

Prenatal Aspects in Alzheimer's Disease

**Rien Verdult, Master's in Psychology, Private Practice
(Belgium)**

ABSTRACT: Alzheimer's disease (AD) is a degenerative brain disorder characterized by a global mental deterioration. Although the etiology is not yet clear, more evidence shows that a prenatal link is possible. Memory disturbances are central in AD and eventually lead to a loss of autonomy and identity. Anxiety becomes the basic feeling of AD patients, as well as experiences of mourning, loss of control, and loss of contact. In the manifest stage retrogenesis is triggered, that is, patients reverse develop and start to re-live their past. In emotional retrogenesis prenatal and perinatal themes can be re-experienced. In prenatal emotion-oriented care sensorimotor relaxation ('snoezelen') is being used to reduce anxiety. The patient is given an environment that reflects the characteristics of a womb, and the nursing staff's approach should be to symbolize the 'good-enough' mother.

KEY WORDS: Alzheimer's disease, etiology, retrogenesis, prenatal themes, emotion-oriented care

INTRODUCTION

Dementia is a clinical syndrome characterized by a global deterioration of mental functioning, afflicting daily life seriously. Alzheimer's disease (AD) is the most common cause of dementia. In about 60% of patients Alzheimer's is the main cause of the mental decline. As people increasingly get older, more of them develop Alzheimer's disease. Prevalence rates go up sharply with age, doubling about every 5 years, at least until the age of 85, when the rise begins to slow. It is estimated that 2-5% of people over 65 years of age and up to 20-25% of those over 85 years endure the disease. Worldwide more than 25 million people suffered various forms of dementia in 2000. Some speak of the Alzheimer epidemic. By 2030 it is expected that there will be 63 million patients, and 65% of these will reside in less developed countries (Swedish Council, 2008).

Rien Verdult, a developmental psychologist and psychotherapist, has worked with Alzheimer patients for 20 years. He also has a private practice as a prenatal psychotherapist for adults and babies. Direct correspondence to: Opleeuwstraat 59, B 3840 Gors-Opleeuw, Belgium. rien.verdult@skynet.be

In 1995 1.9 million Americans 64 years or older had AD. By the year 2015, 2.9 million Americans will have this disease, and of these, more than 1.7 million would need active assistance in personal care. In my country of Belgium (10 million inhabitants) 9.3% of people over 65 years of age suffer from AD. The number of AD patients is expected to rise from 160,000 in the year 2001 to 251,000 in the year 2030.

Alzheimer's disease is a progressive, degenerative and irreversible brain disorder that causes intellectual impairment, disorientation and eventually death. AD has a gradual onset and progressive decline. A German doctor, Alois Alzheimer, described the disease in 1905. The patient he detailed was Johann Feigl, a man in his early fifties and who had severe memory problems, challenges in performing everyday tasks, and language difficulties. He was also disoriented as to time and space. And like Mr. Feigl, most Alzheimer patients have problems with abstract thinking, loss of initiative and decreased judgment. There are changes in their personalities, as well as their moods and behaviors.

There are other types of dementia, like dementia with Lewy bodies, which gets its name from tiny structures that develop inside nerve cells, and which trigger the degeneration of brain tissue. Another common cause of dementia, which is responsible for about 20% of the dementias is a vascular disease by which the blood supply to the brain is diminished. Other causes of dementia include: Creutzfeld-Jacob disease, Binswanger's disease, the frontal lobe dementia or Korsakoff's syndrome. This article is limited to Alzheimer' disease (AD) because this group includes the majority of patients and the other diseases lead to minor variations in their experiential world.

At this moment no cure is available for Alzheimer's. A variety of drug treatments claim to show beneficial effects for patients, such as, cholinesterase inhibitors. However, most drugs have minor and short-term positive effects in the early stages of Alzheimer's disease, and do not cure the patient. A definitive cure for this degenerative disease is not to be expected within the next two decades.

LITERATURE REVIEW

Etiology

The causes of Alzheimer's are not yet fully understood. The etiology is a complex interaction of many genetic, environmental and life-style risk factors, with age and genetics most probably playing the largest part. The hallmark of AD is the extracellular accumulation and deposition of insoluble amyloid protein, to be found in the form of

amyloid plaques. Equally conspicuous is the intraneuronal occurrence of neurofibrillary tangles, consisting mainly of tau-protein. These amyloid plaques and neurofibrillary tangles are characteristic, but not specific to AD. Similar changes can be found in healthy aging processes and in various other neurodegenerative diseases.

Although there seems to be a large heterogeneity in the causes of AD, the amyloid-cascade-hypothesis has taken a central position as a model for the general etiology. This hypothesis was first formulated in 1992 and points to the cytotoxicity of mature aggregated amyloid fibrils, which are believed to be the toxic form of the protein responsible for disrupting the cell's calcium ion homeostasis and thus inducing apoptosis (Hardy & Selkoe, 2002). The amyloid plaques and tangles are considered not to be the key symptoms of AD but rather the cause of the disease. The regulation of amyloid plaques underlies a diversity of cellular and molecular factors. Disturbances in the cerebral glucose metabolism, especially in the hippocampal regions, are a further factor in the pathogenesis of AD. The widespread loss of cortical cholinergic neurotransmission associated with the cognitive deficits is another important factor. The question remains unanswered whether these factors are causing AD or are correlated with AD.

Biomedical and genetic research on AD is dominant in the field. This implies that psychological factors are infrequently included in the etiological research on AD, although it can be speculated that prenatal stress and/or depression can play a crucial role in triggering biochemical brain disturbances. In 1985 I myself did a small-scale research on the influence of early loss experiences in the etiology of AD. When elderly people, born between 1910-1920, had lost one or both of their parents before the age of 10 years, they had a significantly higher risk of becoming demented (Verdult & Miesen, 1986). Although this research had many methodological limitations there was a strong indication that loss experiences could be a risk factor in AD. Early loss experiences can lead to depression in later life. A possible confirmation on the role of loss experiences in AD could be found in the accumulating evidence that suggests that a history of depression constitutes a significant risk factor for developing AD. A Dutch epidemiologic research effort showed that people with a history of depression, particularly with early onset before the age of 60 years, have a three times higher risk of having AD, compared to people who have never been depressed (Geerlings, den Heijer, Koudstaal & Breteler, 2008). Depression can be seen as a major risk factor in AD (Lu, Edland, Teng, Petersen & Cummings, 2009).

Greater activity of the hypothalamic-pituitary-adrenal (HPA) axis

is associated with specific neurological and psychiatric disorders, including Alzheimer's disease and depression. Hyperactivation of paraventricular corticotrophin-releasing hormone (CRH) neurons may form the basis of this increased activity of the HPA axis. This hyperactivation contributes to the etiology of these disorders (Raadsheer, Van Heerikhuize, Lucassen, Hoogdijk, Tilders & Swaab, 1995). Exposure to excessive prenatal stress can alter the level of activity of the HPA axis, and this prenatal setting of stress levels can be consistent into adult life. Research shows that prenatal maternal stress has its effects on the stress of the fetus and newborn (Robles de Medina, 2004), and on the later adult (Nathaliensz, 1999). The HPA axis is prenatally programmed. From this reasoning it can be hypothesized that prenatal stress can be linked to the hyperactivation of CRH-releasing neurons in Alzheimer's disease.

Recently, a link was discovered between prenatal developmental processes and the deterioration processes in Alzheimer's. Researchers at a U.S. biotechnology company, Genentech Inc., proposed a link between the reactivation in AD to that of a process in fetal development (Nikolaev, Mclaughin, O'Leary & Tessier-Lavigne, 2009). This research group suggested that a normal process in which excess nerve cells and nerve fibers are pruned from the developing brain during prenatal life is somehow reactivated in the adult brain and hijacked to cause the death of such cells in AD. In the prenatal brain twice as many neurons as we need are developed. The process of apoptosis clears out unneeded cells. Unneeded cells are neurons that have not succeeded in making new connections. Only neurons that wire together can survive. This prenatal pruning process may be triggered in Alzheimer's. It is not the beta amyloid precursor protein (APP) that kills nerve cells in AD; it is what is involved in the prenatal pruning. The prenatal pruning process is sparked by another fragment of the amyloid precursor protein, namely N-APP, causing a cascade of events that results in the death of unneeded neurons. This prenatal link implies that getting Alzheimer's disease is not just bad luck, but rather is the activation of a pathway that is there for developmental reasons. If this hypothesis could be validated, it can be an important step in understanding the psychopathology of AD. Research on what triggers the reappearance in the adult brain of a process fundamental to its early prenatal development can open a variety of curing possibilities.

