
Neurophysiology/Neuroanatomy

The changes in behaviour, memory and thought processes seen in people with a dementia, such as Alzheimer's disease, may be a direct result of the way the disease affects a particular area of the brain. An understanding of both the structure and function of various parts of the brain will help caregivers understand and better cope with the many changes dementia brings about.

Compiled by editorial staff

Dementia care and the brain

The brain, under normal, disease-free working conditions, is the seat of intelligence, interpreter of the senses, initiator of body movement, and controller of behaviour.

Lying in its bony shell and washed by protective fluid, the brain is the source of all the qualities that define our humanity. It has been called the "crown jewel of the human body."

More has been learned about the brain in the last ten years than in all previous centuries because of the accelerating pace of research in the neurological and behavioural sciences, and the development of new research methods - primarily the imaging techniques: angiography, MRIs, fMRIs, CAT scans, etc.

Structure and function

An understanding of how a healthy brain works is necessary in order to understand what happens when the brain is diseased or dysfunctional, as in a dementia. Often, in neurological disorders, the problem is not in the

structure of the brain, but in how it functions. An understanding of both **structure and function** of the parts of the brain is therefore in order.

Dementias are neuro-degenerative conditions associated with a progressive loss of function of nerve cells or neurons. Alzheimer's disease (AD) is the most common of the approximately 70 known dementing disorders, and accounts for approximately two-thirds of all dementias.

Dementia gradually pervades most areas of the brain. The evolving pat-

tern of damage it causes can vary greatly between different individuals. As a result, each person with the disease may have a complex set of difficulties and experiences which are peculiar to them.

Anatomy

By looking at the functions of a normal brain, we can be aware of the deficits of the specific dementias.

For descriptive purposes, the brain can be divided into three main units:

Neurological Disorders

When the brain is healthy, it functions quickly and automatically. But when problems occur, the results can be devastating. It is estimated that, because of the various neurological diseases, one in five people suffer damage to the nervous system. Some of the major types of disorders include:

- Neurogenetic diseases (Huntington's disease, muscular dystrophy);
- Developmental disorders (cerebral palsy);
- Degenerative diseases of adult life (Parkinson's disease, Alzheimer's disease, Lewy body dementia);
- Metabolic diseases (Tay-Sachs disease, Gaucher's disease [rare]);
- Cerebrovascular diseases (stroke and vascular dementia);
- Central Nervous System trauma (spinal cord and head injury);
- Convulsive disorders (epilepsy);
- Infectious diseases (AIDS dementia);
- Brain tumors

hindbrain, midbrain and forebrain.

The hindbrain includes the upper part of the spinal cord, the brain stem, and a wrinkled ball of tissue called the **cerebellum**. These structures control the body's vital functions; i.e., respiration, blood pressure.

The cerebellum is responsible for learned movements. For example, playing the piano or hitting a ball requires functioning of the cerebellum.

Above the hindbrain lies the midbrain, which controls some reflex actions and is part of the circuitry responsible for voluntary movements. Both these areas are mainly concerned with basic life support functions.

In contrast, the forebrain is responsible for the majority of higher brain functions, such as memory and language. The most important parts of the forebrain are the **cerebrum**,

which is divided into two hemispheres; the **cerebral cortex**, and the structures hidden beneath - the inner brain, or **limbic system**. (See page 8)

The cerebral cortex

The cerebrum is the largest and most developed part of the brain. The thin shell covering the surface of the cerebrum is the cortex (**Latin for bark**). This thin layer (as thick as two computer disks) is tightly crumpled and folded to increase surface area. As a result, the cortex contains an astonishing ten thousand million brain cells (the grey matter).

Beneath this densely packed cortex lie bundles of fibres (the white matter) which transport information around the cortex and to other regions of the brain.

The lobes

The cerebrum can be further divided into four sections or lobes named after the overlying bones of the skull. Each lobe has specific functions:

1. **The occipital lobe**, located at the back of the brain, primarily deals with visual information.
2. **The parietal lobe** lies in the upper rear portion of the brain and is concerned with information about spatial relationships and structure.
3. **The temporal lobe** lies beneath the parietal lobe and is involved in memory functions.
4. **The frontal lobe** is the executive and management centre.

We now need to know how these four lobes contribute to our experience of the world, starting at the back - the occipital lobe.

