Theory of Mind or Mindful of Theory
Brain, Mind, Developmental and Neurological Disabilities

"It is only a matter of time before
psychiatry becomes a branch of neurology"
Professor Vilayanur Ramahandran, neuroscientist

Some months ago in exchanges of correspondence, with a special needs teacher, who had recently engaged in an online conference on autism, commented on the absence of any input from neurologists during the discussions, and only two presentations on brain research. It is hoped that "the matter of time" referred to by Professor Ramahandran is sooner rather than later.
Learning delay, behaviour and mobility problems are a consequence of brain injury, and a part of a very wide spectrum of outcomes, which will be shown to include early death of the foetus, low birth weight as a consequence of early term delivery, need for caesarian section delivery, and finally sudden infant death.
In view of the concerns being expressed from diverse sections of the medical establishment, should the question be posed as to whether a more universal approach is considered, whereby the various disciplines in medical and clinical professions combine their different disciplines in a unified approach to solve what appears to be a costly and wasteful use of scarce resources.

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In the absence of a brain there can be no mind, and the brain is the sum total of all the experiences gathered by sensory systems of sight, hearing, taste, touch, and smell.
In his book entitled L'errore di Cartesio (Descartes mistake) Antonio Damasio says that "on the basis of philosophical principles which turned out to be mistaken, mankind continued to consider the mind and brain separate. Consequently, an attempt was made to describe the mind without reference to neurobiology; mental illness was described without any understanding of neurological anatomy, physiology or chemistry. Descartes mistaken distinction between res cogitans and res extensa still exercises its influence today. It has prevented us seeing that the appearance of a "mind capable of thought" and subsequently of a "more complex mind with a language capable of communicating thought" came long after the appearance of mindless beings. When we are born we are alive, and we only think subsequently. Thought is the result of our structure".

Neurology tells us that in the event that the brain is starved of sensory input it will not grow, nurture the brain and the opposite is true.
There is ample confirmation in the literature that brain function and structure can be altered. In 1979, in an article in the Journal of Learning Disabilities, Doctors Marianne Frostig and Phyllis Maslow stated, "Neuropsychological research has demonstrated that environmental conditions, including education, affect brain structure and functioning." In their book Brain Mind and Behaviour, Floyd E Bloom, a neuropharmacologist and Arlyne Lazerson, a professional writer specializing in psychology, state, "Experience [learning] can cause physical modifications in the brain." This is confirmed by Michael Merzenich of the University of San Francisco. His work on brain plasticity shows that while areas of the brain are designated for specific purposes, brain cells and cortical maps do change in response to experience (learning). It seems that, while learning causes brain growth on the one hand, the lack of learning, no the other hand, causes a lack of brain growth.

An example of lack of learning, causing a lack of brain growth, can be found in the work of Doctors Bruce D. Perry and Ronnie Pollard, two researchers at Baylor College of Medicine. They found that children raised in severely isolated conditions where they had minimal exposure to language, touch and social interactions, developed brains 20 to 30 percent smaller than normal for their age.
What we see, hear, taste touch and experience creates neural pathways in the brain and the neurons created are the means by which all these experiences are stored in the brain as memory.

In the event that the brain is disturbed then the mind is disturbed. In the nineteenth century, neuropathologists were able to carry out autopsies on deceased sufferers of psychiatric disturbance and observed macroscopic alterations in the structure of the brain.
Today, we accept without question, the presence of abnormalities affecting the Central Nervous System (CNS) of individuals exhibiting behavioural, learning and mobility problems, after a mild diffuse brain injury.
In 2001 The American Academy of Neurology (AAN) published in its Journal " Neurology" October 9th issue, a study undertaken by the PET Center at Children's Hospital of Michigan in Detroit on 26 children with tuberous sclerosis complex (TSC). Researchers used MRI and PET examinations to study how brain lesions resulted in common behaviours of autism, including difficulties in social interaction and communication and narrow and repetitive stereotyped behaviour. They found that more than one area of the brain was responsible for autistic behaviour in children with brain lesions, and that autism results from a complex combination of events in different parts of the brain, rather from one single source.
In Feb 2002 the AAN published a report of a study carried out at the Medical College of Georgia, using computerized imaging in the frontal, and temporal lobes of autistic patients, and observed minicolumnar abnormalities. A minicolumn is a basic organizational unit of brain cells and connective wiring, allowing an individual to take in information process it and respond. Thus any changes in size shape or location of the minicolumn will have an effect on the processing capacity of the brain.

A neurological dysfunction can influence only some aspects of a human being's relations with others or, as in the case of autism, the entire capacity of the individual to relate to the world around. This statement thus gives rise to the obvious question: how does brain injury cause a behavior disorder? Why does the same illness in pregnancy or perinatal asphyxia encephalopathy from dystocia childbirth, give rise in some cases to spastic tetraparesis and in others to hyperactivity and stereotypy?
The answer can be found in the functioning of the brain. A paper published by Sudath et al in the New England Journal of Medicine 1990; 322:789-794 studying anatomical abnormalities in the brains of monozygomatic twins discordant for schizophreia. The authors examined MRI scans of identical twins, one twin being normal the other diagnosed with schizophrenia. In 14 out of the 15, the twin with schizophrenia had smaller hippocampi, when compared to the normal twin. In addition, the schizophrenic twin concordantly had larger fluid filled ventricles. It is clear from these findings that even identical twins have different brain anatomy.