Memory Disturbances in Alzheimer's Disease

Cognitive impairment in AD contains memory disorders and higher cortical dysfunctions like aphasia, apraxia and agnosia. The loss of memory is the key symptom of Alzheimer's disease, often appearing first in the deterioration process. It is important to stress that cognitive impairment does not immediately cause a total loss of all cognitive skills. A pattern unfolds in which some cognitive abilities are relatively well preserved and some are seriously impaired. In AD the multimodal association cortex is impaired (including the hippocampus complex in the medial temporal cortex, and the association fields in the dorsolateral and orbitofrontal cortex and the temporal and parietal cortex). Functional disorders in these areas cause impairment of memory for recent events, and in the long run impairment of the long-term memory, purposeful action, language expression and understanding, and three-dimensional perception (Diesfeldt, 2003). Alzheimer's does not seem to have a noticeable impact on some brain functions. Functions of the primary motor and somatosensory cortex, of the primary sensory cortex (auditory and visual), the cerebellum and the basal ganglia remain intact for a relatively long period of time. This distribution of selective loss and preservation is reflected in a specific pattern of lost and preserved memory skills.

In the prevailing models of the architecture of the human memory a distinction is made between short-term memory or working memory on the one hand, and long-term memory on the other hand. In the long-term memory a distinction is made between the procedural, implicit or unconscious processes and the declarative or explicit processes. In the latter, episodic and semantic memory functions can be described. For the purpose of this contribution I shall focus on the long-term memory and more specific the episodic aspects of the memory system. According to Tulving, an authoritative expert in the field of memory research, episodic memories are impossible without semantic memory. There is a strong interdependency between the two processes, meaning that episodic memories depend on prior semantic processes (Tulving & Markovitsch, 1998). All of our personally acquired knowledge, as stored in our semantic memory, is based on episodic experiences. In everyday life we do not have to make an effort to notice that meaningful experiences are almost automatically recorded in the episodic memory. This implies that our episodic memory contains meaningful experiences that we have acquired during our entire life span from the moment we were developing our semantic memory. That

is why it is stated that episodic memory starts with the acquisition of language skills, which make it possible to store general knowledge in the semantic memory and thereby make episodic memory possible. Normally people can find episodic memories from about two years of age, when the semantic memory is functioning enough to make episodic memories possible. Does this mean that we do not have memories from before two years of age? Human beings are unable to retrieve birth memories or prenatal memories from the episodic memory system, but prenatal research has shown that we have other memory systems that make it possible to remember birth and womb experiences (Chamberlain, 1998). Wade (1998) has offered an overview of possible memory systems that can explain this human possibility to remember beyond the limitations of the episodic long-term memory. There is evidence for a physically transcendent memory and a cellular memory source.

The memory decline in AD normally begins with the working memory, which makes it difficult for patients to update data essential for the successful execution of a task, or to retrieve what they have done a few moments ago. In everyday life this implies that with the slightest distraction they forget what they are doing, or they get confused when telling a story, or they forget what they have just been eating or that they have been eating at all. Later in the process the episodic memory system begins to fall apart. This will mean that patients can't remember an ever-growing larger part of their personal history. 'Was I married?' 'Do I have children?' 'What work did I do?' These remain unanswered questions for the patient.

In AD the decline of the declarative episodic memory is not random, but a pattern can be distinguished. Clinical evidence shows that recent episodic memories are lost earlier than older ones. This means that it is possible that an Alzheimer patient cannot remember what he experienced 5 years ago, even though he might have gone through major life-events, such as, losing a spouse, retiring or having serious health problems. At the same time, however, he can remember his childhood fairly well, and with lively details. Essentially, the parts of their lives that AD patients remember goes further and further back in time. The Dutch gerontologist Huub Buijssen (2001) has called this process the gradual and backwards rolling up of the memory. It is also called retrogenesis (Reisberg & Franssen, 1999).

In my clinical work I made a distinction between remembering and reliving (Verdult, 1993). In the initial stage AD patients can retrieve information from the episodic long-term memory, in the same way and with the same limitations as non-patients do. In this beginning stage

the short-term memory is declining, but the long-term episodic memory stays relatively intact. I explicitly use the words 'to remember', because in this initial phase of AD the patient knows when he is talking about his life-span experiences. In other words he can distinguish between actual and past experiences. There is no confusion in time.

However, from the moderate stage of AD on, patients start to live in the past and this past goes further and further back. A qualitative shift takes place in the way an Alzheimer patient deals with memories. Life span experiences are no longer retrieved from the episodic memory in the normal way within the right context of the life span, but they are re-experienced in a regressive way. Alzheimer patients gradually start living in the past. As a rule it can be seen in clinical practice that the more AD patients deteriorate the more they go on re-experiencing the past, and the more they go back in time (Verdult, 1993). Essentially, the part of their lives that they relive or re-experience is further and further back. This process is also called emotional retrogenesis. Mrs. A. was reliving the punishments she received from her violent father, as if she was experiencing his violence in present time. Mr. B. who survived the holocaust was in fact living in a nursing home, but he himself is reliving his concentration camp experiences, which were triggered by the closed doors of the ward. Mrs. C. was re-experiencing her childhood sexual abuse, which made her very defensive in the bathroom during her daily care. These patients have lost the ability to place their life span experiences in the right context. To them these past experiences are happening now and they act according to the feelings that go with these experiences.

Emotion-Oriented Care

When the degenerative process goes on, many Alzheimer's patients become dependent on care. This means that their remaining quality of life mostly depends on how they are taken care of. After the Second World War dementia care showed many of the same characteristics as total institutionalized care, as described by Goffman (1968). His critical analysis of the position of mental patients caused a major shift in psychiatric care. Sociotherapy emerged stating that if the environment can have a devastating influence on cognitive, emotional and social functioning of patients in long-term care facilities, then the environment should be changed in such a way that positive effects can be reached. In geriatric care sociotherapy was introduced in Europe in the late seventies (Verdult & Pelgrims, 1986). As long as there is no

cure for AD, improving and maintaining the quality of life of people with AD should be the highest priority in care giving. During the last decades several psychosocial treatments have been developed in dementia care (APA, 1997). Supportive therapies were generated as well. Folsom (1968) introduced reality orientation. Psychomotor therapy, music therapy, reminiscence, sensorimotor therapy and occupational therapies were also introduced (Verdult, 1989). The therapeutic nihilism was left behind.

A new culture of dementia care had arisen. This shift in care stressed the uniqueness of the patient, a respect for his subjectivity in experiencing events and an answer to his psychological disintegration of a caring and supportive relationship (Kitwood, 1997). Until then it was thought that Alzheimer patients did not have any insight in their disease, and thus were not experiencing their decline. Feelings and emotional needs of Alzheimer patients were denied because of the emphasis on rational insight into symptoms and behaviors. Not knowing, or not being able to describe what exactly is happening to you, does not mean that one has no feelings and no emotional needs. Clinical observations and research have proven that Alzheimer patients respond to their illness, even long after their illness-insight has disappeared. Emotional awareness by AD patients receives greater acknowledgement (Miesen, 1997), yet more responsiveness is needed, for example, AD patients experience the deterioration process whereby they lose their autonomy and identity. This awareness-context brings the patient into a chronic traumatic experience. Becoming demented is not only a confrontation with cognitive impairment, but also with a disintegration of personality. Elderly having AD can be truly called patients in the real sense of the word, namely, they are suffering. In sum, AD is a painful and threatening process (Verdult, 2003).

