Autism
Theory of Mind or Mindful of Theory
Brain, Mind, Developmental and Neurological Disabilities
Making Sense of the Senses

"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.

Brain, Mind, Developmental and Neurological Disabilities

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.

Making Sense of the Senses

"If we do not try to express any psychological or behavioral theory
in terms of neurons, we are unlikely to get to the bottom of the problem."
G.M.EDELMAN

Most autistic children are highly sensitive to changes in the volume of noise. The louder the noise, the harder it gets for the child to tolerate, until the child bursts into tears.
This is caused by a faulty mechanism by which sound is input at the level of the hearing cortex. The first layer of the hearing system in the cerebral cortex consists of neurons that are sensitive to variations in noise levels. The same neurons also distinguish one tone of voice from another: calm or angry. An anatomical or chemical injury to this layer can cause serious disturbances making it difficult to adapt to high frequency sounds.
Human beings can hear sounds between 16 and 16,000 Hz. Clearly as the sound approaches one or other of these extremes it becomes more difficult to listen to the sound, which is either extremely dull or very acute. Brain injured children, including autistic children, have a very low tolerance for high frequency sounds, particularly if the noise is also loud.
Autistic children do not tolerate high frequency sounds but love low sounds, like whispering.
A very quiet and unruffled environment can put a stop to a whole range of strange behavior, such as hiding under the table, staying under water for long periods, sleeping with a blanket over the head, fingers in the ears, making a continuous even sound, staying in the corner of the room. Many autistic children like the corners of rooms because they feel protected against sounds.

The cheek color of autistic children changes very often. Sometimes in the course of a single day they can go from a healthy red to pale gray, particularly around the eyes. This is a symptom of great suffering.
The color changes when the noise level becomes intolerable, but this level is very low for autistic children, since they are hypersensitive to sounds. When the noise level is intolerable the child begins to behave in one of the ways described earlier (hiding under the table, fingers in the ears, etc.). If the level of intolerability is very high the child uses a different mechanism that is even more effective: he becomes pale and cuts himself off from the rest of the world. Even very loud noises no longer penetrate. Call him and nothing happens. It is as though he has suddenly gone deaf. The only way to get the child to come back into the world is to whisper something in his ear. The sound is very low, so there is no need for the nervous system to maintain a high level of defense. A low sound allows the child to lower his defenses and enables him to let sounds back in.
If the child is the source of the sound, or knows that the sound will come, the nervous system gets itself ready and copes with the noise, but if the noise comes suddenly and unexpectedly the child cannot cope and, as it were, shuts down.

A child screaming also makes perfect sense. Often autistic children react in this way to show their suffering or try to use their own voice, an instrument with which they are familiar.

Switching on the TV or staring at the washing machine as it turns and tumbles are familiar traits of behavior in autistic children with an impaired sound pathway. They use this sound, which they themselves provoke, to focus on and drown out all other noises.

For many years very little hope was held out for autistic children in terms of speech ability. But nowadays we know much more about the neurological basis of speech abilities.

Antonio and Hanna Damasio have carried out very interesting studies in this area. They and their team have investigated three different neural structures related to speech. The first structure consists of a large number of neurons in both the right and left hemispheres; these neurons process sensorial inputs. The second structure consists of a numerically smaller number of neurons located mainly in the left hemisphere; these neurons are used for the recognition of phonemes, the combination of phonemes and an understanding of syntax. The neurons of the third structure are also in the left hemisphere; these mediate between the other two. For example, if you want to say a color, the neurons of the third structure place the neurons of the other two structures in communication. The first structure contains the visual sensation of the color and the second the phonemes to pronounce the word. In other words, the memory must contain the correct phonemes and the correct sensory experience. If someone does not have the visual apparatus to perceive colors, forms or movements correctly, the concept of color, form and motion will be lacking, and hence the words relating to these things will also be lacking.

Defective perception becomes defective language and unfortunately autistic children are seriously affected by defects in sight, hearing, touch, taste and smell.