The outcome after brain injury is purely dependant on the size of the disturbance and position in the brain. The position of the lesion in the brain, is again dependent on the timing in the pregnancy, as, at six months of the pregnancy, the only part of the brain developed, is up to mid brain level. At this level disturbance will only have an affect on mobility and sensory motor skills, whilst between 6 and 9 months during which time the upper cortex is being formed, disturbance will affect cognitive and speech skills.
Disturbances during the first three months, during which time the only part of the central nervous system formed is the encephalitic trunk and the old brain, invariably lead to the death of the foetus and miscarriage. Survival of the foetus at this stage leads to a higher risk of an outcome of autism in early months of life.

It is important to note at this point, that, at eighteen months, the most commonly accepted age at which autism is diagnosed, coincides with the next development stage of the infant being transferred from old brain to mid brain level. The injury to the old brain results in lack of neural connections up to the mid brain and consequently development stops or is curtailed. This is borne out from the work of Dr Margaret Bauman who carried our microscopic examination of brain slices from the hippocampus of the brains of 24 deceased autistic individuals. She found that the neurons (brain cells) in this region did not have as many axons or dendrites (neural branches that extend off the body of the brain cells) as they should.

The autistic spectrum disorders are described in the DDSM-IV (Diagnostic and Statistical Manual) under "Pervasive Development Disorders" (PDD): Autistic Disorder (AD), Rett's Disorder (RD), Childhood Disintegrative Disorder (CDD), Asperger's Disorder (AS), and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS).
The diagnostic criteria for the autistic spectrum are extensive, and, unfortunately lead one to believe that autism is of psychological origin, whereas in fact, neurological dysfunction is the creator of the diagnoses.

The last decade has shown a significant rise in the diagnosis of autistic spectrum disorders. This rise is now being shown to be more than just an increased recognition of the disorder, but is shown to be as a result of real increases in actual cases of the disorder. Epidemiological studies worldwide are confirming significant increases in the numbers of brain injured children, and clinical, biological and medical studies are revealing etiology of the disorder. Studies worldwide are now revealing that the major cause of injury to a vulnerable developing brain is a result of a number of causes.

The process of child bearing is historically divided into two distinct time periods prenatal and perinatal and for the benefit of discussion on the subject of neurological disability, the post natal period of up to 1 year is crucial to the development of the infant. It may well be pertinent for the sake of the subject to consider the period prior to conception as important to the outcome of the birth.

Earlier in the discussion I alluded to “the diagnostic criteria for the autistic spectrum” as being “extensive”, I propose that conditions on the autistic spectrum being as extensive, moreover the conditions emanating from brain injury to be extremely extensive.

From clinical, biological, and neurological, evidence published over the last 20 years, there can be considered a strong likelihood of a link to brain injury and disturbances during pregnancy.
Primarily, infections which may cause the death of the foetus should be considered, followed by those which may cause brain injury compatible with survival but leading to neurological disturbances such as autism. Studies have shown that a very high proportion of mothers with autistic children risked miscarriage in the first trimester compared to the average population
For many years now there has been an accepted link between rubella infection of the mother during pregnancy with miscarriage, premature birth low birth weight and physical disability of surviving infants. New studies are showing that many more bacterial and viral infections during pregnancy can lead to similar outcomes. Recent studies now link tobacco smoking during pregnancy with increased risk of an unfavorable outcome of the pregnancy and Fetal Alcohol Syndrome is an accepted outcome of alcohol intake during pregnancy.
The recent plethora of studies into Sudden Infant Death Syndrome all recognise that smoking during pregnancy promotes a higher risk for premature births’, and low birth weight.

Perinatal causes of encephalitic disturbance, include environmental changes to which the new born baby is exposed at the moment of birth; delivery through the birth canal, mechanical trauma, hypoxy (lack of oxygen) following over rapid or over lengthy labour, leading to death or neurological effects such as autism. Caesarean section is an over rapid delivery, recently the National Institute for Clinical Excellence reported their awareness of the increase of elective C-section deliveries and their concerns. At the same time a report from the University of Western Australia, the team led by Dr Emma Glasson, suggested that children born by emergency or elective caesarean section increased the risk of an outcome of autism. Hypoxy causes selective injury to the central nervous system, affecting above all the cortex, base nuclei, and white substance of the hemisphere. The lack of oxygen in a premature foetus leads to selective injury of the periventricular blood islets of the germinal tissue. Hypoxy is also the cause of brain injury during delivery, associated with the use of forceps, strangulation with the umbilical cord.
Tonic reflux of the neck of the birth canal eases childbirth, but the lack of reflux, may lead to dystocic birth, but the lack is itself caused by a disorder of the foetus, so dystocia is not the sole cause of brain injury, researchers often encounter one cause of a brain injury which seems to provoke further vulnerability to a second pathogenic cause of brain injury.

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