

Dissociation: a developmental psychoneurobiological perspective

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Abstract

Dissociation can be defined as the failure to integrate experience. Dissociation is a common symptom of a spectrum of severe psychopathologies, from reactive attachment disorder of infants to dissociative identity disorders, psychotic experiences, borderline personality disorders and post-traumatic stress disorders of adults. The incidence of abuse in the childhood histories of adults with dissociative disorders is extremely high. The adaptational value of dissociation is that it allows survival in catastrophic events. The disadvantage is that when dissociation occurs frequently, the development of neural networks is impaired. Especially disadvantageous are problems which develop with a) higher circuit control over lower circuits, b) primitive parasympathetic regulation by the dorsal motor nucleus, c) memory, d) left hemisphere, e) integration of bodily sensations, f) sense of self, and g) affect and motivation. These aspects are discussed in more detail. The clinician's primary function is as an affect regulator and therapy should focus on integration.

Keywords: Dissociation, Infants, Psychopathology, Developmental

This paper on the developmental psychoneurobiology of dissociation is largely based on the work of Professor AN Schore.^{1,2}

Dissociation is an absence of the normal integration of thoughts, emotions, and experiences into the stream of consciousness and memory.¹ It is a frequent symptom of a range of severe psychopathologies, from reactive attachment disorder of infants to dissociative identity disorders, psychotic experiences, borderline personality disorders and post-traumatic stress disorders of adults.² A central tenet of a developmental psychoneurobiological perspective is that a continuity exists between early traumatic attachment and later severe disorders of personality development.¹ The incidence of abuse in the childhood histories of adults with dissociative disorder is extremely high, coming close to 100%.³

Etiology: early trauma without support

Attachment research has shown that the interactive regulation embedded in the early relationship between an infant and its primary caregiver is of prime importance in the experience-dependent maturation of the infant's brain.² The first two years of life are a critical period for the maturation of the right hemisphere and the limbic system, and this maturation is exquisitely sensitive to environmental influences, as transmitted to the infant through its relationship with its primary caregiver.² This explains why relational trauma such as abuse or neglect during the first two years of life has later permanent effects

on personality organisation, and is more detrimental than during later developmental stages.² The impact of attachment trauma on the developing brain explains why dissociation that begins early in life becomes characterological, while dissociation with a later onset, e.g. in adolescence, does not.²

Dissociation, when it first occurs, results from a 'psychological shock'.² When a traumatic event is experienced, and the caregiver can provide a sense of protection, the child will not experience 'fright without solution'.³ Even when the caregiver is the source of the trauma, the caregiver can help the infant to cope by a process called 'interactive repair', for example by soothing the infant after having scolded it. However, instead of modulating, some caregivers induce extreme levels of stimulation and arousal, very high in abuse, and very low in neglect.¹ In addition to dysregulating the infant, these caregivers withdraw any interactive repair, thus leaving the infant for long time spans in an extremely disturbed psychobiological state that is beyond its undeveloped coping mechanisms.¹ The child's efforts to enlist the caregiver's help, e.g., by crying, or showing fear, are often met with further abuse.¹ These behaviours must thus be inhibited, and so, to survive, the infant must resort to an autoregulatory tactic to deal with overwhelming levels of anguish.¹ Such children thus experience 'fright without solution', and the only way to cope may be by disassociation of explicit from implicit processing to achieve an overall integrated state of mind (i.e. dissociation).⁴

If the developing infant is repeatedly exposed to the cumulative trauma that derives from an interactive dysregulating environment with a misattuning caregiver, the attachment children develop to such caregivers is disorganized.² Because this growth-inhibiting context generates a severe and long-lasting degree of negative affect in the infant, it rigorously limits its expressions of an attachment need for self-protective purposes.² The infant thus

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shifts from interactive regulatory modes into long-enduring, less complex autoregulatory modes.² These subcortical-limbic organizational patterns are primitive strategies for survival.²