The Hemispheres

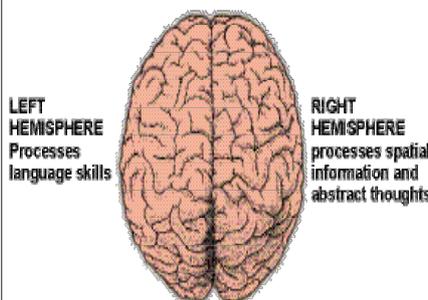
The **cerebrum** is split into two **hemispheres** by a deep fissure. Despite the split, the two hemispheres communicate with each other through a thick tract of nerve fibres (**corpus callosum**) that lies at the base of this fissure.

Although the two hemispheres appear to be mirror images of each other, they are different. For instance, the ability to form words seems to lie primarily in the left hemisphere, while the right controls many abstract reasoning skills.

For some unknown reason, nearly all of the signals from the brain to the rest of the body, and vice-versa, cross over on their way to and from the brain. This means that the right cerebral hemisphere primarily controls the left side of the body and the left hemisphere primarily controls the right side. When one side of the brain is damaged, the opposite side of the body is affected.

Certain principles govern the organisation of the brain. The division between the hemispheres signals one of these principles - **lateralisation**.

Lateralisation literally means that



A longitudinal fissure divides the brain into two halves or hemispheres that communicate with each other

some functions are best performed by the left side of the brain, while other functions are mainly supported by the right side. Not all functions are equally shared. For example, language tends to be a function of the dominant hemisphere (usually the left side).

Information is also represented in a map-like ordered fashion within the brain. For example, the **motor cortex**, which sends movement instructions to the muscles, is organized so that parts of the body which are physically close (i.e., hand and wrist) are controlled by groups of brain cells in proximity. ■

Occipital lobes

Although the eyes are the source of visual information about the world around us, it is actually the brain which does most of the hard work.

The eyes convert sensory information about light into electrical impulses, which are passed to the brain for interpretation. This division of labour means we have to distinguish between visual acuity and perception.

Visual acuity - the ability to see small objects - is achieved by the workings of the eye itself.

Perception is achieved by the occipital and parietal lobes, with information about colour, shape and movement being processed separately by the occipital lobe before it is passed on to the parietal lobe for combination into a complete three dimensional picture of the world. As a result, someone may have difficulty seeing what an object is, despite both eyes being in perfect shape.

The occipital lobes help in the

visual recognition of shapes (associated with the right occipital lobe), and colours (with left occipital lobe).

Parietal lobes

The functions of the parietal lobe are somewhat more diverse, with a significant difference between the dominant and non-dominant sides.

• Dominant parietal lobe

The dominant parietal lobe (usually the left half) can be thought of as being concerned with things we have to put together into an order or structure. Tasks such as reading and writing (which require putting letters and words together) and calculation (which involves ordering and combining numbers) are critically dependent on the dominant parietal lobe. This side of the parietal lobe has also been heavily implicated in a condition known as *apraxia* (impairment of learned purposeful movements even though there is no sensory or motor impairment).

Dressing apraxia is the most common, and reflects, not only a lack of co-ordination, but an actual forgetting of movements required to achieve a goal (i.e., fastening a button).

As if to compound such problems, the dominant parietal lobe is also responsible for our body sense; that is, knowing our left from our right and sensing where one limb is in relation to the rest of our body.

• Non-dominant parietal lobe

In contrast, the non-dominant parietal lobe (usually the right half) could be thought of as our “3D centre.” As mentioned previously, this part of the parietal lobe receives visual information from the occipital lobe. The function of the non-dominant parietal lobe is to combine such information into a 3D representation of the object being viewed. Damage to this area leads to

a symptom known as *visual agnosia*, an inability to recognize objects, faces or surroundings.

Because visual information is processed separately from other modes of sensation, it is possible for individuals to fail to recognize a familiar face by sight, but to know who they are once they speak.

The non-dominant parietal lobe also contributes to our understanding of space, but in a different way from its dominant counterpart. While the dominant parietal lobe deals with our body sense or personal space, the non-dominant portion helps us locate objects in external space and to calculate the location of objects relative to one another and ourselves; (for example, when we are reaching to pick up a dinner fork or cup of tea).

The temporal lobe

The temporal lobes deal primarily with memory functions. The dominant temporal lobe is specialized for verbal (word-based) memory and the names of objects. We rely upon our non-dominant temporal lobe for our memory of visual (non-verbal) material; i.e., faces, scenes etc.

The frontal lobe

The frontal lobe contains several parts which all act together to form our executive or management centre.

The lateral or outer surfaces of the frontal lobe appear to be critical for organizing and planning our actions and learning new tasks.