If we just think back to the example of a child hyper auditory, it is not difficult to understand the problems the child has in acquiring the proper use of language.

We know that the first layer of the hearing cortex controls the volume of sounds. The second layer is used to distinguish a series of sounds, their pitch and rhythm.

The second layer enables us to understand speech. Every spoken word has two elements: volume and rhythm. The first layer of the cortex receives the sound of the word and distinguishes the volume; the second captures the rhythm. The result is that the word can be memorized and learned. The pathway is obligatory from the first to the second layer. The first layer filters sounds, especially volume, that reach the second layer and hence influences its structure. If the first layer does not carry out its filtering function properly, the second layer slows its activity and is unable to distinguish the rhythm of words and hence to distinguish and understand spoken words.

Autistic children are often unable to control the volume of the sounds they hear, although there are many different forms of this disability. In some cases the difficulty is so severe that no sounds reach the second layer; the child is “acting deaf.” In such cases the second layer is deprived of information and proper linguistic abilities are impossible. If the sensitivity to differences in volume is less pronounced, sounds do reach the second layer and hence the child is able to make certain distinctions between words. Speech is by

no means perfect but the child is often able to repeat some words or to make sounds that are similar to words. If the dysfunction is quite small, the second layer is able to carry out its functions properly and in this case the autistic child acquires superior language abilities compared to the norm. If impairment of a sensory pathway is only slight, the child develops special mechanisms to offset the disturbance, and may be able, for example, to learn an entire telephone book by heart, finish a complicated jigsaw puzzle in a few minutes or reproduce a drawing at sight, and so on. Sometimes these abilities reach extraordinary heights but the other disabilities of the child are generally so severe that the child is not independent.

Children are able to juggle objects, defy gravity. But what was most astonishing is the fact that they seemed to pay no attention whatsoever to what he was doing during these moments. Their eyes are elsewhere. Children spend hours in this way, sometimes throwing things across the room or from the balcony, or leaving them neatly arranged at the edge of the table.

It is amazing the deceptive nature of the behavior of autistic children. That way of looking askance at things was not a sign of lack of interest, but the only way children could actually see things. By looking out of the corner of their eyes they perceived the object. The juggling act was actually a way of focusing on it, seeing it better.

For many autistic children the only way they can see is to move their heads sideways and look out of the corner of the eye.

The visual apparatus of human beings is quite complex: essentially it involves breaking elements down into separate entities and then recomposing the picture as it was. Of course we are not aware that this is what we do. Every time we look at an object, a person, the scenery, a group of people, neurons analyze color, form, depth and motion. The neurons that recognize color are all located in one area of the brain, and so on, for other functions.

There are five such vision areas in the brain that for simplicity's sake we can call V1, V2, V3, V4 and V5.

The first area, V1, is also known as the layered or primary area. This area receives the images falling on the retina. These images are then passed to V2, which links up to the other areas making up the area of associative vision, or the pre-layered cortex.

Area V3 receives information from V1 and V2 concerning form. Areas V4 and V5 also receive information from V1 and V2, but V4 concerns form and color. While V5 concerns depth and movement.

Once V3, V4 and V5 have done their jobs, the message is sent back to V1 and V2 along pathways that integrate the information, so the image is recomposed.

It is as if the elements of a picture were poured into a funnel. Everything goes through the neck of the funnel and mixes together: color, form, depth and movement.

In order to see the world normally these areas must be perfectly balanced; any imbalance can cause distorted vision.

Serious damage in the V1 and V2 areas causes total blindness while damage in the pre-layered area (V3, V4 and V5) causes difficulties in perceiving form.