In the eighties Naomi Feil introduced a new method for communicating with Alzheimer patients. This counseling method was called validation therapy (Feil, 1982). Her message was: 'step into their shoes' instead of trying to orient Alzheimer patients to the reality of the health care system. It was believed better to enter their inner world, acquiring insight into the emotional and associative way in which Alzheimer patients respond to their perceptions and experiences, and communicate with empathy to their feelings and emotional needs. Her method meant a new direction in the care for dementing elderly, namely, emotion-oriented care (Verdult, 1993). Surprisingly, historic research showed that Alois Alzheimer himself already pleaded for a 'non-restraint' method in the care of psychiatric

patients, including the patients who suffered from the disease that later became to carry his name (Jürs, 1999). Research on emotion-oriented care showed that demented nursing home residents were more balanced emotionally. They also had a smaller increase in dissatisfaction with the current situation (preservation of positive self-image) (Finnema, 2000). Emotion-oriented care does not have positive effects on cognitive and social adaptation but does improve the functioning of Alzheimer patients, although there is limited evidence for the effectiveness (Finnema, Droës, Ribbe & Van Tilburg, 2000). Problem behavior, such as apathy, depression and aggression, does not seem to be reduced by emotion-oriented care however (Schrijnemaeker, et al., 2002). Most of the mentioned research is done in the Netherlands where there is a high level of institutional care. Research on the effect of validation therapy on American nursing home residents showed a slightly positive effect on problem behavior, especially depression and verbal aggression (Toseland, et al., 1997). These studies can be criticized. The number of studies are limited; the studied populations have a high degree of heterogeneity; the research is mostly done on validation therapy and not on other emotion-oriented models; although the goals seem comparable, namely improving the well-being of patients, the methods to achieve these goals can be totally different; and finally, nursing staff only received short introductions to, and training in, the used approach. In my opinion a valuable research study would be one where the attitudes or viewpoints of nursing staff could be changed in the direction of a person-centered emotion-oriented care with the hypothesis that the effects on the emotional functioning of AD patients would improve.

The Experiential World of Alzheimer's Patients: Loss of Autonomy

Continuing with this theme, a new perspective in dementia care was introduced with more empathy for the suffering of AD patients. However, most new models lack a comprehensive view on the experiential world of AD patients. That is why I introduced a new model for describing the inner experiential world of AD patients. I called this model a *person-centered experiential approach* (Verdult, 1993, 1997, 2003). A crucial assumption in this model is that anxiety is the basic feeling of Alzheimer patients. The loss of cognitive abilities and consequently the loss of autonomy and identity make Alzheimer patient suffer painfully. Their growing insecurity and despair undermine their inner strength. In my model the inner world of Alzheimer patients can be described by four main factors: the loss of

autonomy, the process of retrogenesis, hospitalization and its long-term effects, and personality changes. In this article I shall only discuss the loss of autonomy and retrogenesis.

The threatened self. The loss of autonomy makes it difficult for patients to produce decisions and judgments, to fill their lives with purpose, and certainly, to take care of themselves. AD patients become more and more depended on their caregivers. In the initial stage of the disease the environment can conceal first manifestations because they mostly don't interfere significantly with daily activities. The patients, however, are often aware of, and attempting to understand and deal with, the functional decline. Cortical information processing is still possible, although problems arise more and more. Becoming more forgetful, misplacing things, minor speech difficulties like not finding the right word, or orienting problems, can be denied or minimized by the patient. The mental changes associated with AD are in this early stage rather non-specific and mainly experienced by the patient himself.

This initial stage I call the stage of 'the threatened self'. As the patients are experiencing the ever-growing loss of capabilities, the future is under threat: 'What's going on?' 'What can I do about it?' or 'Where will this end?' The patients can feel insecure and uncertain. The more they are confronted with their losses, the more they start to grieve. General grief responses, such as denial, sadness and despair, can be commonly seen in AD patients. Alzheimer patients have a period of denial, in which they try to minimize their dysfunctions. They try to prove to themselves that they are still in control and nothing serious is happening. After this period of denial more grief responses becomes apparent. AD patients can be very sad, sometimes depressed; despair can lead to outbursts of intense crying; unable to accept their decline patients can become angry. As the AD process is a fight one can never win, the ongoing decline can make the patient desperate.

The lost self. In this manifest stage the progress of the mental deterioration is dominated by dysmnesia, dysphasia, dyspraxia and dysgnosia. These symptoms lead to further loss of autonomy, daily life gets more confused and taking care of oneself becomes more difficult. Disorientation in time, place and persons make patients more anxious. The AD patients behave somewhat out of control and organizing their lives is difficult. Their coping strategies start to fail more and more and this makes it difficult to handle daily life situations. Emotional information processing is dominant in this stage.

I call this manifest stage of the Alzheimer's process the 'the lost self'. In this stage the loss of control is producing a lot of anxiety in

patients. If one does not know where one is, or what is expected in a situation, or what is being said to you, then life becomes out of control. Patients are more and more looking for containment. By creating controllable situations, by collecting items, by sticking more to rituals and habits, by making their world smaller, or by claiming contact, these patients try to establish safer environments. By using the 'strange situation' as research model, Miesen (1992, 1993) showed that Alzheimer patients have attachment patterns just like children do. The classical Bowlbian attachment patterns can be seen in Alzheimer patients. These findings show that AD patients feel more and more insecure and look for attachment figures that can offer more security and containment. In this stage the process of retrogenesis gets started and identity loss generates feelings of being lost.

The vanishing self. In the final or terminal stage AD patients become fully depended on their caregivers. The mental deterioration continues, involving most cognitive functions. Declarative memory disorders are complete, verbal communication becomes sporadic and unreliable, endless repetition of movements can become apparent, the patients become apathetic. More and more the patients seem to be locked up in an inner world that loses contact with the outer world. The anxiety results from the bodily-felt isolation, which is a not-human condition. Mental functioning is reduced to sensorimotor information processing.

I call this terminal stage the stage of the 'vanishing self'. Their experiential world can be described as a prototaxic way of being. By a prototaxic experience Harry Stack Sullivan meant a way of being in which there is no sense of time and place, and one experiences oneself as separated from the environment (Chapman & Chapman, 1980). In the final stage of Alzheimer's disease, other people no longer seem to exist and an interpersonal world changes more and more into an intrapersonal world. Said another way, the AD patient closes himself off from the outside world and seems to vanish into a difficult inner world in which body sensations and elementary sensorimotor input seem to dominate. It is in this final stage that prenatal themes start to reappear. The body's posture and movements become more and more fetal.

Retrogenesis: Loss of Identity

Barry Reisberg, professor of psychiatry at the New York University School of Medicine created the theory of retrogenesis in AD, stating that the decline in AD mimics regression toward infancy. Clearly AD

patients are unable to function as adults (i.e., the inability to hold a job, handle finances, or solve abstract problems). They lose social and moral functioning, the ability to make choices about clothing, the ability to take care of themselves, speech, and lose control over bladder and bowels. Thus, Reisberg's research gives us a better understanding of the process of Alzheimer's disease. Retrogenesis brings more definition to the AD process. I myself have described this process as a return to childhood, as a process of losing identity (Verdult, 1993). When AD patients start to live in the past they have lost the largest part of their history. For example, if an AD patient starts living in his adolescent period, reliving his boarding school experiences, he then has lost his identity as an adult, a middle-aged and old-aged person.