In learning to drive, for example, these brain areas help us put together a very complex sequence of movements, which at first seem difficult and clumsy, but gradually become more smooth and automatic.

For someone with damage to the lateral or outer surface area of the frontal lobe, it is like being a learner all over again with many multi-stage tasks such as riding a bicycle, pour-

ing and drinking a glass of milk, or cooking a meal, etc., becoming very difficult because the pattern or plan of action has been lost.

Perseveration

Damage to these lateral areas can also cause people to get stuck on what they are doing (referred to as *perseveration*).

As the frontal lobe interacts with many other brain areas, especially the temporal lobe, this perseveration may take the form of repeating the same word over and over again, or repeating an action until stopped, like banging a table.

Motivation

Moving towards the division between the two hemispheres, the middle portion of the frontal lobe generates our motivation and general impetus. If this part of the brain is affected, people can lose their “get-up-and-go”, becoming lethargic and reluctant to get out of bed or perform a particular activity. It is important to realize that, what might be perceived as slovenliness by some, could be a direct consequence of the loss of cells or cell connections in this brain area.

Regulating behaviour

The regulation of our behaviour appears to be governed by a third area of the frontal lobes, the *orbito-basal* area, located in the curvature at the very front of the brain. In healthy people this part of the brain helps to monitor, control and moderate our behaviour, as in preventing (*inhibiting*) us from saying something rude when someone has annoyed us.

To help one understand this idea of a failure of inhibition, we may look at some peculiar aspects of our own healthy behaviour. For example, we may pull a light cord on entering a room despite the fact the light is already on. In doing this, we fail to inhibit or break out of a programmed pattern of behaviour. In Alzheimer’s,

this situation is exacerbated and can lead to swearing or other behaviour which the individual would have previously deemed inappropriate.

The cerebellum

Located behind the brain stem, the cerebellum helps co-ordinate movement (balance and muscle co-ordination, i.e., talking, running, dancing, playing a musical instrument).

It plays a prominent role in motor and non-motor learning. Motor learning includes learning of movement sequences necessary to perform certain motor tasks such as the sequential movements necessary to ride a bicycle; non-motor learning refers to activities not requiring the learning of movement sequences.

The cerebellum also plays a prominent role in cognition, including planning of daily activities, speed of information processing, memory, and other cognitive processes.

Because speech involves the co-ordination of multiple muscles of the chest, mouth, throat and tongue, damage to the cerebellum will result in speech abnormalities, i.e., slurred speech. Damage also results in disorders in cognition, including the timing and planning of activities.

To ensure individualized, strength-focused and knowledge-based care for residents, it is important that caregivers discover ways to understand the meaning of their behaviours. One method is through the use and understanding of the cognitive screening tests such as the Mini Mental State Exam, as well as the modified version (3MS), among others.

An equally important source of information is the results from brain scans (CT, MRI, SPECT). This information provides insight into the functioning of the brain. By knowing the area of the brain involved in a neurodegenerative disorder, a stroke or head injury, we will be better prepared to understand behaviours.

The limbic system

Deep within the centre of the brain, sitting atop the spinal column is a walnut-sized area with a number of structures, all of which are critical for human behaviour and survival. These structures, part of the *limbic system*, are the gatekeepers between the spinal cord and the cerebral hemispheres. They not only determine our emotional state, but modify our perceptions and responses in relation to that emotional state, and allow us to initiate and regulate body functions and movements without thinking (autonomic nervous system). Like the lobes in the cerebral hemispheres, the structures in the limbic system come in pairs: each is duplicated in the opposite half of the brain.

The *hypothalamus*, about the size of a pearl, directs a multitude of important functions. It wakes you up in the morning, and gets the adrenaline flowing during a test or a competition. The hypothalamus is also an important emotional centre, controlling the *hormones* that make us feel exhilarated, angry, or unhappy.

Near the hypothalamus lies the *thalamus*, a major clearing house for information going to and from the spinal cord and the cerebrum.

An arching tract of nerve cells leads from the hypothalamus and the thalamus to the *hippocampus*. This tiny nub acts as a memory indexer, sending memories out to the appropriate part of the cerebral hemisphere for long-term storage and retrieval when necessary.

The *basal ganglia* are clusters of nerve cells surrounding the thalamus. They are responsible for initiating and integrating movements. Parkinson's disease, which results in tremors, rigidity, and a stiff, shuffling gait, is a disease of nerve cells that lead into the basal ganglia.