One of the world's leading experts on vision in human beings, Semir Zeki, has noticed that people with brain injury in the pre-layered area, who therefore have difficulty identifying forms, tend to rotate their heads in order to create movement, or prefer objects which move such as objects seen on television. Typical behavior of autistic children is to tilt the head in order to look at things out of the corner of the eye. Another is to frenetically rock or rotate an object, or to watch television programs featuring things in rapid motion, such as the list of characters, actors and crew at the end of a film. They also like to see the same scene of a cartoon over and over again, or watch quiz shows like "The Price is Right" or "Wheel of Fortune."

A lot of evidence concerning autistic children points to injury of the pre-layered cortex. This is not necessarily wrong, but there is also evidence of other types of disturbance such as the inability to distinguish between similar images, or to see small things on an object's surface or to remember a visual experience such as scenery or the route along a road.

It is therefore more correct to say that autistic children process visual inputs differently. This different perception may be associated with injury in the occipital lobe (the area related to vision) and may involve one or more areas of vision.

A slight but widespread injury at an early age may not have serious immediate consequences but certainly alters the neurological organization of the central nervous system. To make things simpler we could say that the brain of a newborn child is like a building site where the engineer is running things. An injury is like a construction event that won't change the scaffolding but causes all of the engineer's orders to be carried out slightly wrong, with consequences that may make the building unsteady or unusable. It is as if the engineer were speaking a slightly different language from the builders following his orders. The inputs are skewed, making the building out of shape. The neurons—the builders in our metaphor—simply carry out orders as best they can, building things the way they seem to have been told. The result in autistic children is a series of neurological maps that don't correspond with the real world, giving rise to antisocial and unusual behavior.

In the case of vision, only V1 has an efficient neurological organization at birth. This means that the baby uses this area immediately in order to see. But it also means that this area is the most exposed to injury during pregnancy.

In addition, the primary visual cortex is just like the engineer on the building site: all other areas of vision are organized by V1 despite the fact that the messages go through V2. If the orders coming from V1 are in this rather odd language the rest of the visual apparatus is built wrong.

Lack of perception of color could be caused by injury to the specific area dealing with color perception (V4) or to the primary visual cortex (V1) failing to give the right messages to V4. If the only defect in vision were the perception of color, it would probably be correct to assume the injury has occurred in V4. But autistic children generally have many defects of vision, so it is far more likely that V1 has been injured and the entire language of the engineer is off-cue. Usually V4 is unlikely to be injured since autistic

children generally receive injury during pregnancy or in the first few months of life, and V4 practically does not exist at this time.

So the hypothesis is that autistic children receive injury to V1 before or just after birth. Now we need to explain how this determines their behavior.

Let us recap what we know about the primary visual cortex: first, it works from birth. Second, it receives all visual inputs. Third, it communicates with all other areas. Fourth, serious injury causes total blindness. Now we can go backward over these points. Autistic children are very rarely blind Usually their injury is not severe. The layered visual cortex (V1) sends information to all other areas, determining the way in which these areas develop. In order to do this, the neurons in V1 must be able to recognize each visual input and understand where to send it. The nerve cells in V1 that recognize a stimulus are contained in the layer. Nerve cells responding to movement are found in sub-layer IV c α, the cells recognizing forms in sub-layer IV c β and other cells responding to color and spatial orientation in layers II and III.

The presence of nerve cells responding to all types of visual stimulus in, V1 means, this area of the brain is independent. In other words, with this area alone we can perceive forms, colors and movement. But this does not make for proper vision. We would find it difficult to focus on nearby objects, would see few differences in color and would only be able to see the outline of an object by moving it. We would see like a newborn child who only has the V1 area of the brain available for seeing. But in the case of a newborn child, vision improves as the functions of the pre-layered cortex come into play.

The pre-layered cortex receives its orders from V1, which is organized in layers, each one responding to a particular stimulus and each transmitting input to other areas. The nerve cells in these areas activate a series of synapses that excite and inhibit, forming a kind of circuitry.

One of the circuits called the magnocellular circuit, transports information concerning depth and movement. Another called the parvicellular circuit, transports information concerning form and color.