Patients with Alzheimer's *reverse develop*. Clinical and neurological studies have compared the mental and physical stages of infant and child development to the reverse process of AD. This is also called a process of degenerative developmental recapitulation. Retrogenesis is defined as the process by which degenerative mechanisms reverse the order of acquisition in normal development (Reisberg & Franssen, 1999; Reisberg, Franssen, et al., 1999; Reisberg, Franssen, Souren, et al., 2002). A distinction can be made between neuropathologic, neurologic, functional and cognitive retrogenesis (Rogers & Arango, 2006). According to the theory of neuropathologic retrogenesis parts of the brain that undergo late myelination in child development are first to undergo demyelination and develop pathologic intra- and extra cellular changes in AD. From Paul McLean's triune brain evolution theory (MacLean, 1990) it can be deduced that AD patients degenerate from cortical functioning, to limbic system functioning, to brain stem functioning. The brain deterioration in AD patients reflects the reverse of brain evolution in mankind. Neurologic retrogenetic research has shown that the so-called developmental reflexes, like sucking, hand and foot grasp reflexes, the rooting reflex and the Babinski plantar extension reflex re-appear in patients with advanced stage AD. Two Dutch researchers of Reisberg's team have shown that the motor decline in AD is mirroring the psychomotor development of the child (Souren & Franssen, 1993). Infantile reflexes, which mark the emergence from infancy in normal development, are indicators for the terminal stage of AD.

As there is a link between neuropathological declines in AD and progressively worsening cognitive and functional abilities, it can be expected that the deterioration of the brain can be linked to clinical manifestations in different stages of the AD process. The stages in AD can be translated into corresponding developmental ages. Functions

learned in childhood, like clothing and self care are being lost in AD. In cognitive retrogenesis the cognitive performance mirrors the pattern of cognitive development in children as described by Piaget (McGregor, 1991; Verdult, 1993). Patients in the initial stage still have cortical functioning and Piagetian formal operational functioning. Mildly demented patients return to concrete operational and preoperational functioning, while severe or terminal patients return to a sensorimotor level of cognitive functioning.

I myself described the emotional retrogenesis in AD as a crucial part of the loss of identity (Verdult, 1993). In the decline of AD three aspects of emotional retrogenesis can be distinguished. These are the re-living of emotions that belong 1) to social roles, 2) to traumatic lifespan experiences, 3) and to unfulfilled basic needs from childhood and infancy. Mrs. D. can be called a schoolbook example of emotional retrogenesis. In the stage of the lost self, she started to look after her small children and became very upset when she could not do so, thinking they might get lost after school. About a year later she experienced herself as a young girl, needed to go home, because her parents were waiting for her. About another year later she regressed to become a toddler playing with her feces when she was angry about something. When I left the nursing home she was in fetal position, lying in bed all day.

In emotional retrogenesis Alzheimer patients normally start to re-experience former social roles that they fulfilled in their early adult lives. Usually they return to age of about 25 to 30 years old. Mrs. E. is an eighty-year-old woman who was a secretary in a big company. Every morning she was looking for the way out to go to work, and became frustrated and angry at not being allowed to go, fearing that she might get fired. Dr. F. was a retired physician who did his consultations on the ward, not only giving advice to his fellow patients, but also trying to examine them. Mr. G. was a fisherman and in the nursing home he would still bring baskets full of fish to the fish market, where he tried to sell his product.

This period of re-experiencing former social roles is followed by a period of re-experiencing personal traumatic experiences. I agree with Feil that Alzheimer patients re-live old conflicts and traumatic experiences (Feil, 1982). I don't agree that they do this in order to resolve these conflicts and traumas, and thereby restore their dignity. Not only have I never seen this in my clinical work, but also there are psychotherapeutic and theoretical considerations that underline my thesis and suggest that this is not possible in Alzheimer's disease (Verdult & Visser, 1990; Verdult, 1991). Their re-living can be seen as

a way of trying to resolve trauma on an unconscious level, but no healing, in the strict sense, takes place on this level. This is visibly a painful reality for AD patients. Mrs. H. was a 75-years-old AD patient who could be in full panic because she was delivering a baby and the midwife (it was in a Dutch nursing home!) was not there yet. Mr. J. was a prisoner of war during World War II and during his emotional retrogenesis he anxiously re-lived his war experiences, fifty years after the ending of the World War II. Mrs. K. was 82-years-old and every day she sat for hours at the window waiting for her parents to come and get her. It is a well-known fact that Alzheimer patients can look for their parents as if they are still alive. Miesen (1992) called this process in Alzheimer patients parent fixation. Mrs. L. was a 73-year-old woman who almost daily escaped from the nursing home where I worked. We usually found her back at her childhood house where she lived with her parents in the first fifteen years of her life, sitting in the front of the house waiting for her parents to come home.

The process of retrogenesis has been questioned. First, behaviorally-oriented practitioners don't believe in a "second childhood" for Alzheimer's patients. This is because in general they deny the influences of past experiences on actual functioning. Second, and the more fundamental question is: How far can Alzheimer patients regress? Scientific psychologists state, if they accept the hypothesis of retrogenesis, specifically, that AD patients can regress to the beginning of memory, do they mean the declarative, episodic memory? Episodic memory requires a certain level of language acquisition, so this usually is at the age of two or three years old. But in my clinical practice I have seen patients sucking on everything they can get in their hands, just like babies do. I have also fed patients, unable to feed themselves or to be fed with a spoon, with milk bottles. I have seen terminal stage patients in a fetal position showing birth movements. Reisberg's model of retrogenesis is based on the premise that development starts at birth. He spoke of a mild AD patient with mental abilities and habits of a 12-year-old child regressing to levels of successively younger children and even infants (Reisberg, 1999). Prenatal psychology has shown the scenario differently: prenatal and postnatal development demonstrates continuity. Postnatal life is a continuation of prenatal life. Human development does not start at birth but at conception. Why then should the process of retrogenesis stop at birth? In my view the process of deterioration in AD stops where development has begun, and that is at conception. Or stated otherwise: where life or consciousness began, Alzheimer's disease will end (Verdult, 1997).

Prenatal themes. Emerson (1998b) stated that recapitulation is the most important concept in understanding the impacts of birth, prenatal life and obstetrical interventions. He defined recapitulation as the process whereby people unconsciously recreate past events and traumatic experiences in their lives. They do so in an attempt to externalize traumas from the unconscious so that they might be dealt with in here-and-now-reality, and cathartically released from their systems. Recapitulation and retrogenesis differ on the aspect of adult functioning. In recapitulation the person still keeps his adult functioning, or can come back into adult functioning. It might be that his unconscious takes over control, but regardless, there always remains a part of a functioning adult. In retrogenesis no return to adult functioning is possible; the here-and-now has become the there-and-then. Present and past fall together. In this contribution I don't go into recapitulation, although I don't deny that this process is also active in AD patients, as it is in all adults. What I want to describe here is what Alzheimer patients do when they are re-living birth and prenatal life.

The process of retrogenesis gradually brings Alzheimer patients further and further back into childhood, infancy and finally into birth and prenatal life. Let it be clear that not all AD patients re-live prenatal life, only very severe terminal patients can re-experience prenatal themes. Most patients have died before they even enter this final stage of dementia.

Birth. As perinatal experiences and possible trauma can be stored in the body, birth movements can be seen in terminal patients. If they still can change lie-positions, to my experience, terminal Alzheimer patients prefer to lie on their birth lie-side, when they have returned to fetal position. The birth lie-side is the position of the baby during the birth process. It is the side of the baby that lies on the mother's spinal cord, and this can be seen in postural body signals (Emerson, 1996a). These postural signals of birth can also be observed in AD patients. When AD patients are turned on their non lie-side, in the context of a decubitus (bed sore) preventive strategy or because nurses don't respect the preferred lie-side, they try to turn back to their birth lie-side. If they are unable to turn themselves, restlessness occurs. Emerson (ibid.) also described the birth process in four stages, based on movements of the baby's head and torso in relation to the mother's pelvis. Stage 1 is the stage of the transverse or descending cranium in which feelings of being stuck can be dominant. Mrs. M. was an 81-year-old patient in the severe final stage of dementia. She was lying in bed almost all day, and mostly in the fetal position, in flexion (chin to the

chest). Her legs were pulled to the abdomen. In this position she was often seen head-banging against the frame of the bed, which can be interpreted as a retrogenetic re-living of Stage 1 birth trauma. Moving her from her chair into bed, a transition, could activate a sympathetic shock pattern in her; she could be shouting intensively, trying to bite the nurses. Transition and cooperation are also Stage 1 themes. In Stage 2, the rotational cranium, the head turns towards the sacrum. This rotational movement can be observed when a terminal AD patient turns in his bed not on his back, but with his head down, as if the mattress was the pelvis. I have seen AD patients saying "No" for hours, a well-known symptom of Stage 2 trauma. In Stage 3 the head is going to be born and it has moved to extension. One can see the typical Stage 3 posture, head in the neck, in severe patients. The pushing of the legs is typical in this stage. Mr. N. was put in his wheel chair to go to the bathroom for a shower. He was a 77-years-old terminal and severely demented man. When he was set up he put his head in his neck and started to push with his legs, trying to get out of his fetal position into an extension position. If cord complications have occurred during birth, it can be observed that patients have a tendency to move backwards and they can be extremely activated if they are covered with blankets.

