DEMENTIA
Metamorphosis in Care

CLAIRE BIERNACKI
Day Services Manager
Older People's Services
Derbyshire Mental Health Services NHS Trust

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Anniversary Logo Design: Richard J. Pacifico

Library of Congress Cataloging-in-Publication Data

Biernacki, Claire.
Dementia : metamorphosis in care / by Claire Biernacki.
p. ; cm.
Includes bibliographical references and index.
ISBN-13: 978-0-470-01997-9 (pbk.: alk. paper)
1. Dementia. I. Title.
[DNLM: 1. Dementia. 2. Dementia – nursing. WM 220 B588d 2007]
RC521.B54 2007
616.8′3 – dc22

2006032098

A catalogue record for this book is available from the British Library

ISBN-13: 978-0-470-01997-9

Typeset by TechBooks, Delhi, India.
Printed and bound in Great Britain by TJ International Ltd, Padstow, Cornwall.

This book is printed on acid-free paper responsibly manufactured from sustainable forestry in which at least two trees are planted for each one used for paper production.

Dedication

I would like to thank Ruth, Caitlin and Sophie for their patience, support and encouragement throughout the completion of this book and express gratitude to those whose feedback and advice have been invaluable, in particular Sue O'Dea and Petrina Brown. I would also like to thank all those who have contributed to my education, in particular Jack, Olive, John, Phyllis, Mona, Kath and many, many others.

Contents

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Preface

The past 20 years have seen an explosion of research and anecdotal evidence which calls into question virtually everything previously presented as fact with regard to the experience of dementia and people who have dementia. In 1997, the publication of the late Professor Kitwood's seminal work *Dementia Reconsidered* seemed to galvanise the activity, not only of academics but also of front line practitioners in seeking to re-define the meaning of care for persons with dementia. The term 'Person Centred Care' has become synonymous with this movement toward improving dementia care.

In the decade following Kitwood's publication, research has investigated aspects of dementia that would have been considered ridiculous 20 years ago, particularly aspects concerning the psychosocial elements of dementia and the subjective experience of dementia. As the evidence mounts, what is repeatedly confirmed is that there is a desperate and urgent need to alter care practices. Unfortunately what is also evident is that in reality little, if any, tangible change has occurred, despite this wealth of evidence. Persons with dementia still routinely find themselves in situations where their needs are secondary to those of others, where their value as individuals and indeed as human beings is denied. As someone who has worked in dementia care for 20 years, I have experienced the excitement that the arrival of 'Person Centred Care' generated and the disappointment that, at the point of delivery, care services seem barely to have registered a ripple of positive change. The personal frustration of this continuing situation led me to seriously consider leaving dementia care altogether. Instead I wrote this book.

This book takes the reader on a journey beginning with the history of dementia care, through the maze of ever increasing evidence into areas where positive change is taking hold and where cause for optimism is high; and into dark areas where people with dementia are emotionally abandoned and a sense of futility prevails.

The book uncovers the evidence for positive dementia care in a way that enables practitioners to understand why change is necessary, reinforcing the importance of theory to practice and detailing the skills practitioners need to acquire, in order to become expert practitioners in the dementia care specialism.

What this book will show is that approaches to positively influence the experience of dementia are readily available and clearly achievable. More than that, this book will show that adopting evidence-based approaches to care will improve the work satisfaction of practitioners. The book leaves practitioners at a crossroads, at a point in the evolution of the dementia care specialism, where choices have to be made with regard to how practice develops. Do we continue as we have been, digesting the

evidence and changing the language of care but not really impacting on the quality of life of individuals in our care? Or do we accept that responsibility for initiating and supporting positive change belongs to each one of us who has chosen to spend their working lives caring for persons who have dementia? My sincere hope is that this book fosters a desire to adopt the latter approach.

1 Dementia in Context

INTRODUCTION

Chapter 1 provides an overview of dementia in the context of today's society, looking at what dementia is, the differential diagnoses and describing the main types of dementia together with some of the less common presentations, such as dementia in people with learning difficulties and in people of working age. This chapter considers how predicted changes in population are likely to impact on society and those living with dementia and considers dementia care from an historical perspective. The stigmatisation of people with mental health needs, including people with dementia, is a fact of our society that research finds to have altered little despite a move of care of these people into the community; this is described also in this chapter.

Many recently published texts on the care of the person with dementia deliberately avoid a definition and detailed description of signs, symptoms and types of dementia. This is understandable in an effort to de-medicalise care and to encourage the carer to look beyond the medical model, which is often seen as the antithesis of new models of care that encompass a person centred approach. I am including such a definition and description here for several reasons.