The magnocellular circuit starts with some rather large ganglial cells (hence the name) connected to cells on the retina that recognize movement. The signal is then projected onto the V1 sub-layer IV Cc α, where spiny star-shaped nerve cells respond to the stimulus, activating an exciter synapse. The message processed by these cells is then transmitted via V2 to V5 where specialist neurons further analyze the message.

The parvicellular circuit starts with some rather small ganglial cells (hence the name) connected to cells on the retina that recognize form and color. The signal related to form is projected to the sub-layer of V1, IV c β, and to some cells in layers II and III where the remaining cells respond only to color. The messages processed by these sub-layers are sent to V3 and V4 respectively.

The cells contained in sub-layer IVC b are exciters, but those in layers II and III excite a group of cells contained in layer VI of V1—smooth star-shaped nerve cells—which inhibit and modulate the effect of exciter cells. Two observations can be made at this point. The first is that the pathway carrying messages about movement and the pathway carrying a message about color and form are completely separate. The second observation concerns the nature of some cells in V1: they are generally exciter cells but are

regulated by inhibitor cells. The message that is finally sent back to V1 is the result of the balance between these two types of cells.

If the pathways are separate, one may be injured without affecting the other. For example the parvicellular circuit may be damaged but the magnocellular circuit may be intact.

This is probably what occurs in autistic children.

The magnocellular circuit is the first to develop; the receptors or rods at the edge of the retina develop and detect movement. The ganglial cells creating the magnocellular circuit start from there. Autistic children seem to be able to detect movement perfectly well, almost too well. So it is unlikely that this circuit has been damaged. It is more probable that the processing of the image in the parvicellular circuit is affected.

The balance between inhibitor and exciter cells is very delicate. A slight injury to the visual cortex can have serious consequences. For example, if these cells are not perfectly balanced, the information processed in sub-layer IV c β and in layers II and III concerning the form and outline of objects, affects perception. The alteration is transmitted to V3, which then works on faulty input. It may be that V3 receives excessively detailed information about parts of an object, without providing the outline, so that the child is bombarded with information about single parts of objects without perceiving them as wholes. Autistic children with this vision defect recognize people not from their faces but by the shape of their ears or their shoes. They are also able to notice tiny changes in two almost identical figures and to reproduce images effortlessly. They can do jigsaw puzzles at a very astonishing speed and notice a tiny crumb on the floor.

Conversely, the effect of imbalance between inhibitor and exciter cells in the parvicellular circuit, may also send too little information to V3, so shapes are indefinite. Autistic children of this sort need to use special strategies to distinguish shapes.

It is well known that to distinguish the outline of an object motion is helpful. Autistic children are masters of this. Similarly, a child may place things along the edge of a table in order to make the edge clearer or touch things with the tips of the fingers. Another method is to create strong color contrasts. The way children played with saliva in front of the window was a way of creating light contrasts. With hands shade is created that fell over the eyes; the saliva had a prismatic effect, separating colors.

The way children continuously move their hands is a torment for parents. Even if they kept hands relatively still, they continue playing with things around him—a pen, a piece of paper, a leaf, to rock in front of their eyes.

Autistic children have problems perceiving depth. Every time they come close to a carpet they seem to raise their feet as though they were about to climb onto a step A lack of depth perception, i.e. the inability to see how far away things are, creates enormous difficulties. It is like walking in mid-air, without having any idea of how far away the floor or the wall is. It makes stairs difficult to climb and the ceiling appearing to fall. Children with this difficulty often trip over colors and designs.
One of the consequences of this problem is that in large open spaces autistic children often scream in order to receive an echo and calculate the distance to the walls. Or they throw things to see when they impact with the wall.
Our sense of depth is a complex process that requires the combination of information from both eyes. The information is sent from V1 to V5 but it would be mistaken to think that V5 is the area where the information is definitively processed. Depth information has nothing to do with objects in motion. On the contrary, it often requires the use of a distant, stationary object. An injury to the primary visual area means that all information coming from this area may be erroneous, particularly if the sensory input comes from a stationary object. Therefore the correct sense of depth is one of the most common difficulties.