The following list of functions is far from complete since, every day, researchers are discovering new connections associated with the limbic system:

- sets the emotional tone of the mind;
- stores highly-charged emotional memories;
- modulates motivation;
- controls appetite and sleep cycles;
- directly processes the sense of smell;
- modulates libido.

The structures of the limbic system are closely aligned to the frontal and temporal lobes. The *amygdala* has been found to play a strong role in aggression by tying a certain image (from the frontal lobe area) with the "feeling" it has about the image.

The amygdala tends to be "over reactive" in people with Alzheimer's disease. The shrinking of the hippocampus and the deficit of the neurotransmitter acetylcholine has been implicated, especially in Alzheimer's disease.

In proximity to the amygdala and hippocampus is the olfactory area; this explains the impact that smell has on our emotions and memories.

The neurotransmitters dopamine, serotonin, and norepinephrine are awash in the limbic area of the normal brain. ■

If we can combine all this knowledge with the ever increasing technology which permits us to see how a brain affected by dementia changes, we may be able to more successfully predict, treat and manage the various symptoms associated. ■

Bibliography

• Drubach, D., *The Brain Explained*; Prentice Hall Health, Upper Saddle River,

New Jersey; 2000.

• Gilman, S. and Newman, S.W., *Essentials of Clinical Neuroanatomy and Neurophysiology*; 9th ed.; F.A. Davis Company, Philadelphia, PA; 1996.

• Lauterbach, E.C., *Psychiatric management principles in neurological diseases* (Chapter 1; p.1-40); in *Psychiatric Management in Neurological Diseases* (E.C. Lauterbach, editor); American Psychiatric Press, Inc., Washington, D.C.; 2000.

Reminiscence technique calms agitated AD sufferers

Researchers in Australia have improved upon a technique that helps dementia sufferers. The treatment, called "Simulated Presence Therapy," involves a close relative recounting, on a tape cassette, an event or memory from the resident's past that is likely to have a calming effect on agitated sufferers of Alzheimer's or other dementias.

Topics such as descriptions of the childhood home, friends, activities, favourite nursery rhymes and how the resident met his/her spouse are possible events that can be taped and recounted.

Tapes are played to the resident during times identified as periods of likely agitation or distress.

This "simulated therapy" is only suitable for residents with certain dementias (i.e., Alzheimer's) and those with intact hearing and some verbal ability.

Behaviours such as screaming, disturbing and aggression are said to be extremely amenable to this therapy, based on the well-accepted belief that the earliest memories of Alzheimer's sufferers are the last ones to fade away.

Because of short term memory problems associated with AD and other dementias, the tape is new every time it is heard by the patient.

(Source: *Medical Post*, February, 2002)

The cerebrum, or outer part of the brain, is the largest in area. Its folds, fissures and patterns outline and contain the functional lobes: frontal, parietal, occipital and temporal - which are named after the overlying bones of the skull. The cerebellum is located near the brainstem.

Searching for the key to weight loss in Alzheimer's disease

Patients with Alzheimer's disease tend to be thinner and weigh less than patients suffering other types of dementias.

In normal aging, fat is redistributed from the peripheral areas to the truncal (torso) areas of the body. In Alzheimer's, however, this redistribution is reversed.

This difference in body composition and a decrease in body weight by Alzheimer's patients has not been explained by nutritional intake, or malabsorption (Renvall, 1993).

Because patients with Alzheimer's disease show a significantly greater loss in weight than patients with other forms of dementia, nutrition has been considered the possible culprit.

For example, researchers noted that Alzheimer's patients had a number of nutritional deficiencies: vitamins A and B₁₂, folate, thiamine, vitamin E, beta-carotene, and choline. Impairments, ranging from memory loss to dementia, may be produced by these vitamin deficiencies (Halliwell, 1997).

There is also evidence of a changed glucose metabolism in patients with Alzheimer's (Bucht, 1990); and Claggett (1989) stated that low blood sugar values are found in patients with AD due to a changed carbohydrate metabolism. Carbohydrate metabolism is important in producing acetylcholine, which has been implicated in Alzheimer's symptoms (Bucht, 1990).

Further, brain cells do not store nutrients. Thus, a reduction in glucose in the blood flowing to the brain, as occurs in AD, would leave brain cells unable to properly function, thereby causing cognitive difficulties. Whether these decreased levels of nutrients in some way cause, or result from, Alzheimer's disease, is unknown. If a relationship can be established, it would argue for a nutritional link in Alzheimer's disease.

