our findings in an acute care setting are consistent with retained learning capacity in mildly retarded adults and diagnosed autistic individuals over time (Rutter, 1970).

Relatively stable features of autism raise the prospect of validation through
longitudinal study (Ornitz, Rivito, 1976). But scant investigation of episodic manifestations in combined adult disability is manifest (Many, 1979). Several studies concerning course and prognosis maintain cutoff periods at early or late adolescence (Lotter, 1971; Brown, 1960; DeMyer, 1973). Other studies describe a spectrum of disorder including childhood psychosis (Bettelheim, 1967; Bender, 1971). The follow-up studies of Rutter (1970) are a noteworthy exception.

One understated role for the medically trained psychiatrist is triage and differential diagnosis. Physical and behavioral observations, as well as lab tests may present opportunities for both diagnostic and treatment alternatives. This was exemplified when PPH was diagnosed only after recognizing the significance of short stature and truncated digits in 35 year-old patient AJ.

Despite a general tendency to view the potentials for remedy or cure as problematic (Ornitz, 1976), and treatment as suppression or management (Campbell, 1975; Rimland, 1978), attending to medical pathology, reconstructing individual maturational histories, and applying individual sensory-perceptual retraining appear to hold the best potential for active remedy in adult patients with combined intellectual, emotional and behavioral impairments who meet the applicable criteria of autism.

Seth Many MD
Sharon Spring NY, 1983
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