Firstly, throughout my years of clinical practice I have frequently witnessed carers accuse people with dementia of misbehaving, acting up and pretending they are unable to do something they could do only yesterday, and responding to this perceived misbehaviour with anger, and indeed punishment. This section of the book is about enabling carers to understand that the way in which the person with dementia behaves or communicates is directly affected by the damage that disease has caused to their brain. It is not behaviour designed to frustrate or anger the carer and responding to the behaviour with anger and admonishment is more than unhelpful; it is tantamount to abuse. It is now known that such a response on the part of carers is likely to result in much of the behaviour termed 'challenging' that people with dementia are labelled with. Interactions stemming from anger on the part of the carer have no place in the development of what is becoming the specialism of dementia care.

Secondly, knowledge of an individual's specific diagnosis, where it can be established, is vital in ensuring that correct information and advice are offered in a timely manner; for example, how the illness is likely to progress, what treatments are available, the likelihood of the disease being passed to children and other information the individual may seek.

Finally, while current evidence supports the view that interpersonal care based on a person centred model is the approach most likely to improve the quality of

day-to-day life for the person with dementia, it is medical science that will provide the cure as it has begun to provide treatments. Just as we need to discard aspects of the medical care model that are detrimental to the individual's well-being so we need to take care that we maintain awareness of medical developments that will enhance our practice.

WHAT IS DEMENTIA?

There are surely more definitions of dementia than there are forms of dementia (approximately one hundred), so giving a definitive description is difficult. However, the World Health Organisation (1992) seems to encompass aspects that most agree on when it describes dementia as:

> A syndrome due to disease of the brain, usually of a chronic or progressive nature, in which there is impairment of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language and judgment. Consciousness is not clouded. The cognitive impairments are commonly accompanied, and occasionally preceded, by deterioration in emotional control, social behaviour, or motivation.

Thus this impairment has repercussions for the ability of people to manage aspects of their daily lives to their previous standard or ability. Tasks taken for granted such as washing, dressing, carrying on with work, hobbies and relationships become increasingly difficult. If the person lives long enough with dementia it may well become impossible for him or her to undertake these tasks independently or to communicate needs in easily understood ways.

The process of dementia is that it is a progressive syndrome; symptoms become more marked and impact more and more on the individual's life, eventually pervading all areas. For the purposes of loosely describing this process, different stages can be identified and are described in Tables 1.1 to 1.3. Consideration is given to the effects of dementia on cognitive function and the impact on behaviour and the emotions the individual may express as a result of the experience of dementia. This categorising is not intended nor should it be used as a means of labelling individuals who have dementia. It is incorrect to assume that any individual will have all of the symptoms or experiences described in the order described, because of course it is not only the process of disease and accompanying dementia that must be taken into account. We must also consider that person's personality, life history, physical state and social relationships.

WHAT IS NOT DEMENTIA?

Most diseases causing dementia are at present incurable, for example Alzheimer's disease, vascular disease and Lewy body disease. However, there are causes of

Table 1.1. Early stage dementia

Cognitive symptoms	Behaviour	Emotional response
Mild impairment of short term memory Difficulty with language and reading Difficulty making decisions Difficulty concentrating Difficulty making judgements Hallucinations (rare)	Forgetting birthdays, anniversaries, appointments Losing things Getting lost while driving Forgetting peoples' names Looking to others to confirm correctness of words or actions or conversely rejecting assistance offered Confabulating – making up information in the absence of the ability to recall correct information	Worry Social embarrassment Fear of going mad Suspicion Anxiety Denial Frustration Irritability Depression Tearfulness Unconcern

Table 1.2. Middle stage dementia

Cognitive symptoms	Behaviour	Emotional response
Loss of short term memory Patchy long term recall Dysphasia Difficulty planning, sequencing and using judgement Disorientation to time, place or person	Unable to pursue hobbies Unable to drive safely Misidentifying family members Loss of friendships and social contacts Disturbed sleep Difficulty with daily activities – washing, dressing, preparing food, shopping Walking and getting lost Self-harm attempt (rare)	Withdrawal Depression Paranoia Agitation/anxiety Apathy Anger Frustration Incongruence/lability Aggression Bereavement reaction Acceptance Tearfulness Unconcern