References

- Bucht, G. and Sandman, P., Nutritional aspects of dementia, especially Alzheimer's disease, *Age Aging*; 19(4); p.32-36- 1990.
- Claggett, M., Nutrition factors relevant to Alzheimer's disease, *Perspective in Practice*; 89(3); p.392-396: 1989.
- Halliwell, B., Antioxidants and human disease: a general introduction, *Nutrition Review*; 55(1); p.544-552; 1997.
- Renvall, M., Spindler, A., et al., Body composition of patients with Alzheimer's disease, *Journal of the American Dietetics Association*; 93(1); p.47-52; 1993.

Neurological basis of memory

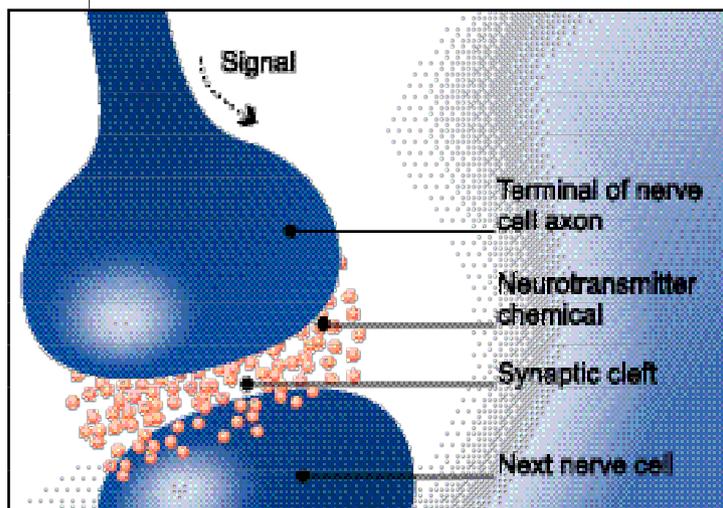
Significant progress has been made over the past few decades in our understanding of memory function (Squire and Zola, 1997). Using a variety of methods in both animals and humans, the regions of the brain that are important for memory have been mapped.

Interestingly, different types of memory seem to be supported by different brain regions. For example, short-term memory is typically more dependent on the frontal lobes, whereas long-term memory is more dependent on a portion of the temporal lobe called the hippocampus. Likewise, different types of memory are dependent on particular neurotransmitter systems. For example, short-term memory is more dependent on the neurotransmitter dopamine, whereas long-term memory is more dependent on the neurotransmitters acetylcholine and glutamate.

These advances in our knowledge of the neural basis of memory can have direct implications on treatment of individuals with memory disorders. For example, at present the only approved medications for AD are cholinesterase inhibitors, which increase the amount of acetylcholine in the brain. These medications improve memory function in patients with Alzheimer's disease. ■

Reference: Squire L., Zola S., Amnesia, memory, and brain systems: *Philosophical Transactions of the Royal Society of London*; 352; p.1663-1673; 1997.

How drug therapies work in Alzheimer's disease



The synapse

Under normal conditions, the electrical nerve impulse travels down the axon of the neuron to a synaptic knob or vesicle. This impulse triggers the vesicle to release neurotransmitters that cross the synaptic cleft to receptors in the target or post-synaptic neuron. A response or excitation occurs and the connection is continued.

The primary functional unit of the brain and nervous system is the neuron. All sensations, movements, thoughts, memories, and feelings are the result of signals that pass through the tens of millions of neurons. Neurons transmit signals to other neurons through a process called the **synapse**.

We have learned a great deal about neurons by studying the synapse - the place or process where chemical signals pass from one neuron to another. A single neuron (**the presynaptic neuron**) may form thousands of synaptic connections with adjacent neurons or cells.

Neurotransmitters

During a synapse, the two neurons do not come directly into contact. Their surface membranes are separated by a gap called the **synaptic cleft**. When the electrical signal from the presynaptic neuron, travelling along its **axon**, reaches its terminus or **synaptic knob**, it cannot bridge the cleft directly. Instead, the electrical impulse stimulates tiny sacs or vesicles containing **chemical neurotransmitters**. The sacs release the neurotransmitters into the synaptic cleft, crossing the cleft and attaching to receptors on the neighbouring neuron (**the postsynaptic neuron**). These receptors can change the properties of the receiving cell; and if the receiving cell is also a neuron, the signal continues to the next cell. Signals can be transmitted across a synapse in one direction only - from the

presynaptic neuron or cell, to the postsynaptic neuron or cell.

Depending on their function, there are a number of different types of neurotransmitters that can be involved in a synapse. **Acetylcholine**, for example, is called an excitatory neurotransmitter because it makes neurons more excitable.