Mothers try to hug her child and the child moves away as if irritated. Over the years many psychologists have investigated this problem and have come up with a number of theories, none of them satisfactory. These theories are usually based on a conflicted relationship between mother and child and have never had any scientific basis. For years, mistaken diagnoses were made on the basis of these theories and treatments often damaged the child and parents more than the illness itself.
This was happening to children and families at the same time that the world's libraries were filling up with books dedicated to the physiology of the nervous system and the processes of responding to sensory stimuli. It would have been sufficient to read practically any one of them to come to a different conclusion.
For any human being the sense of touch is intimately associated with survival. Hence the neurological organization governing the sensory pathway for touch is particularly sophisticated.
This neurological organization helps us to distinguish between light touches and harder contacts, and to feel heat. This is made possible by an inhibitory system for tactile sensations, that filter some out. It is like turning on a radio and looking for a particular station. When you first switch the radio on you cannot distinguish between all the messages arriving; it's just noise. By turning the tuning knob one message emerges stronger than the others and then entirely replaces the previous noise. This is how the neurological organization of tactile inputs works, by choosing one sensation and removing others. Every day we are bombarded with tactile sensation—the touch of clothes against the skin, the feel of a wristwatch, and so on. All of these signals would lead to total confusion if we were not able to "tune in" to one sensation or another. The inhibitory system is exactly this form of tuning in to one signal. We use it to gain tactile information about the world around us.

Tactile information travels along pathways—nerve fibers—that go from the extremities to the center and vice versa. The nerve fibers that carry information about light touches do not carry information about heat or harder contacts; these are carried by other nerve fibers. It is like a three-lane highway where the traffic moves in parallel to the same destination.

In order to enable us to distinguish between one type of tactile sensation and another, groups of receptors on the skin carry out different activities. Some receptors are excited only by very light touches, others only by heat and still others only by deeper contacts.

When we are affected by heat, our heat receptors pick up the information but other receptors are not stimulated since they cannot read this type of input. In other words each type of receptor only picks up signals of a certain type, and is unaffected by other types of signals. We can see that the receptors are specialists, making the tactile sensory pathway extremely efficient.

When the light-touch receptor picks up information it transmits the stimulus along the correct pathway, heat receptors along the heat pathway and so on. The three types of nerve fiber carry only one type of information.

Draw a circle on the palm of your hand then try to stimulate the center of the circle with a paintbrush. In this way you are stimulating only the sensors of light touch. All the other receptors in the circle have been inactive because they cannot read the stimulus. If you then take a pin and prick the center of the circle, the receptors of harsh sensation come into play and the sensors of light touch remain inactive. If you then touch the palm of your hand with an ice cube the receptors of heat will be excited but no others.

The three pathways transmit only one type of information to the cortex, which does not accept the signal as it is, but tends to modify it. The basic function of the cerebral cortex is to receive all inputs from the outside world and to put them together into one message. To do this it inhibits certain inputs, as in the example above.

Inhibition of tactile messages takes place in the bulb and thalamus where exciter and inhibitor neurons are distributed—as in the case of all sensory pathways—with exciter neurons at the center and the inhibitor neurons at the extremities of these brain areas. The part of the input that reaches the center is transmitted to the next nucleus. The part that hits the edge is inhibited. The closer to the edge the impulse is received, the stronger the inhibition. The next nucleus receives only the portion of the input that hits the center. The pathway hands on the message from nucleus to nucleus in this way until the input finally reaches the cerebral cortex.

All sensorial pathways—sight, sound, touch, taste and smell—have inhibitors. In the case of touch there are three types of inhibitors because there are three pathways and three types of input: light touch, deep contact and heat.