Table 1.3. Late stage dementia

Cognitive symptoms	Behaviour	Emotional response
Loss of short and long term memory Loss of judgment, planning and sequencing skills Severe dysphasia or aphasia Dysphagia Loss of ability to respond to own needs or express needs in easily understood ways	Unable to anticipate or meet daily needs – washing, dressing Incontinence Constant walking Difficulty with eating, drinking and self-feeding Social withdrawal Rejection of assistance – violence Repetitive actions and words/sounds	Placidity Agitation Anger Aggression Anxiety and panic Depression Complete withdrawal from interaction with others

dementia or more specifically conditions that appear to mimic dementia that are open to treatment. These include nutritional deficiencies, particularly B12 and folate, alcohol or drug abuse (although prolonged abuse can lead to irreversible dementia), minor head injury, depression, brain tumours and infective disease. All of these conditions can result in symptoms that are associated with dementia. A close examination of the onset, progression, nature and consistency of symptoms may aid accurate diagnosis but misdiagnosis can occur.

In the 1970s it was optimistically proposed that anywhere between 10 and 40 % of dementias might be reversible. A recent updated meta-analysis undertaken by Clarfield (2003) puts that figure very much lower. Ensuring thorough investigation to rule out a treatable cause or to treat what is treatable, however, is vital. As Clarfield points out 'comorbidity should always be treated for its own sake and in the hope that cognitive decline may at least be delayed'. It is therefore important that we are able to recognise and identify conditions that present with symptoms similar to dementia syndrome to enable appropriate treatment to be provided and to avoid misdiagnosis. Depression is one differential diagnosis that needs serious and thorough investigation. Differentiating between dementia and depression can be extremely difficult. In cases where the individual has a history of depressive episodes this may more strongly indicate depression. Although it is unusual for a first time episode of depression to occur in people aged over 65 years (outside of bereavement) this should not be discounted. Differentiation is further complicated by the fact that depression can and often does present co-morbidly and there is increasing evidence that depression can be an early warning sign of dementia.

Symptoms seen in both depression and dementia include memory impairment, disturbances in patterns of sleep, loss of interest in things that have been previously enjoyed, poor motivation and slowed thought processes. The underlying reasons for these changes are different. In dementia the damage to the brain is the causative factor. For individuals with depression recent memory is impaired as the ability to pay attention to and concentrate on new information is adversely affected as a result of depression, making the task of storing new information difficult. A further complication for people with depression may be that they fear this memory loss *is* dementia, which adds to their depressive feelings. Working with people with this type of memory loss can be very effective; teaching memory techniques and improving levels of concentration may be enough to resolve the difficulty. For people with dementia this is not the case as impairment in ability to learn new techniques is usually prohibitive.

Disturbances in sleep behaviour can also occur in both depression and dementia, but people with depression may have difficulty falling to sleep or may wake very early, whereas individuals with dementia more commonly experience disturbance throughout the night, sometimes due to disorientation to time. Therefore investigating the nature of sleep disturbance is important.

Dissecting the strands of common symptoms in this manner can assist accurate diagnosis but it is vital to differentiate as depression can be successfully treated. Importantly where a definitive diagnosis cannot be made the depression should be treated whatever its origin is thought to be, as this will improve the life quality of the

individual if they prove to have dementia and support resolution of a true depressive illness.

A more accurate diagnosis of other conditions that produce symptoms similar to dementia can be slightly easier. A full screen of the individual's physical condition will highlight infectious illness which can cause an acute confusional state. It is also the case that acute confusion, as the term implies, has a sudden onset. Symptoms found in dementia can present in a matter of hours or days as the infection progresses; such onset is atypical in dementia. Again it is important to realise that where dementia exists an acute confusion caused by infection can also develop and again treatment to improve life quality and resolve acute symptoms is essential.

Nutritional deficiencies and alcohol and substance misuse may also be highlighted through a full physical screen and by taking a thorough history. While treatment of the former can be relatively easy to achieve, the abuse of alcohol and drugs, particularly for prolonged periods, is notoriously difficult to overcome. There is, however, an opportunity for those working with younger people who abuse such substances to include advice on dementia as a consequence of long term abuse in the hope that such health promotion may have a role in reducing the incidence of drug and alcohol related dementia in the future.

Where any individual presents with symptoms that are suggestive of dementia an important first step in diagnosis is to rule out any causes of symptoms that are not dementia. The treatable causes of dementia-like symptoms are certainly more straightforward than approaching the treatment of dementia, but it should be remembered a that definite diagnosis of dementia does not preclude the individual from suffering further illness that can potentially exacerbate symptoms and that when this occurs treatment is required. Therefore a person with dementia who exhibits an acute exacerbation of symptoms should have a physical screen to identify a physical health need that may be responsive to treatment, and thus it may be possible to treat acute symptoms and promote optimum life quality.