Acetylcholine governs muscle contractions, causes glands to secrete hormones, controls heart beat, and is present at numerous other sites in the brain and nervous system where it is involved in transmitting signals between neurons.

Malfunctioning of neurons that release the acetylcholine neurotransmitters is of major significance in Alzheimer's disease. Alzheimer's disease initially affects memory formation - which is known to be associated with a shortage of acetylcholine. The box below lists several other important neurotransmitters and their functions.

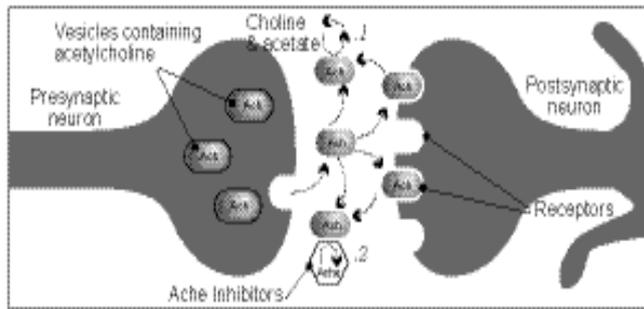
The cholinergic system

Due to breakthroughs in Alzheimer's research in the 1990s, acetylcholine has received a great deal of attention. The actions or functions of acetylcholine are called **cholinergic** actions (of the cholinergic system). Of the many neurotransmitter systems altered by Alzheimer's disease, the cholinergic system appears to be one of the most vulnerable.

Under normal circumstances, acetylcholine (*Ach*) is broken

Neurotransmitters and their functions

- **Glutamate** is an excitatory neurotransmitter. During learning and when using memory, high levels of glutamate are released into the synaptic cleft. It is heavily implicated in Alzheimer's disease, and an important consideration in new drug therapies.
- **Serotonin**, an inhibitory neurotransmitter, is involved in many functions throughout the body; some of the primary ones include controlling states of consciousness (sleep), mood states and sensory perception. It functions to constrict blood vessels and regulate temperature.
- **GABA (gamma-aminobutyric acid)**, also an inhibitory neurotransmitter because it tends to make cells less excitable. Drugs that increase GABA levels are used to treat seizures and tremors in Huntington's disease.
- **Dopamine** is an inhibitory neurotransmitter involved in mood and the control of complex movements. The loss of dopamine activity in some portions of the brain leads to the muscular rigidity of Parkinson's disease.
- **Norepinephrine**, like some other neurotransmitters, acts as a hormone as well as a neurotransmitter (catecholamines). Also called noradrenaline, norepinephrine is important in stimulating nerve pathways that control or maintain heart beat, blood flow, and response to stress. ■



(1) The neurotransmitter acetylcholine (ACh) is broken down by the action of the acetylcholinesterase enzyme (AChE). Acetylcholinesterase-inhibiting drugs block this action (2), thereby increasing the amount and duration of excitation of the synaptic acetylcholine.

down to choline and acetate by an enzyme called **acetylcholinesterase (AChE)**. When too much of this enzyme is present, the amount of acetylcholine is reduced. Thus, there may not be enough of the acetylcholine to make the synaptic connection and excite the next (postsynaptic) neuron into action. Excessive amounts of acetylcholinesterase enzyme are to be found in Alzheimer's disease. To make more acetylcholine available, drugs that inhibit or block the action of the acetylcholinesterase enzyme are used.

Cholinesterase inhibitors

Discoveries over the past decade have provided us with drugs called **cholinesterase inhibitors**. These drugs have the capacity to inhibit the catabolic or break down action of *AChE*. This inhibiting of *AChE* increases the amount of acetylcholine for synaptic connections to be made to the next neuron and exciting other neurons into action and the producing of

As Alzheimer's disease progresses, however, and fewer and fewer acetylcholine neurotransmitters remain intact and functional, the effects of the drug diminish.

Significant impact

Although not indicated for the more advanced cases of Alzheimer's disease, these drugs have had a significant impact for caregivers. They have delayed placement in long-term care facilities that can be measured in years. Also, many residents, although placed in nursing homes for reasons other than a dementia, do contract Alzheimer's.

Early diagnosis and treatment with the acetylcholinesterase-inhibiting drugs would extend and enhance quality of life - not to overlook the saved resources in AD care that would have been required at an earlier stage. The slightest of improvements mean a great deal to individuals with AD, as well as to family members and nursing home caregivers. ■

Alzheimer's drug therapy using a different neurotransmitter system

In Canada and the United States, the only drugs currently approved for treating Alzheimer's disease are the anti-cholinesterase inhibitors. These drugs do not attack the disease, but assist the brain so it can compensate for the loss of neurons that communicate via the neurotransmitter, acetylcholine. The action of the drug prevents an enzyme from breaking down acetylcholine, thereby slowing the progress of the disease, although the improvement is not dramatic, and the efficacy of the drug is limited to mild and moderate AD.