Advantage: Dissociation is adaptive in the short-term

Dissociation can be defined as the failure to integrate experience with the adaptive function that it allows a person not to become overwhelmed in the face of trauma.³ When a person experiences a threat, an immediate response is alarm, which is accompanied by hyperarousal of the sympathetic nervous system.³ If the threat continues for a long time, or the person is largely helpless, continued high arousal cannot be sustained, and the person may enter a dissociative state, which is characterised by freezing and a trance-like state.³ The switch in state from sympathetic hyperaroused-terror into additional activation of parasympathetic hypoaroused conservation-withdrawal hopelessness and helplessness, follows the subjective evaluation (perception) of the imminent threat as one that cannot be evaded or modified.² When the infant perceives that it is fatally helpless in the face of brutal danger, it yields to it.²

The abused infant's abrupt state switch from sympathetic hyperarousal into parasympathetic dissociation is the sudden shift from an ineffective strategy of struggling requiring massive sympathetic activation to the metabolically conservative immobilized state mimicking death, which is associated with the dorsal vagal complex.² The activity of the dorsal vagal complex in the brain stem medulla decreases blood pressure, metabolic activity and heart rate, despite increases in circulating adrenaline.² This elevated parasympathetic arousal allows the infant to maintain homeostasis in spite of sympathetic hyperarousal.² The inhibitory vagal brake in such cases is mainly provided by the rigid vegetative dorsal motor vagus, and not the more evolved 'smart' nucleus ambiguus that is necessary for social communication.² The vagal brake must be withdrawn when the individual shifts from a state of low to high metabolic demand, as would occur in interaction with the dynamically changing environment.² The vagal brake makes any involvement in dyadic play states, with the creation of high levels of arousal and metabolic energy for brain biosynthesis, impossible.² Dissociation is a primitive defense, and in early traumatized developmental psychopathologies more complex defenses never arise.²

Disadvantage: Dissociation is maladaptive in the long-term

Dissociation begins as a protective mechanism to maintain the integrity of the self in the face of catastrophic trauma, but can become a threat to optimal functioning if it becomes a routine response to stress of less than catastrophic magnitude.⁵ The cost of experiencing dissociative states frequently as a child is a sensitised neural network, with less and less necessary to evoke subsequent dissociative states.³ Also, individuals who dissociate often, have problems in exiting the state of conservation-withdrawal.² Once dissociated they stay in this massive autoregulatory mode for long periods of time.² During these times they are inaccessible to the external environment, and thus resistant to attachment communications and interactive regulation.² If this happens regularly, the avoidance of emotional contexts prevents emotional learning.² The pathological walling off or dissociation from stress and pain has devastating effects on self, and therefore psychobiological functions, which are discussed in more detail in the next section.

Psychobiological pathology: Dissociation is a dis-association

Basic research in affective neuroscience demonstrated that emotional and social deprivation precludes the normal development of cortical and subcortical limbic areas, and leads to 'neurological scars' that are the cause of ensuing behavioural and cognitive insufficiencies.¹ The neural abnormalities are discussed in more detail below, referring specifically to the impaired development of higher circuit control over lower circuits, primitive parasympathetic regulation by the dorsal motor nucleus, memory, left hemisphere, integration of bodily sensations, sense of self, and affect and motivation.