Evidence for Practice

What Is and What Is Not Dementia

Dementia is a progressive syndrome caused by diseases of the brain

Symptoms include problems with thinking, impairment of memory, orientation, comprehension, calculation and learning capacity, language and judgement difficulties and changes in behaviour

The behaviour and communication of people with dementia are affected by the damage the brain experiences as a result of dementia

Knowledge of the individual's diagnosis enables us to provide timely and appropriate information

It is vital to differentiate between dementia and treatable causes of dementia-like symptoms

Treatable causes of dementia-like symptoms include depression, nutritional deficiency, head trauma and alcohol abuse

Accurate diagnosis and the treatment of what is treatable is vital

Individuals with dementia may suffer from depression, infection or other illness that can exacerbate their symptoms, any change in behaviour requires investigation to rule out or treat these illnesses

ALZHEIMER'S DISEASE

Alzheimer's disease (AD) accounts for between 50 and 70 % of all dementias. It was first described by Alois Alzheimer in 1907. His case study portrayed the symptoms of a 51-year-old woman with a five-year history including the inability to care for herself and rejection of help, disorientation, failing memory and difficulty reading and writing. There was a gradual increase in severity of the symptoms and hallucinations also developed. The second case Alzheimer described was that of a 56-year-old man. Possibly as a result of Alzheimer's two cases AD was initially considered a rare form of pre-senile dementia.

In normal functioning the brain has two types of brain cell: the neuron and glial cell. Neurons are responsible for processing information and ensuring the brain works; glials protect and support neurons. Simply put, neurons form complex connections to complete brain tasks such as memory and language. Communication between neurons takes place at junctions termed synapses. Synapses pass information via chemical signals called neurotransmitters. Increased loss of one of these neurotransmitters, acetylcholine, is strongly associated with severity of AD. The damage caused by the disease process results in the brain becoming increasingly ineffective at passing messages essential for language, controlling movement and solving problems.

Why these changes happen and why some people but not others develop AD is not yet understood. It is thought probable that a combination of genetic propensity and biochemical, environmental and immune processes, all of which are being investigated, may prove to have a part to play. A very small number of people will inherit AD genetically, where a parent has the mutated gene a child will have a 50 % chance of developing the disease. Onset of the disease in this instance is generally between the ages of 40 and 50. As with all genetically inherited diseases it is important that those at risk are given the opportunity of counselling to enable them to make informed life choices, including whether or not to have children, to consider how to plan for the possible eventuality of their own ill-health and to support them in living with the prospect of developing such a life changing disease.

AD generally has a slow onset. Initially people may encounter difficulty recalling recent events, people's names, telephone numbers or where they put things. Memories

from childhood or early adult life are usually recalled with little trouble. This can be puzzling as logic might suggest that what happened yesterday should be fresher in the mind than what happened when you were, for example, six years old. Although this memory difficulty may cause some concern it is often put down to the process of growing old. Health promotion that encourages people with memory difficulties to seek advice early will enhance the possibility of early diagnosis and treatment and provide reassurance where there is no cause for concern.

As the disease progresses symptoms of dementia become more pronounced, word finding difficulties become more common and memory impairment more serious. Appointments may be missed, medication overlooked and minor accidents can occur. At this stage it is difficult to rationalise what is happening as part of the ageing process.

At present there is no cure for AD but there are drugs that are licensed and were initially recommended by the National Institute for Clinical Excellence (2001) for the treatment of mild and moderate AD in the UK. These drugs, known by the group name acetylcholinesterase inhibitors, include donepzil, rivastigmine and galantamine and are discussed further in Chapters 2 and 8. While these drugs do not represent a cure for AD they do represent hope, a commodity in such short supply in dementia care that it has limited development of the specialty and consigned people with dementia to a bleak future. The hope presented is the hope that something rather than nothing can be done about this disease and the hope that the advancement of such drugs is the first step on a speedy journey to realistic cure or treatment to reverse the course of the disease.

Evidence for Practice

Alzheimer's Disease

AD accounts for 50–70 % of all cases of dementia

Seeking early diagnosis is advisable

Drug treatments in the form of acetylcholinesterase inhibitors are the first line treatment recommended for use in AD

In a minority of cases AD can be genetically inherited

Genetic counselling must be available for individuals where genetic inheritance is a possibility

VASCULAR DEMENTIA

There is disagreement in the literature as to whether vascular dementia is the second or third most common cause of dementia. Some experts suggest it may indeed be the most common in people over the age of 85. However, vascular dementia accounts for approximately 20 to 25 % of all dementias. The Alzheimer's Society

(www.alzheimers.org.uk) reports that of the 750,000 people in the UK living with dementia one-quarter has vascular dementia.