As I mostly worked in Dutch nursing homes, and with patients from the cohort of 1910-1930 of whom 99% were born at home, no birth trauma due to obstetrical interventions were observed by me. I can imagine that Alzheimer patients born cesarean can show the themes that perinatal psychology associates with this traumatic birth. Tactile defensiveness, difficulties with being interrupted, experiencing loss of control easily, feeling invaded quickly, or showing shock patterns related to the cesarean surgery as can be seen in babies and adults born by this method (Emerson, 1998; Verdult, 2009a). I presume that these manifestations can be observed in Alzheimer patients who relive their traumatic birth experiences.

Prenatal life. All early experiences leave imprints in our bodies and shock experiences leave the most intense imprints. The earlier in life shock takes place the more intense the bodily imprint will be. Prenatal shock will program those brain systems that are in a critical stage of development, especially the brain stem and the oldest parts of the limbic system, and their connections to the autonomic nervous system. Shock can program the autonomic nervous system permanently. Prenatal shock is stored in the body as patterns of tone and tension, as postures or movements (Emerson, 1996b). The hypothesis of retrogenesis would deduce that the earlier the imprint has taken place

in prenatal life, the longer it can be activated in AD. Or said otherwise, the earlier programmed, the later activated in AD. In sum, prenatal shock activations can be seen in AD patients in body symptoms and body movements associated with shock.

In the terminal stage (the 'vanishing self' stage) a distinction is made between the locomotory and sensory sub-stages. Repetitive motions and sounds characterize the locomotory sub-stage of the terminal stage of AD. The symptoms in this sub-stage can be associated with sympathetic shock activation. A terminal AD patient can demonstrate feeling endangered when he is actually responding to past associative links from his prenatal traumas. As the vulnerability increases in AD, even minor interventions can lead to activation. His sympathetic nervous system can be 'turned on' causing a higher arousal, greater hyperactivity, accelerated and deeper respiration in response to the need for more oxygen, an increase of blood flow to the muscles and decreased blood flow to the cortex, increased vigilance to the environment, and suppression of all physical system not essential for defense. His sympathetic activation is a defense against threat. If the sensory sub-stage movements vanish, an increasing loss of self-awareness can be observed; patients don't respond unless through a combination of close contact, nurturing touches, soft speech and eye contact. This sensory sub-stage can be seen as parasympathetic shock activation. As relational isolation and lack of self-awareness increase, anxiety intensifies too. If the sympathetic defense systems (fight-or-flight) are unsuccessful in assuring safety, the parasympathetic nervous system (or freeze) becomes the next line of defense (Porges, 2001). The parasympathetic reaction can be described as follows: many functions of the body begin to slow down, leading to a decrease in heart rate and respiration, a sense of numbness, shutting down within the mind, and separation from the sense of self. This could be an exact description of the sensory sub-stage of the 'vanishing self stage' in AD.

Of course these interpretations are speculative. The first problem is that body functions, like breathing, cardiovascular circulation, thermoregulation, digestion, secretion, motion, and neurological functioning, decline in AD, especially in the terminal stage of the disease. The autonomic nervous system declines along with the central nervous system. This makes it difficult to distinguish between the symptoms related to the deterioration of the body functions and shock activation. The second problem is that little or nothing is known about the prenatal life of patients. Most patients don't, nor do their families know anything about their prenatal life, but clinical hypotheses can be confirmed by the results of the treatments that are based on these

hypotheses. An example can illustrate my position on this subject. Mrs. O. was lying in bed in fetal position for almost one year when I first met her. It was not possible to communicate verbally with her. The nursing staff found out that she became more open for contact when they used vibroacoustic stimulation. I became involved with Mrs. O. because the nurses observed that she could get “aggressive”, as they called it, in certain situations. For me Mrs. O. was a vulnerable prenat, who felt invaded by too much sensory input and by nurses who asked too much of her. My hypothesis was that she had experienced a strong umbilical affective shock, a prenatal defensive against people who gave too little love and attention. When the care giving of Mrs. O. became less invasive, and changed into more ‘giving’ instead of ‘asking’, her ‘aggressiveness’ was reduced. I considered this as a confirmation of my hypotheses, knowing of course that other interpretations are possible.

To discuss all possible traumatic experiences and their re-appearance in retrogenesis in AD would be lengthy. However, I have seen severe AD patients showing ‘naval radiation’, as described by Hartley (1995). Prenatal psychomotor development is organized around the navel center, from which it radiates through the whole body. Navel radiation is associated with umbilical affect. Frank Lake defined umbilical affect as the feeling state of the fetus as brought about by blood reaching him through the umbilical vein. This maternal-fetal ‘affect flow’ transmits the emotional state of the mother to the fetus by way of the umbilical cord (Maret, 1997). This affect flow forms the blueprint for later attachment relations and every prenatal trauma will affect this first relationship negatively. Personally, I think that Frank Lake’s ‘ideal stress/trauma’ index can be associated with safe attachment; his ‘coping’ level of prenatal stress with ambivalent attachment; ‘oppositional stress’ can be linked to avoidant attachment and ‘trans marginal stress’ to disorganized attachment patterns (Verdult, 2009b). As demented patients show attachment behaviors (Miesen, 1992, 1993), I myself am looking for attachment signals, even in severely demented patients, like in the ‘vanishing’ stage. Disturbances in the prenatal attachment relation can manifest in a threat to dissolve/ disintegrate, a threat of being destroyed, of being expelled, or of being invaded (Krens, 2001). In the process of prenatal emotional retrogenesis the threat to dissolve can be seen in strong self-attachment behaviors (i.e., sitting with arms strongly crossed over the chest, or repetitively caressing one’s own hands/arms); the threat of being destroyed can lead to strong defensive behaviors (i.e., pushing away people, or overstretching/arching). And the threat of being

expelled can lead to reaching out for contact and showing sucking and clamping behaviors. The threat to be invaded can be seen in defensive and aggressive behaviors or in extreme flexed body positions. Umbilical movements can be observed in patients in the terminal stage of AD.

I can imagine that cellular movements, as described by Farrant and Larimore (1995), can be observed in prenatal AD patients. When I worked with Alzheimer patients, I myself was not yet trained enough in these early stages of prenatal development to be able to observe these egg and sperm movements in patients. But I would not rule it out that preconception and conception imprints can be retrogenetically activated in AD.

The Symbolic Womb: Prenatal Emotion-Oriented Care for Alzheimer Patients

As a developer and strong supporter of emotion-oriented care (Verdult, 1993), I try to promote an extension to prenatal care in AD (Verdult, 1997). Three major components of Prenatal EMotion-oriented cARE (PREMARE) can be identified, namely: 'snoezelen' (sensorimotor relaxation), the adapted 'womb'-environment, and the attitude of the nursing staff.

' *Snoezelen* (sensorimotor relaxation). In severe AD patients the senses are the gateways to contact. 'Snoezelen' is a psychosocial method, developed in the Netherlands, which employs active sensory stimulation to share and enter into the experience of the demented patient in an attempt to increase and maintain his/her well-being (Achterberg, Kok & Salentijn, 1997). 'Snoezelen' was originally developed in the field of mentally disabled persons. It was introduced in geriatric care in the late eighties. 'Snoezelen' refers to primary activation by selective input of sensory stimulation for deeply regressed or withdrawn patients. The word is a combination of the Dutch words 'snuffelen' (to sniff) and 'doezelen' (to doze). It is also referred to as multi-sensory stimulation. It can be defined as an approach which actively stimulates the senses of hearing, touch, vision, taste and smell in a resident-oriented, non-threatening environment. It is intended to provide individualized, gentle sensory stimulation without the need for higher cognitive processes, such as memory and learning, in order to achieve or maintain a state of well-being. It can be applied in a special room with an array of equipment, offering multiple stimulations, and covering all sensory channels (Van Weert, 2004).