If the cerebral cortex controls the inhibition process properly, there is no problem. But if the cerebral cortex has been injured, this control function is impaired and the balance between exciter and inhibitor receptors is thrown askew. Essentially three types of dysfunction result: the nucleus may transmit too much information to the next nucleus by not inhibiting the input at the edge. This nucleus in turn passes on too much

information so it finally reaches the cerebral cortex in too intense a form. This is called hyperactivity. Alternatively the nucleus may inhibit too much of the stimulus at the edge and in the center. In this case the stimulus is overinhibited, leading to hypoactivity.
The third type of dysfunction is called “white noise” and involves the addition of impurities in the input. Inputs are regulated by exciter and inhibitor receptors. White noise occurs when the inhibitor receptors, instead of filtering out an input, actually contribute to it so the message that reaches the cerebral cortex is not a message derived entirely from the outside world but has been polluted along the way by inhibitor receptors.

Autistic children can be diagnosed for hyper or hypo vision, hearing, touch, taste and smell or for white noise in any of these sensory pathways.
For the tactile pathway things are a little more complicated. To say that an autistic child has hyper or hypo tactile activity or white noise in the tactile pathway means nothing at all. If the type of pathway is not specified, the statement makes no sense. It may be that the child has hyperactivity of light touch, and/or hypo activity of deep touch. In other words the diagnosis is meaningful only if related to one or more of the three tactile pathways.
For example, how can you understand which tactile pathway is affected? Why does behavior often seem contradictory? Why does a child not like to be hugged but then go off and bite his own hand?
I managed to reply that close observation of an autistic child shows what the child constantly refuses and what he or she constantly looks for. The analysis of this behavior leads to a correct diagnosis of tactile disorders.

The search for strong stimuli such as beating the knees against the floor or the head against a wall, or biting the hand, would seem to indicate hypo activity of deep tactile feeling. Conversely, the inability to accept a caress or hug, or the touch of clothes on the skin (particularly socks, shoes, caps and glasses), or difficulty taking a shower, brushing one’s teeth, or combing one’s hair, suggest hyperactivity of light touch. Hypoactivity at the deep tactile level combined with hyperactivity at the surface level occurs in 90% of cases of autism; this means the child has areas of the body, such as the soles of the feet, which are hypersensitive but at a deep level virtually insensitive. A child with this disturbance often walks barefoot or on the tips of the toes to reduce contact with the floor. But if he is forced to wear rather rigid shoes or to walk over a cobbled surface he cannot walk on tiptoes or go barefoot so he marches, slamming his feet down onto the surface in order to look for deep tactile feeling.
Hypo tactile children bite their wrists. The way to stop them biting their wrist was to massage it very firmly, replacing the search for deep tactile feeling with another stimulus. But the problem is not just hyposensitivity of the wrist. The pressure of teeth also fails to stimulate sensation, so there is a problem of hypoactivity in the mouth too.

There are other signs of this hypoactivity, children, they can eat almost the entire packet of biscuits in one mouthful. This is common in children who suffer from hypotactile sensitivity inside the mouth. Not only do they bite themselves and things in their grasp, they often eat in this way because it is the only way that they can feel what they are eating. Conversely, a child who finds it difficult to eat even soft foods, or is very sensitive to the temperature of food, or prefers eating very small mouthfuls has the opposite problem: hypersensitivity.

If the choice is based on a tactile experience (soft, crunchy) then the problem is one of feeling; if the choice is based on taste (sweet, sour, acidic, salty) then the problem is based on taste. The stronger the taste, including detergents, soil, stools and other inedible substances, the more likely the child is to be suffering from hypo taste. Hyper taste tends to lead to a choice of almost tasteless food.

But smell also plays a part in the choice of food. A disturbance to the sense of smell also determines other types of behavior, such as the child dirtying himself with excrement or rubbing saliva over his body or over things around him in order to produce an acrid smell that he can recognize.

Hyper smell can cause the child to vomit every time he smells something strong; he may also have trouble urinating or defecating because of the smell.

