The effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health

Schore, A. N. (2001) The effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal*, **22**, 7-66; *and:* Schore, A. N. (2001) The effects of early relational trauma on right brain development, affect regulation, and infant mental health *Infant Mental Health Journal*, **22**, 201-269.

In these two articles Allan Schore outlines his integration of work in attachment, neuroscience, the psychophysiology of stress and psychiatry to form a psychoneurobiological developmental model of early attachment experience and the developing brain. The articles themselves are lengthy, highly detailed and heavy reading (unless you are highly versed in neuroscience) but well worth the effort. They provide a highly detailed picture of the neurological processes that appear to underlie the attachment mechanisms presented as models for regulation of affect and working models of self and other.

The following summary integrates material from both articles.

Summary

There is a human brain growth spurt that occurs between the third trimester in-utero to about 18-24 months of age. This involves the development of the right limbic brain (limbic system and cortical association areas) which is involved in social cognition, attachment and caregiving behaviours, as well as and regulating bodily and affective states. This system is in growth spurt for the first 18 months of life and remains dominant for a further 18 months.

This brain growth occurs through a process of `ontogenetic plasticity` in which there is an overproduction of synaptic connections, many of which are discarded through a process of competitive interaction that selects only those connections that best match incoming environmental information. Through this means, right brain structural and functional development is adaptive to environmental experiences. The developing right brain limbic system of the child and the established systems of the adult are coupled through affective synchrony to the extent that they effectively form a single biological entity where the caregiver influences the infant`s neurobiological development. This is illustrated by the sympathetic cardiac acceleration and parasympathetic deceleration that occurs in both mother and infant during episodes of play.

Infant brain growth is also sequential with the later developing cortical structures adaptively regulating the earlier appearing subcortical systems and effectively reorganising the control mechanisms. A three level model is proposed in which increased interconnectivity allows information from lower circuits to be processed at higher levels, allowing the later developing systems to operate in an executive capacity, although early and late systems all remain coupled and influential. This development represents a move from initial reflexive operations to greater top-down control from the cortex and the consequent appearance of more subtle and complex control systems for regulating affect across this developmental period.

Some systems highlighted in the paper include the following:

- At 8 weeks, rapid metabolic changes occur in the primary visual cortex and occipital context, which are influenced by visual experience. At this time, the most potent environmental stimulus is the mother`s face. Early face-to-face emotional interaction rapidly develops. The secure mother`s instinctive response to this is to follow the infant`s sequencing by sensitively attuning to the infant`s desire to engage in affective face-to-face communication or to disengage. Visual input of mother`s face is associated with elevated levels of neurotrophins (brain nourishing chemicals) which are synaptogenetic (help produce synaptic connections) and are known to perform a growth-promoting role in the postnatal development of the cortex.

- The limbic system, which is involved in emotional regulation, is in critical growth during the early postnatal period and it`s growth and organisation is influenced by the interactions between infants and caregivers. Across this period, caregivers serve to amplify the infant`s positive affective states by engaging in interactive play and also serve to calm negative affective states by dealing with environmental stressors and by emotionally engaging with the infant. Through ontogenetic plasticity, the interactions with a sensitive mother that assist in regulating the infant`s arousal levels also promote the development of the infant`s own brain

structures for regulating affect. Both positive and negative affective experiences are relevant as transitions from positive to negative and then back to positive affect teach the child that negative affect can be endured and overcome.

● The last developing structure in the orbital prefrontal region or `senior executive` of the socio-emotional brain. This is dominant for non-conscious processing and expressing of emotional information and for regulating internal physiological state via the hypothalamo-pituitary adrenocortical and the sympathetic-adrenomedullary axes according to internal and external feedback.

In terms of differential effects of caregiving, Schore suggests that secure attachments promote brain growth and development, resulting in the development of more complex internal organisation for regulation of affect and giving greater flexibility in response to emotional stimuli. In contrast, abusive caregivers induce traumatic states that negatively impact on neurological development through induced hyperarousal as well as by failing to engage in affective communication and play that would positively influence the experience dependent maturation of the infant`s brain.

Prior to birth, high levels of maternal stress hormones regulate gene expression in the foetus affecting brain development. Furthermore, it is well established that stimuli such as maternal alcohol consumption and smoking affect foetal brain growth and both of these activities may be associated with parental avoidance or rejection.

Mutually escalating arousal levels resulting from traumatic caregiver-infant interaction initial result in hyperarousal of the sympathetic nervous system (SNS). This can progress to a disassociated state described by Perry et. al. (1995) as `fear-terror` in which the parasympathetic system (PNS) becomes over-activated and the individual passively disengages. In this state, the infant is effective following a survival strategy of conservation-withdrawal, the equivalent to feigning death due to apparent hopelessness and resource depletions. In both of these states, which remain long lasting without maternal re-regulation, both the SNS and PNS are hyperactivated and developing limbic connections are damaged by exposure to excitotoxic neurotransmitters such as glutamate which selectively induce neuronal cell death. This can result in permanent functional impairment of limbic circuitry.

● During the first 9 months, such traumas affect the development of the rapidly growing amygdala and early limbic systems. This pruning of developing neuronal pathways would result in poor regulation of subcortical systems and suggest poor autonomic regulation. This would reduce capacity to experience positive emotional states and reduce executive affective regulation. This is supported by neurobiological studies indicating such damage affects later ability to form social bonds and emotional functioning.

● From 9 to around 18 months, the long dendridic connections that facilitate interconnectivity of cortical and subcortical brain areas tend to become pruned leading to `parcellation` or poor integration between different hierarchical layers of the corticolimbic system. This would reduce capacity to executively inhibit hyperaroused states such as terror.

Such early adaptive dysfunctions would be long lasting and would most obiouvlsy manifest under later conditions that were stressful and called for flexible responses to challenging conditions. Schore suggests that organised but insecure attachment may be the result of one or the other of the above forms of developmental deficit while disorganised attachment would be characteristic of both. This is equated with observations of `disorganised` infant behaviour (Main & Solomon,1986) such as low stress tolerance, brief periods of disorganisation and approach-avoidance behaviours.

In short, these two articles offer a detailed and convincing neurobiological explanation of likely mechanisms underlying the development of individual differences in attachment during infancy. They particularly support Bowlby`s control systems approach and a regulation of affect conceptualisation of attachment. They also seem to indicate the presence definate traits, programmed by early experience, that may be at the core of attachment processes and behaviour.