I prefer to call it sensorimotor relaxation, as the goal should be reducing anxiety by relaxation in a safe environment. In my approach of 'snoezelen' three aspects are important: first 'snoezelen' is a way of making affective body contact, secondly the goal should not be stimulation but relaxation, and thirdly the kind a sensory input offered to severe terminal patients should reflect the retrogenesis of the sensory development in prenatal life (Verdult, 1997).

First, I presume that human contact remains essential in AD. This contact is not rational using cognitive processing through the neocortex, but emotional and sensorimotor, using emotional processing of the limbic system and sensorimotor processing of the brain stem. By 'snoezelen' a sensorimotor atmosphere of safety, bonding and containment can be created. It can be said that a couple that creates a romantic atmosphere to get closer, is using 'snoezelen' to get into deeper contact. The same is true for terminal AD patients. This atmosphere can open the AD patient, can break through the isolation, and can make emotional body contact possible. A relationship can be re-established with severe AD patients who have a strong tendency to withdraw in a closed world. I wish to stress that this kind of body contact must respect the patient's boundaries, for several reasons. Touch is an emotional stimulus that originates from the earliest prenatal periods and thus remains intact until the last moments of the Alzheimer process. Gentle touching helps severe AD patients to orient to the outside world, and makes contact on an emotional level possible. As anxiety is the basic feeling of AD patients, every contact should attempt to reduce this anxiety.

Second, originally the aim of 'snoezelen' with the mentally disabled was to provide an opportunity for intellectual and social development. That's why stimulation was stressed. But in AD patients the goal of 'snoezelen' should not be stimulation but relaxation within the boundaries of a bonding relationship. Stimulation (or enrichment) has the risk of overstimulation, and this risk increases sharply as the more severe patients lose more neural connections. Sensory input should be fine-tuned to the AD patient's rest-capability to make sense out of sensory input. I have seen too many special 'snoezel' rooms that resemble disco techs, overloading patients with highly intense and diverse sensory inputs. This brings no relaxation, no opening, no contact, no safety, but more activation or withdrawal.

Third, in the prenatal development of the senses a more or less fixed pattern can be seen. The first tactile senses and the initial elements of the proprioceptive system develop. The auditory senses starts to develop from 16 weeks of gestation; then taste and smell. The

visual sense is last to develop. If one accepts the thesis of retrogenesis, this would imply that Alzheimer patients keep responding to sensory input in the reverse way. The visual sense disappears before taste and smell. The auditory sense would start to decline thereafter. If vision, taste, smell and sound have disappeared, vibro-acoustic hearing, elements of proprioceptive sense and touch remain relatively intact. Touch is the last sense to remain. To disappear in the context of the deterioration in AD means that the senses remain relatively intact, but the brain connections have been lost, making sensory information no longer able to be processed. 'Snoezelen' with fetal terminal AD patients should take this reverse prenatal development of the senses into account. Visual stimulation can be offered in the beginning of the 'vanishing stage'. This can be done with spotlights, colorful materials, bubble units, flashlights linked to music, or video projections. When patients no longer respond to visual stimuli, aromatherapy can be used to stimulate smell sensations. Through aroma streamers, aromatic oils that reduce stress and anxiety can open up the patient. Taste sensation can be given if patients are extremely orally oriented. Sweet is preferred. When severe patients start to close their eyes more and more and seldom open them, auditory stimuli can be given. Music therapy is very suitable. Rhythm is important and the closer this rhythm is to the heartbeat the better. As we know from the research by Tomatis (1991) prenatates, and thereby severe terminal Alzheimer patients, prefer Mozart to Beethoven, and Vivaldi to Bach. Prenatal hearing is actually more like vibro-acoustic stimulation. Sound vibrations reach the fetus through the uterine wall and stimulate the whole body, not just the ears. Vibrations can be very relaxing. A Belgian company developed a special chair through which vibrations can be given to the whole body. Research showed that this vibrating chair could achieve deep relaxation. Also waterbeds are used, sometimes in combination with vibrations. Hydro sound baths can be very relaxing. The bath is like a womb and the vibrations, which can be modulated in intensity, can be experienced as prenatal vibration on the body. The deeper the patient is vanishing, and the deeper he is in his prenatal life, the developmentally youngest gateways to contact should be used. As Montague (1971) showed, touch is the most important sense. It starts to develop as early as 6 weeks after gestation. As it develops that early it remains intact a very long time in AD. So the most important way of communicating with vanishing patients is by touching. Words no longer reach them and don't make sense to them, but through affective body contact primitive bonding can occur.

The adapted 'womb' environment. In the context of 'snoezelen' or multi-sensory stimulation for AD patients the questions are whether the environment should be enriched and in what ways it should be enriched. Research on sensory stimulation showed that a sensory enriched environment has positive effects on mood and social functioning of AD patients (Baker, Dowling, Wareing, Dawson & Assey 1997; Spauull & Leach, 1998). How severely demented the researched patients were is unclear. In the beginning of my experimental work with 'snoezelen', I worked with Mrs. P. She was 79-years-old and severely demented. She had not spoken for many months, lying in bed most of the day, being fed with a straw. I brought her to the special 'snoezel'-room, where we had all kinds of materials with which we could stimulate sensory input, like the *feel-bag* filled with rice and other products, light and sound effects, mirrors, dolls and teddy bears, bubble units and so on. After 20 minutes working with Mrs. P, trying different inputs, all the sudden she said, loud and clear: 'When is this going to be finished?' She dropped into her isolated state again and withdrew completely from the situation, which in my opinion was too overloading for her. I learned from Mrs. P. that overstimulation in severe AD patients can have reverse effects; too much multimodal sensory input creates anxiety and the only defense she had was to withdraw. My conclusion was that an enriched environment should be tuned in with the emotional, communicative and cognitive disabilities of the patient.

PREMARE should be provided in a special environment that has the qualifications of a 'good-enough' womb. According to me, a 'good-enough' womb should have the following characteristics: (a) it is a cocoon in which one can feel safe and secure; (b) the boundaries of the cocoon provide protection; (c) there is a continuous supply of nutrients necessary for well-being; (d) the umbilical dialogues are enriching and facilitating, and not burdening or demanding; (e) there is no overload of sensory input and uni-modal information processing is possible; and (f) there is no stress nor anxiety. These characteristics can be translated to the good-enough womb in PREMARE. The patient's bed or room, and the nursing home ward, or the special 'snoezel'-room in the nursing home should all have these characteristics.

Severe terminal Alzheimer patients remain most of the day in bed and this area can be turned into a womb easily, so that a cocoon is created. With pillows a womb can be constructed that fits to the prenatal posture of the fetal AD patient. With a mosquito net the womb can be closed. A dimmed red and blue light can create the visual surroundings of a womb.

Boundaries of the symbolic womb are to facilitate protection and this implies that they should be respected. Nurses are not allowed to enter the womb surroundings without permission. This is of course not verbal permission, but through body signals patients can communicate openness and willingness for contact, or withdrawing, rejection and closing. Invasive interventions are avoided. No pressure or stress is put upon the patients.

Scheduled routines are no longer suitable. As these patients have less aware moments, personal care is carried out in the more aware periods, not according to planned schedules. Feeding is also done in the more aware moments and can be done by the bottle, as sucking is the best way to feed severely terminal patients. It is better to feed patients more times a day in small portions, than twice or three times a day on fixed moments. Liquid food is preferred as sucking remains the last way of food-intake. If AD patients start to suck more, a straw can also be used to feed them.