Development of higher circuit control over lower circuits is impaired

An efficient mature orbitofrontal system can adaptively regulate both sympathoadrenomedullary catecholamine and corticosteroid levels, and therefore hyper- and hypoarousal.² It can also facilitate or inhibit the defense reaction of the amygdala.² However, an efficient mature orbitofrontal cortex never develops in those individuals who had unfavourable childhood experiences without support.² In trauma, sympathetic hyperarousal is followed abruptly by hyperparasympathetic dissociation.² Relational trauma induces high levels of cortisol in the infant's developing brain.² These dysregulating environmental experiences trigger intense shifts of ergotropic and trophotropic arousal that lead to chaotic biochemical variations in the infant brain, which in turn cause extensive oxidative stress and apoptotic damage of synaptic connections within the dual limbic circuits (one excitatory and one inhibitory).² Since the orbitofrontal areas are connected to the dual limbic circuits and both branches of the autonomic system, a widespread developmental parcellation or thinning of these connections would lead to an ineffective regulation of the autonomic nervous system (ANS) by higher centers in the central nervous system (CNS).² This deficit means that under stress there would not be a counterbalancing system between the sympathetic-excitatory and parasympathetic-inhibitory components of the ANS, i.e., a loss of a coupled reciprocal mode of autonomic control.² An ensuing fast uncoupling of both frontolimbic circuits would occur even following low levels of interactive stress, characterised by emotional lability and state shifts.² This organisation of autonomic control prevents the integration of lower more primitive autonomic states that permits the development of new higher states.² But stress may also take the prefrontal areas off-line, allowing the more usual responses mediated by the subcortical structures to control behaviour.² This occurs all too often in a severely developmentally compromised immature frontolimbic system.²

Primitive parasympathetic regulation by dorsal motor nucleus

The experience-dependent maturation of orbitofrontal areas that regulate the parasympathetic system, a development that is slower and later than the sympathetic system, is of particular importance.² The orbitofrontal areas, like the amygdala, have direct inputs into the medulla, including the medullary reticular formation and medullary noradrenergic neurons in the nucleus of the solitary tract.² These are the locations of the medullary vagal system, but it is now known that there are two parasympathetic vagal systems, a late developing "mammalian" or "smart" system in the nucleus ambiguus which is responsible for communication via facial expressions, vocalizations, and gestures, and a more primitive early

developing 'reptilian' or 'vegetative' system in the dorsal motor nucleus of the vagus that causes the shutdown of metabolic activity during immobilisation, death feigning and hiding behaviours.² The central nucleus of the amygdala has extensive connections into the dorsal motor vagal nucleus and is involved in passive coping, immobile behaviour and parasympathetic activity.² Both of these vagal systems are right lateralised.² As stated earlier, the inhibitory vagal brake in such disorganized systems is predominantly provided by the rigid, fixed vegetative dorsal motor vagus, and not the more evolved and flexible 'smart' nucleus ambiguus that allows for social communication.²

Memory

Implicit memory of traumatic events is encoded without explicit processing due to divided attention, amygdalar discharge and release of stress hormones.⁴ The extent to which the individual is detached, interferes with the process of elaborative encoding.⁶ Fluctuations in the level of detachment during trauma may be one factor that contributes to the fragmentary quality of traumatic memories.⁶ The absence of a narrative version of events that occurred thus leads to an inability to integrate, and the memories remain in an unstable state of implicit activation.⁴

Left hemisphere

Early growth-inhibiting social environments without emotional support, lead to an inefficient right brain vertical cortical-subcortical system, with poor right-to-left orbitofrontal communication.¹ This results in alexithymia, 'no words for feelings'.¹ Early emotional learning of the right hemisphere, especially of stressful episodes, can thus be unknown to the left hemisphere. Sensitisation of the opioid response may contribute to the dreamlike state of detachment (cortical disconnectivity hypothesis: impaired functioning of the left frontal cortex, thus 'speechless terror').⁶ Dissociated experience thus tends to remain unnamed by thought and language, and is numbed to full participation in the life of the rest of the personality.² Such pathological representations (of a dysregulated-self-in-interaction-with-a-misattuning-other) are accessed when the individual is stressed, as would occur specifically in attachment related contexts.²

Bodily sensations

It is important to emphasise that in traumatic abuse the individual dissociates not only from the external world, but also from painful stimuli originating within the body.² All pain is stilled and a comforting numbness ensues, due to a sudden massive elevation of endogenous opioids.² An inefficient orbitofrontal-cingulate higher limbic circuit would be unable to regulate pain, and a lower amygdala limbic level driven dissociation would take over.² Cutting, a common form of self-destructive behaviour, may be an effort to autoregulate out of the distorted pain sensitivity linked with the elevated opioid activity of the dissociative state.²