Vascular dementia occurs when the blood supply to parts of the brain is restricted or blocked and the brain is deprived of oxygen; cells in the brain then die and symptoms of dementia result. Stroke or a series of strokes are common causes of vascular dementia. Indeed, stroke increases the risk of dementia by more than ninefold. A series of ministrokes leading to an accumulation of brain cell death and symptoms of dementia is termed multi-infarct dementia.

The risk factors for vascular dementia are similar to those for stroke disease and include hypertension, raised blood lipids, diabetes mellitus and smoking. As each of these risk factors is, for many people, linked to lifestyle choices, health promotion for the younger generation may impact on future numbers developing vascular dementia. Equally, the ability of medical science to treat stroke early may mean that greater numbers of people survive strokes but will live with stroke related dementia.

While education on risk factors may help reduce future numbers of sufferers it may also be possible to reduce the risk of further damage or multiple infarcts in those with an existing condition by advising and enabling them to choose healthier lifestyle options. Treatment with antihypertensives to control blood pressure and statins to lower blood lipids may also play a role. Amar and Wilcock (1996) report that 'evidence exists that controlling vascular risks such as hypertension and diabetes and using an antiplatelet drug can improve cognitive functioning'. Thus encouraging people with vascular disease to stop smoking and reduce fat in their diet and take prescribed drug treatments can have a positive impact on the progress of vascular dementia.

Evidence for Practice

Vascular Dementia

Vascular dementia accounts for at least 20 % of all dementias

Vascular dementia results from restricted blood flow to the brain and risk factors are similar to those for stroke disease

Offering health promotion advice on diet, alcohol consumption and smoking may reduce the risk of younger people developing vascular dementia

Choice of healthier lifestyle options by someone with existing vascular dementia may reduce the risk of progression of dementia

Although there is currently no treatment for vascular dementia, treatment to reduce risk of further vascular damage such as antiplatelets, antihypertensives and statins may help

Further strokes or ministrokes (transient ischaemic attacks) can occur and may result in further impairment

LEWY BODY DISEASE

Lewy body disease (LBD), also referred to as Lewy body dementia, diffuse Lewy body disease or senile dementia of Lewy body type, is characterised by fluctuating levels of alertness and cognition, hallucinations (most commonly visual), Parkinson's symptoms and the presence of dementia symptoms. LBD shares characteristics with both AD and Parkinson's disease and this can make accurate diagnosis difficult. The Parkinsonian symptoms commonly experienced are shuffling gait and falls. Fluctuation in ability, particularly cognitive ability, is not unusual in dementia, but with LBD this fluctuation can be from hour to hour and the difference between the best performance ability and the worst is more marked than in other dementias. It is vital that carers acknowledge this fluctuating ability and respond appropriately. For example, a person who was able to wash and dress independently in the morning may not be able to undress and get ready for bed in the evening; this is a result of the person having LBD not because he is awkward or seeking to annoy the carer by feigning inability or through laziness.

Temporary disturbances in consciousness are a feature of LBD, where the individual may be unresponsive for several minutes; such episodes can be confused with transient ischaemic attacks. Hallucinations often take the form of people or animals and can obviously be very distressing. One of the more difficult aspects of treatment of such psychotic symptoms is that the first choice of treatment in most other conditions, neuroleptic medication, has been found to be unsafe for people with LBD. There have been severe sensitivity reactions reported including increased drowsiness and impairment of consciousness and Keith (2002) reports a two- to threefold increase in mortality. On a more positive note Keith (2002) also reports that acetylcholinesterase inhibitors (the first line of treatment in Alzheimer's disease) elicit improvement in LBD and while they are not currently licensed for use in LBD they may soon become the first line of treatment.

Evidence for Practice

Lewy Body Dementia

People with Lewy body dementia experience gross fluctuations, often on a daily or even hourly basis, of cognition and functional ability

People with LBD experience symptoms associated with Parkinson's disease and are prone to falls

Hallucinations are common in the experience of someone who has LBD, which can be very distressing

Neuroleptic medication, often used to treat hallucinations, can be harmful when used as treatment in people with LBD

Acetylcholinesterase inhibitors may become the recommended treatment for LBD