The control of the sphincter requires a separate discussion. A disturbed sense of smell clearly plays an important role, but other elements come into play. First of all, tactile sensation is important since these activities involve the sense of feeling. Urinating and defecating stimulate a large number of tactile sensations and a child with hyperactive surface feeling may actually feel pain and therefore not want to go to the bathroom. But not only surface feeling is involved. The toilet seat is often colder than the body temperature so a child with hyperactive feeling in relation to temperature may be disturbed. This type of child will have difficulty with potty training and will want to use nappies to maintain the temperature of the genitals.

Another factor contributing to problems urinating and defecating is related to sight. Boys often find it difficult to judge the distance and depth of the toilet and are afraid they might fall in.

Children often can’t reach the floor when they sit on a toilet and this for autistic children can cause a loss of balance that terrifies them. Sometimes it is sufficient to place something under the child’s feet—even a pile of books—to overcome this problem.

A child with hypoactive activity in the area of deep sensation tries to stimulate deep feeling with harsh impacts, such as falling, or by biting or touching parts of the body in contact with mucous, such as the nostrils, ears, eyes and genitals. They are not trying to find sexual pleasure but sensation pure and simple.

Axons (extensions of neurons) are located at the level of joints, muscles and tendons. They communicate the slightest muscle or tendon contraction and the slightest change in the angle of the joint. This enables us to understand where we are in relation to space. In other words, part of our perception of the world is our perception of ourselves within it. The inputs from muscles, tendons and joints travel along neural pathways

that are adjacent to those of deep tactile sensation, so an injury impairing one pathway may also produce a dysfunction in the adjacent pathway.

Hypo tactile children have an imprecise notion of themselves in the deep feeling pathway. If you ask them to touch their nose they may touch their ears. Body contortions are quite common. An imprecise notion of one's body means it is difficult for the autistic child to draw the human body. Autistic children generally draw the human body with massive distortions between the body and legs or the hands and head. The size of the hands or head in the drawing often corresponds to the perception the child has of that part of his body. All tests related to the interpretation of drawings should bear this in mind; otherwise the results would have no scientific foundation.

Emotions are influenced by environmental and cultural factors.

A man raised in a mountain district finds the peaks of mountains more breathtaking than the view of the sea. A sailor has a different relation to the sea than others, who have not been to sea. Their emotions are influenced by their experience. Education also has a bearing on feelings. An opera lover does not respond to jazz in the same way that a jazz fan does. The environment, culture, and education are all strong factors in our everyday emotions. But they are nothing if the perception of the outside world is incorrect.

How would we experience a sunset if we suddenly went color-blind? Our sense of the importance of color would change. Our relationship to beauty would no longer be the same. And if we had never perceived colors huge parts of popular culture related to sunsets would be lost on us.

What if we had no sense of smell? Any defect in sensory perception changes emotional capability.

But how is this possible? Aren't emotions intimate and unique, the product of the individual mind? How can they just be the result of perception?"

Maybe the thought is not comforting, but the fact remains that they are the result of perception. Human beings are culturally inclined to think of emotions as inscrutable and private. Religion, some philosophical thought, and some psychology tend to encourage this view. But it is based on erroneous thinking. Human emotions are the product of culture, tradition and the environment because they are transformed into sensory inputs; they are perceptions. A faulty sensory mechanism precludes the proper development of emotions. This might seem rather cold, but neuroscience has proven this conclusion, and other religious and psychosocial theories—which we might feel drawn to for a number of reasons—have proven to have no scientific basis..

If the sensory mechanisms could be retrained and corrected, behavior would improve and the emotions would evolve.

Nothing is worse for a parent than to be told that nothing can be done, that it would be better to put their energies into their other children or, worse, to seek psychiatric help.

Making Sense of the Senses based on extract from publication “Children who do not look you in the eye, The Secrets of Autistic Behaviour”
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With kind permission of the authors.

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The Autism Centre
May 2006