Sense of self

The excessive unregulated dissociation that results from early relational traumatic attachments is the major mechanism that creates what Balint called the 'basic fault', a deep and all-encompassing sense that there exists within a fault that extends widely to include the whole psychobiological structure of the individual, and that is experienced as a feeling of emptiness, being lost, deadness, and futility.^{1,7}

Affect and motivation

A failure of orbitofrontal function is seen in the hypometabolic state of dissociation. This dysfunction interferes with the orbitofrontal role in processing motivational information, and therefore manifests as a deficit in organizing the expression of a regulated emotional response.¹ The active coping strategies of Lichtenberg's attachment-affiliation, exploratory-assertive, aversive, and sensual-sexual motivational systems all disintegrate in subcortically programmed survival states of passive disengagement, conservation-withdrawal, energy depletion and dissociation, the escape where there is no escape, the last-resort defensive strategy.^{1,8}

Conclusion

Dissociation can be defined as the failure to integrate experience. The adaptational value is that it allows survival in catastrophic events. The disadvantage is that when it occurs frequently in childhood, as for example when an infant is abused by its primary caregiver, the development of neural networks is impaired. Especially disadvantageous is the lack of development of higher cortical control over subcortical structures, the sensitisation to dissociate even with minor stressors, the prevention of further emotional learning, desomatization and impaired memory of traumatic events coupled with deficient left hemispheric linguistic processing of adverse experiences. The result is a progressive impairment of the ability to adjust, take defensive action, or act on one's own behalf, as well as a blocking of the capacity to register affect and pain.² To the extent that dissociation prevails, fragmentation of the self occurs, because integration is not a function of the self, it is the self.⁵

This clearly implies that therapy should focus on integration, on accessing traumatic implicit memories, and bringing them into awareness in order for integration to become possible.¹ The emotional relationships in the process of psychotherapy should cover the deficiency caused by the lack of emotional relations in early childhood.¹ The clinician's primary function is as an affect regulator for the patient's primitive, traumatic states, including those affective states that are walled off by dissociation.¹

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Commentary

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The article by Panzer and Viljoen offers a cogent overview of recent models of the etiology of pathological dissociation. This interdisciplinary information is drawn from the fields of developmental psychology, developmental neuroscience, and infant psychiatry. Developmental information is now being rapidly absorbed into child psychiatry and neuropsychiatry, and updated attachment theory is a central source.¹ In this integration, recent data on the mechanisms that underlie the attachment bond of emotional communications between the mother and infant have been integrated with current studies on the developmental neurobiology of attachment. Thus, it is now thought that attachment relationships are formative because they facilitate the development of the brain's major self-regulatory mechanisms.^{2,3} Furthermore, the field of developmental psychopathology, an outgrowth of attachment theory, is now generating complex models of both normal and abnormal development, information that is directly relevant to clinical psychiatry.

This interdisciplinary perspective is especially valuable to a deeper understanding of trauma, a phenomenon that impacts both mind and body, and thereby severely impairs both psychological and biological functions. Although Freud⁴ struggled with the concept of trauma over the course of his career, in his last work he asserted that trauma in early life is especially psychopathogenic, and that it effects all vulnerable humans because "the ego...is feeble, immature and incapable of resistance." This is so because early trauma negatively impacts the infant's developing brain. Recent researchers of "maltreatment-related (pediatric) posttraumatic stress disorder" conclude that severe trauma of interpersonal origin can override any genetic, constitutional, social, or psychological resilience factor, that specifically a dysfunctional and traumatized early relationship is the stressor that leads to posttraumatic stress disorder, and that the overwhelming stress of maltreatment in early childhood is associated with adverse influences on brain development.⁵