Middle and late stage AD drug therapy

In Europe, particularly Germany and Britain, the standard prescription for many people in middle and later stages of Alzheimer's is a drug called *memantine*, which works on a different neurotransmitter. This drug blocks the action of the neurotransmitter, **glutamate**, which is overproduced in the brains of people with AD and can cause the death of neurons by over-exciting them. At the present time, in North America,

memantine is in phase III clinical trials (human trials).

During the 8th International Conference on Alzheimer's Disease and Related Disorders held last July in Stockholm, Sweden, the results of a clinical trial on *memantine* were presented by researchers from New York University.

This year-long trial included 252 people with advanced Alzheimer's disease. It showed that patients who received *memantine* maintained more cognitive abilities and were less impaired on ADLs than patients who took placebos.

Administering drugs in combination

The benefits, similar to those with the anticholinesterase inhibitors, were not dramatic. Nevertheless, a speaker at the Conference, Dr. Ezio Giacobini of the University of Geneva in Switzerland, pointed out that, in spite of the modest improvements provided by the two drug therapies, he saw no reason why the two therapies could not be given in combination to possibly enhance the benefits. ■

Understanding interventions in dementia care

A number of concepts pertaining to dementia behaviour must be understood if interventions are to meet with success. These concepts are, for the most part, common to all dementias.

(A preamble to the following presentation on ‘cognitive screening and interventions’)

• Reality orientation

A person with dementia is unable to retrieve past memories at will. The person with short-term memory deficit is unable to remember after ten minutes. This means that the person with dementia has only NOW. This also means that they will constantly be looking around, will become bored if there is no stimulation, and may even walk away. It also means that they are highly sensitive to the cues that care staff and visitors are emitting. Thus, a shift change at a facility can be a difficult time since, if a resident with dementia feels that he/she is only visiting (why else would he/she be in this place?), when the staff say good-bye and leave, the resident will take the hint and leave as well.

In past caregiving theory, “reality orientation” meant bringing people into the “now” or into the present. For example, if a resident asked for his/her mother, he/she would be told that she was dead. Theorists have since learned that, with dementia, this approach doesn’t help. All it does is introduce more distress.

Today, when we use “reality orientation,” we use what they can see, hear and feel right now in order to make sense in their world. For example, the resident who gets up at 3 a.m. can be shown that it’s dark out and still too early to get up. Or, the resident who needs to go to work can be told it’s Sunday or a holiday, or that it’s snowing out and the roads are blocked.

• Validation

Validation is the art of assisting the person to feel that the problem, and the responses to the problem, are acknowledged and understood” (Robins, 2000).

Naomi Feil, in a recent book - “*The Validation Breakthrough*” (Feil, 2002) - observed that many old-old, who have ignored or denied the need for important life tasks when they were younger, enter a period of their lives when they feel the need to resolve unfinished business in order to die in peace. Her principles of validation blend well with the knowledge that, as the brain atrophies in people with dementia, they regress in time and will often relive past issues.

The caregiver, in order to understand validation

theory, will have to appreciate and accept the following fundamental and humanistic beliefs and values:

- All people are unique and valuable - no matter how disoriented they are.
- There is a reason behind the behaviour of old-old disoriented people, just as there is a reason behind the behaviours in those with dementia.
- Behaviour in the old-old is not merely a function of anatomic changes in the brain, but reflects a combination of physical, social and psychological changes that take place over a life-span. For people with dementia, their behaviour is often the reliving of past issues.
- Often, when painful feelings are expressed by a resident with dementia, if validated (i.e., acknowledged and understood), these painful feelings will diminish.
- On the other hand, painful feelings that are ignored or suppressed, will gain strength and become “toxic.”
- Because a person with dementia is no longer capable of remembering new information, it is impossible for this person to change their behaviour.
- Empathy is the art (or gift) of having an awareness of, or insight into, the feelings, emotions, and behaviour of another person and their meaning and significance. Possessing this art as it relates to dementia care builds trust, reduces anxiety and restores dignity.

Establishing validation, can be accomplished at a number of levels, the most basic being to simply listen and observe. Consider the following scenario:

Betty would not come into the dining room for lunch. No matter what the caregiver said, Betty would continue pacing, and even increased her speed of pacing.