The 'umbilical dialogues' with fetal AD patients will be reflected in the nursing staff attitude of person-centered and emotion-oriented care. As we have already seen, sensory input is reduced and selectively offered to the patients, depending on his/her level of prenatal sensory functioning. Multimodal stimulation is avoided, as overstimulation produces more anxiety. The good enough womb offers uni-modal stimuli.

The attitude of the nursing staff. In PREMARE the attitude of the nursing staff is crucial for the well-being of the patients in the 'vanishing stage' of the deterioration process. According to me, this attitude should have the characteristics of prenatal bonding relationship, as severe terminal AD patients relive prenatal life. I presume that the need to bond and to receive adequate resonance remains intact. The prenatal bonding and resonance basic emotional needs of the fetal AD patient are: (a) the need to be contained and not lose boundaries or be overwhelmed; (b) the need to feel safe and not feel threatened by unexpected harming interventions; (c) the need to feel connected and not be isolated or rejected; and (d) the need for space and not to be invaded or violated (Stroecken, 2000; Krens, 2001; Verdult, 2004; Verny, 1996).

The nursing staff needs to have the qualities of the 'good-enough' mother, as described by Winnicott, offering containment, holding and affective body contact (Verdult, 1994). Mothering qualities are crucial in this final stage of dementia. As Perls stated, contact is an ego-function, but terminal AD patients have lost contact with reality, lost contact with their inner feelings and needs, and lost the possibility of

verbal communication. The pre-expressive self (Prouty, 1998) still remains. This means that although the terminal AD patient is vanishing more and more, there is still a very reduced but noticeable tendency to express themselves, to convey body sensations and primary emotions, even if these sensations and emotions can no longer be integrated. As severe terminal AD patients function more on a brain stem level, nurses have to be aware of sensorimotor information processing; they have to slow down, adjust the pacing to the patient, and avoid disruptions in contact. The nursing staff has to empathically respond and accommodate to these rudimentary expressions of the self. Prouty's contact reflections (1976) have proven to be very successful in communicating with terminal AD patients. The ultimate goal remains to keep in touch with the vanishing patient, to avoid him gliding into complete isolation.

CONCLUSION

Prenatal factors play a role in the etiology of Alzheimer's disease, although the mechanisms and consequences are not yet clear. In recent years more research has shown that many diseases have a developmental origin. Epigenetic research is becoming a hot item. In the long run it will appear that AD is a complex interaction between genetic, environmental and developmental factors, including triggers that originate from prenatal life. As Alzheimer's is a very complex disease, there is still a long way to go before this dramatic illness can be stopped or prevented.

In the meantime the best possible care should be provided to AD patients who suffer intensely from this devastating disease. PREMARE is based on clinical observations, interpretations and sometimes speculations. More theory building and research should be done. I hope that this article can contribute to an improvement of care for severe AD patients, in respect of their pain.

From my clinical experience, emotion-oriented care is the best way to provide for Alzheimer sufferers. Patients need support, containment and affective contact to deal with the loss of autonomy and identity. The more they re-live childhood, infancy and prenatal life, the more they need an empathetic 'good enough' mother, who is emotionally available, sensitive and responsive to expressed emotions, body sensations and body postures dating from prenatal life. This 'good enough' nurse is not afraid of what is going on in the deepest experiential world of the fetal terminal AD patient. Ideally he or she has experienced his or her own prenatal life and birth, so that severe

terminal patients are met easily in their prenatal needs. To be seen and to be heard, as Winnicott stated, is the most crucial aspect in life, in prenatal life, in infancy, in childhood, in adulthood, in old age, and also in dementia. Emotion-oriented care giving should meet the 'vanishing patient' on this level of functioning, in this re-living of his/her past. By recognizing his prenatal experiential world, anxiety can be reduced. Essentially, emotion-oriented care means: staying with the pain!

REFERENCES

- Achterberg, I., Kok, W. & Salentijn, C. (1997). 'Snoezelen': A new way of communicating with severely demented elderly. In B. Miesen, & G. Jones (Eds.), *Care-giving in dementia: Research and applications* (Volume 2). New York: Routledge.
- American Psychiatric Association (1997). Practice guideline for the treatment of patients with Alzheimer's disease and other dementias of late life. *American Journal of Psychiatry*, 154(5 suppl), 1-39.
- Baker, R., Dowling, Z., Wareing, L-A., Dawson, J. & Assey, J. (1997). Snoezelen: Its long-term and short-term effects on older people with dementia. *British Journal of Occupational Therapy*, 60(5), 213-218.
- Buijssen, H. (2001). Dementie (Dementia). In: Buijssen, H., *Psychologische hulpverlening aan ouderen (deel 2) (Psychological counseling to the elderly (part 2))*. Baarn (Netherlands): HB Uitgevers.
- Chamberlain, D. B. (1998). *The mind of your newborn baby*. Berkeley, CA: North Atlantic Books.
- Chapman, A. H., & Chapman, M. C. (1980). *Harry Stack Sullivan's concepts of personality development and psychiatric illness*. New York: Brunner/Mazel.
- Diesfeldt, H. (2003). Neuropsychology of memory disorders in dementia. In P. De Deyn, E. Thiery, & R. D'Hooge. (Eds.), *Memory, basic concepts, disorders and treatment*. Leuven, Belgium: ACCO.
- Emerson, W. R. (1996a). *Somatic birth simulation*. Petaluma CA: Emerson Training Seminars.
- Emerson, W. R. (1996b). The vulnerable pre-nate. *Journal of Prenatal and Perinatal Psychology and Health*, 10(3), 125-142.
- Emerson W. R. (1998). Birth trauma: The psychological effects of obstetrical interventions. *Journal of Prenatal and Perinatal Psychology and Health*, 13(1), 11-45.
- Farrant, G. & Larimore, T. (1995). Six universal body movements expressed in cellular consciousness and their meaning. *Primal Renaissance: the Journal of Primal Psychology*, 1(1), 17-24.
- Feil, N. (1982). *Validation therapy: How to help disoriented old-old*. Cleveland: Edward Feil Productions.
- Finnema, E. J. (2000). *Emotion-oriented care in dementia: A psychosocial approach*. Groningen, Netherlands: De Regenboog.

- Finnema, E. J., Droës, R. M., Ribbe, M. W. & Van Tilburg, W. (2000). The effects of emotion-oriented approaches in the care for persons suffering from dementia: a review of the literature. *Journal of Geriatric Psychiatry, 15*, 141-162.
- Folsom, J. C. (1968). Reality orientation for the elderly mental patient. *Journal of Geriatric Psychiatry, 1*, 291-307.
- Geerlings, M. I., den Heijer, T., Koudstaal, P. J. & Breteler, M. M. (2008). History of depression, depressive symptoms, and medical temporal lobe atrophy and the risk of Alzheimer's disease. *Neurology, 70*, 1258-1264.
- Goffman, E. (1968). *Asylums: Essay on the social situation of mental patients*. New York: Pelican Books.
- Hardy, J. & Selkoe, D. J. (2002). The amyloid hypothesis of Alzheimer's disease: Progress and problems on the road to therapeutics. *Science, 297*, 353-356.
- Hartley, L. (1995). *Wisdom of the body moving: An introduction to body-mind centering*. Berkeley, CA: North Atlantic Books.
- Jürgs, M. (1999). *Alzheimer: Spurensuche in Niemandland. (Alzheimer: Tracking symptoms in Nobody's land)*. Munchen, Germany: Econ Ullstein List Verlag
- Kitwood, T. (1997). The concept of personhood and its relevance for a new culture of dementia care. In B. Miesen, & G. Jones (Eds.), *Care-giving in dementia; research and applications* (volume 2). New York: Routledge.
- Krens, I. (2001). The first relationship. *Journal of Prenatal and Perinatal Psychology and Health, 13*(1/2), 9-30.
- Lu, P. H., Edland, S. D., Teng, E., Petersen, R. C., & Cummings, J. L. (2009). Donepezil delays progression to AD in MCI subjects with depressive symptoms. *Neurology, 72*, 2115-2121.
- MacLean, P. D. (1990). *The triune brain in evolution*. New York: Plenum Press.
- Maret, S. (1997). *The prenatal person: Frank Lake's maternal-fetal distress syndrome*. New York: University Press of America.
- McGregor, J. (1991). Cognitive performance on Piagetian tasks by Alzheimer's Disease patients. *Research in Nursing & Health, 15*(1), 11-18.
- Miesen, B. (1992). Attachment theory and dementia. In B. Miesen, & G. Jones (Eds.), *Care-giving in dementia; research and applications*. New York: Routledge.
- Miesen, B. (1993). *Gehechtheid en dementia (Attachment and dementia)*. Nijmegen: University Press.
- Miesen, B. (1997). Awareness in dementia patients and family grieving: A practical perspective. In B. Miesen, & G. Jones (Eds.), *Care-giving in dementia; research and applications* (volume 2). New York: Routledge.
- Montagu, A. (1971). *Touching: The human significance of the skin*. New York: Harper & Row.
- Nathaliens, P. (1999). *Life in the womb: The origin of health and disease*. New York: Prometheus Press.
- Nikolaev, A., McLaughlin, T., O'Leary, D. & Tessier-Lavigne, M. (2009). APP binds DR6 to trigger axon pruning and neuron death via distinct caspases. *Nature, 457*, 981-989.
- Porges, S. W. (2001). The Polyvagal Theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology, 42*(2), 123-146.
- Prouty, G. (1976). Pre-therapy: A method of treating pre-expressive psychotic and retarded patients. *Psychotherapy: Theory, Research and Practice, 13*, 290-295.
- Prouty, G. (1998). *Pre-therapy and the pre-expressive self. Person-centered Practise, 6*(2), 80-88.