Trauma in the first two years is typically not a single incident, but ambient and cumulative, and for this reason it is best characterized as "relational trauma".⁶ In a number of contributions I have offered interdisciplinary evidence which suggests that severe relational trauma, especially neglect and/or abuse, alters the development of specifically the right

brain, the biological substrate of the human unconscious.⁷ This hemisphere is in a growth spurt in the first 2 years of life and dominant for the first 3. It is now well established that prolonged and frequent episodes of intense and unregulated interactive stress in infants and toddlers have devastating effects on not only the development of stable and trusting attachment relationships but also on the establishment of psychophysiological regulation. Indeed, the right hemisphere specializes in the unconscious processing of social and emotional information, the regulation of bodily states, and attachment functions.^{1,2}

In my work I suggest that attachment trauma embedded in a growth-inhibiting interpersonal environment induces a developmental failure of the experience-dependent maturation of the right-lateralized emotional brain. A developmental impairment of this system is expressed as a severe limitation of the essential activity of this hemisphere - the control of vital functions supporting survival and enabling the organism to cope actively and passively with emotional stress. This structural limitation of the right brain, the hemisphere dominant for the human stress response, is responsible for the individual's inability to regulate affect, which is at the core of trauma psychopathology. Furthermore, early relational trauma and pathological attachment histories are stored in not the explicit-declarative but the implicit-procedural memory system. These early imprints endure in right hemispheric autobiographical memory. In the child psychiatry literature Gaensbauer concludes, "The clinical data, reinforced by research findings, indicate that preverbal children, even in the first year of life, can establish and retain some form of internal representation of a traumatic event over significant periods of time".⁸

But early relational trauma impacts more than the memory systems. Because it severely disorganizes the developing brain, it also induces neuropsychological cognitive-emotional vulnerabilities, enduring deficits that negatively affect the child's ability to integrate the traumatic experience, and thereby interfere with the emergence of a resilience mechanism that can cope with later environments of relational stress. Bowlby⁹ postulated that the major negative impact of early traumatic attachments is an alteration of the individual's normal developmental trajectory, and Krystal¹⁰ asserted that the long-term effect of infantile psychic trauma is the arrest of emotional development. Recent psychoneurobiological models suggest that this arrest is specifically in the right brain, and is manifested in a number of early-forming attachment pathologies, severe personality disorders that are frequently accompanied by psychosomatic symptomatology.⁷

There is now a great deal of interest amongst clinicians in

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intense, traumatic affects, such as terror and rage. But in recent work I have suggested that we must also deepen our understanding of the early etiology of the primitive survival defense that is used to cope with traumatic, overwhelming affective states – dissociation.¹¹ Nijenhuis¹² is now describing not just psychological (e.g., amnesia) but “somatoform dissociation,” which is associated with early onset traumatization, often involving physical abuse and threat to life by another person. Somatoform dissociation is expressed as a lack of integration of sensorimotor experiences, reactions, and functions of the individual and his/her self-representation. Clinical studies show that dissociation is a suppression of autonomic physiological responses, especially when recalling traumatic memories. A recent fMRI study demonstrates that while exposed to traumatic material PTSD patients in a dissociative state show no increase in heart rate and an altered corticolimbic pattern lateralized to specifically the right hemisphere. In commenting on the right lateralization of dissociation the authors speculate upon “the possibility that childhood trauma sets the stage for lateralized responses”.¹³

Trauma authors are now asserting that if early trauma is experienced as “psychic catastrophe”¹⁴, the survival mechanism of dissociation represents “detachment from an unbearable situation”¹⁵, “the escape when there is no escape.”¹⁶ Dissociation, the last resort defensive strategy, may represent the greatest counterforce to effective psychotherapeutic treatment of personality disorders. Panzer and Viljoen appropriately conclude their incisive article with thoughts about the application of this developmental psychoneurobiological knowledge to the psychotherapy of severe psychopathologies. Their contribution has direct implications for not only intervention but also for models of early prevention.

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