A second caregiver smiled and joined Betty in her pacing. The caregiver then stated that she noticed that Betty seemed upset, and started to slow the speed of the pacing. Betty then said, “Where’s my husband?”

Betty’s husband had been dead for over ten years. The caregiver recognized that reality orientation would not help Betty come for lunch. She then said to Betty:

Where do you think he is? It’s lunch time.”

Betty stopped pacing and, looking at the caregiver, said: “He’s working.”

With a shrug, the caregiver said: “It’s lunchtime, so let’s

have lunch together.” The caregiver was then able to take Betty to the dining room, help her find a place to sit and Betty started eating.

Once Betty’s feelings had been identified and she was able to make sense of them, she was relieved of her stress and able to follow staff directions. Had Betty’s feelings (anxieties) not been understood and acknowledged (validated), they would probably have intensified.

The caregiver enters the resident’s world by trying to identify the feelings being expressed. When a resident feels accepted, no matter what he/she is trying to express, and acknowledged for whatever he/she is feeling, anxieties tend to defuse themselves.

• **Mirroring**

Mirroring is the technique of connecting with a person by copying or doing what the person is doing. In the previous scenario, the caregiver paced with the resident. This technique involves joining in the actions, sounds or even breathing rate of the resident. Common sense dictates that certain actions not be mirrored.

After a few minutes of sharing (or mirroring) the other’s action, the staff member can identify his/her own feelings and observe the response of the resident in order to determine if it is what that resident is feeling. Often, once the feeling has been correctly identified, the action will decrease. If not accurately identified, the action tends to increase as the anxiety increases.

Once the resident’s feeling has been identified, he/she can be acknowledged for feeling it, and that such feelings are okay. The caregiver can then use the techniques of reminiscing, touch (hugging the resident), or “real” time distracting (see **reality orientation**).

As the caregiver does what the resident is doing, such as pacing, or other reasonable behaviour, the caregiver can slow down or enter another area and the resident will follow. Once the “connection” has been made, the resident will mirror what the caregiver is doing as a form of compliment.

• **Doing a “U-turn”**

Because of changes in the functioning of their brain, people with dementia are often unable to physically turn around. You will see them in the hall facing a wall, and they will stay there until rescued.

This inability to turn around is an important consideration when a caregiver wants to turn a resident in another direction. Walk with them, distract them ver-

bally, and walk in a semi-circle or arc; they can re-enter the door they left through because of short term memory loss. Do not try to physically turn them around, as they have lost the physical ability to do so. ■

References

- Robins, C.J., Ivanoff, A.M. and Linehan, M.M., “Dialectical Behaviour Therapy (Ch. 21); W.J. Livesley, editor. Handbook of Personality Disorders: Theory, Research and Treatment; Guilford Press, New York, NY; p.437-459; 2001.
- Feil, Naomi, The Validation Breakthrough (2nd edition). Health Professional Press; Baltimore, MD; 2002.

The preceding has been compiled from notes and excerpts from previous articles submitted by Irene Barnes. See page 25 for biography.

Agitation and unmet needs

Agitation is another well-known and troublesome behaviour that has been misconstrued by caregivers, according to psychologist Jiska Cohen-Mansfield of the Hebrew Home of Washington in Rockville, Maryland.

AD patients are known for their agitated behaviours: screaming, pleading, pacing, disrobing, rummaging, hitting, kicking and biting. Not too long ago such behaviour was considered a manifestation of psychosis, or nuisance behaviour that comes with dementia. The interventions were either psychotropic drugs, restraints or ignoring.

Cohen-Mansfield suspected, as many others have since, that these agitated behaviours were propelled by unmet needs. She had staff personnel observe AD patients around the clock, noting what circumstances triggered the agitated behaviours. It was noted that some patients would scream or moan when it was dark and they were alone.

Hypothesizing that this behaviour might be a reflection of their fear and loneliness, she implemented three interventions:

- (1) caregivers would visit one-on-one with the patient at the problem time;
- (2) play a video-tape of a family member talking to the patient, and
- (3) play music enjoyed in the past.

According to Cohen-Mansfield, all three interventions made a difference. As a group, the screaming and moaning decreased by approximately 50% when one-on-one interactions or the video-tape was the approach. The music tape reduced the agitation by one-third.

These one-on-one interactions appear to be one of the most fruitful interventions in Alzheimer’s care. ■

Edited and abridged, from *Science*, Vol. 282, Nov. 6; 1998.