- Raadsheer, F. C., Van Heerikhuizen, J. J., Lucassen, P. J., Hoogendijk, W. J., Tilders, F. J. & Swaab, D. F. (1995). Corticotropin-releasing hormone mRNA levels in the paraventricular nucleus of patients with Alzheimer's disease and depression. *American Journal of Psychiatry*, 152, 1372-1376.
- Reisberg, B. (1999). Retrogenesis: Landmark Alzheimer's research adds scientific precision to the phrase. *Presidential Address, International Psychogeriatric Society*. Vancouver, August.
- Reisberg, B. & Franssen, E. H. (1999). Clinical stages of Alzheimer's disease. In M. J. de Leon (Ed.), *An atlas of Alzheimer's disease*. Carnforth (UK): Parthenon.
- Reisberg, B., Franssen, E. H., Hasan, S. M., Monteiro, I., Boksay, I., Souren, L. E., Kenowsky, S., Auer, S. R., Elahi, S. & Kluger, A. (1999). Retrogenesis: clinical, physiologic and pathologic mechanisms in brain aging. *European Archives of Psychiatry and Clinical Neuroscience*, 249 (suppl 3), 28-36.
- Reisberg, B., Franssen, E. H., Souren, L. E., Auer, S. R., Akram, I. & Kenowsky, S. (2002). Evidence and mechanisms of retrogenesis in Alzheimer's and other dementias: Management and treatment import. *American Journal of Alzheimer's Disease and Other Dementias*, 17(4) 202-212.
- Robles de Medina, P. G. (2004). *Prenatal maternal stress and its effects on fetal development*. Utrecht: University Press.
- Rogers, H. & Arango J. C. (2006). Retrogenesis theory in Alzheimer's disease: Evidence and clinical implications. *Anales de Psychologia*, 22(2), 260-266.
- Schrijnemaeker, V., Van Rossum, E., Candel, M., Frederiks, C. M., Derix, M. M., Sielhorst, H. & Van den Brandt, P. A. (2002). The effectiveness of emotion-oriented care on elderly people with cognitive impairment and behavioural problems. *International Journal of Geriatric Psychiatry*, 17, 926-937
- Souren, L. E. & Franssen, E. H. (1993). *Verbroken verbindingen (Lost connections)*. Lisse, Netherlands: Swets & Zeitlinger.
- Spaull, D. & Leach, C. (1998). An evaluation of the effects of sensory stimulation with people who have dementia. *Behavioural and Cognitive Psychotherapy*, 26, 77-86.
- Stroecken, G. (2000). *De stem van het jonge kind (The voice of the baby)*. Leuven, Belgium: Garant.
- Swedish Council on Technology Assessment in Health Care (2008). *Dementia: Etiology and epidemiology*. Stockholm, Sweden, SDU.
- Tomatis, A. (1991). *Pourquoi Mozart? (Why Mozart?)*. Paris, France: Fixot.
- Toseland, R. W., Diehl, M., Freeman, K., Manzanares, T., Naleppa, M. & McCallion, P. (1997). The impact of validation group therapy on nursing home residents with dementia. *Journal of Applied Gerontology*, 16, 31-50.
- Tulving, E. & Markowitsch, H. J. (1998). Episodic and declarative memory: Role of the hippocampus. *Hippocampus*, 8, 198-204.
- Van Weert, J. C. (2004). *Multi-sensory stimulation in 24-hours dementia care*. Utrecht, Netherlands: NIVEL.
- Verdult, R. (1989). De beleving van dementie; de rol van de activiteiten begeleiding (Experiencing dementia; the role of occupational therapy). *AS, maandblad voor Activiteitenbegeleiding (AS, journal of occupational therapy)*, 12, 7-10.
- Verdult, R. (1991). Vraagtekens blijven bij validation therapie (Remaining questions on validation therapy). *Tijdschrift voor ziekenverzorging (Journal of Nursing)*, 3, 88-91.

- Verdult, R. (1993). *Dement worden: een kindertijd in beeld (Dementia: A childhood in pictures)*. Baarn, Netherlands, HB uitgevers
- Verdult, R. (1994). Belevingsgericht begeleiding bij demantie (Emotion-oriented care in dementia). *Hospitalia*, 3, 179-183.
- Verdult, R. (1997). *Contact in nabijheid; snoezelen met ernstig demente ouderen (Close contact: sensory stimulation of dementing elderly)*. Leuven, Belgium: ACCO.
- Verdult, R. (2003). *De pijn van dement zijn; problem gedrag bij dementerende ouderen (The pain of dementing; problem behaviour of dementing elderly)*. Baarn, Netherlands: HB uitgevers.
- Verdult, R. (2004). De competente foetus: op weg naar hechting (the competent fetus: on its way to attachment). *Tijdschrift voor Vroedvrouwen (Journal for Midwives)*, 10(1), 15-19.
- Verdult, R. (2009a). Ceasarean birth in adults. *The International Journal of Prenatal and Perinatal Psychology and Medicine* (in press).
- Verdult, R. (2009b). Die Neuverdrahtung des Gehirns; Entwicklung des Gehirns, pränatale Bindung und die Konsequenzen für Psychotherapie (Rewiring the brain; brain development, prenatal attachment and its consequences for psychotherapy). In P. Schindler (Ed.), *Am Anfangs des Lebens (In the beginning of life)*. Basel (CH): Schabe Verlag (in press).
- Verdult, R. & Miesen, B. (1986). *Old pain? Loss experiences and dementia*. Paper presented at the 3rd Conference on Advances in Preventive Geriatrics, Bratislava.
- Verdult, R. & Pelgrims, J. (1986). Bijzondere ouderenzorg in verzorgingshuis 'De Bijster'; een sociotherapeutische aanpak (Special care for the elderly in nursing home 'De Bijster'; a sociotherapeutic approach). *Tijdschrift voor Ziekenverzorgenden (Journal of Assisting Nurses)*, 14, 417-422.
- Verdult, R. & Visser, R. (1990). Ervaringen met validationtherapie (Experiences with validation therapy), *Tijdschrift voor ziekenverzorging (Journal of Nursing)*, 15, 474-478.
- Verny, T. R. (1996). Isolation, rejection and communication in the womb. *Journal of Prenatal and Perinatal Psychology and Health*, 8(3), 287-294.
- Wade, J. (1998). Two voices from the womb: evidence for physically transcendent and a cellular source of fetal consciousness. *Journal of Prenatal and Perinatal Psychology and Health*, 13(2), 123-147.