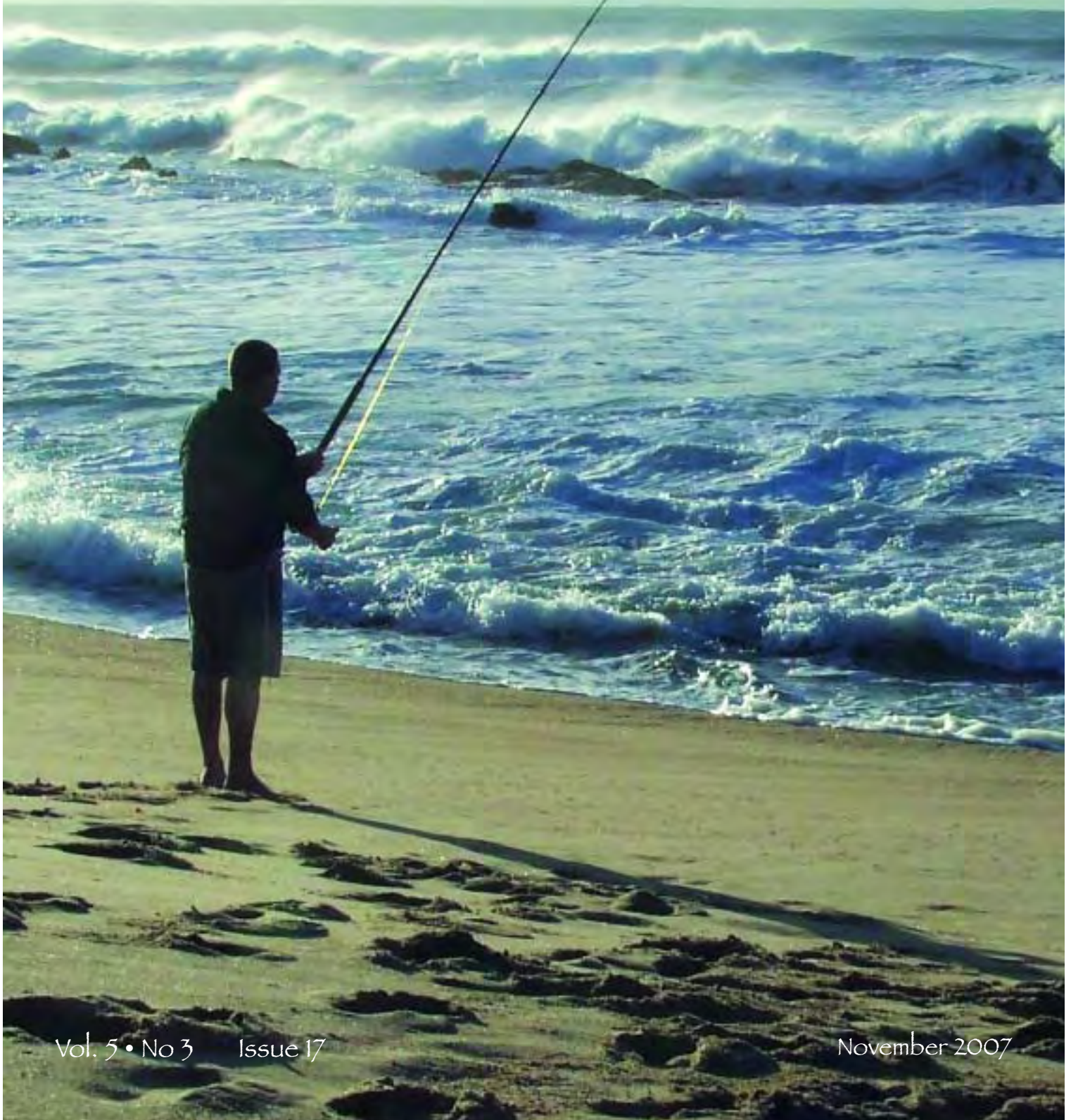


Serenity

Anxiety and Depression – A Positive Outlook



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Are we acting responsibly?

One of the best skills people can learn is “to take responsibility for one’s decisions and actions.”

This is learned through practice and experience, through repetition and often some suffering. People who act responsibly also feel in control of their own destiny and they take ownership of the consequences.

For all partners in the health care industry to take ownership of their destiny, they need to be made responsible for decisions and actions they take. They should be made to feel in control of their own destiny. A legal framework that incentivises this should be created.

Government is entrusted by law to provide this legal framework for South Africa to enable the roleplayers in the private health care industry to live happily and work successfully in South Africa and to help those in need.

Legislation should be aimed at regulating particular sections of the South African community to ensure a happy, sustainable and productive environment for all. At times legislation is not seen as popular, but can have a dramatic effect on our lives and in the end be very successful. One such example is the Tobacco Products Control Act, Act No. 83 of 1993. Not a popular act when amended in 1998 by the then Minister of Health, it also did not prove to be a deterrent to people smoking less or to stop smoking, but it ensured very effectively the rights of smokers to smoke and of non-smokers to live in a smoke free environment.

Examples of legislation that have not proved to be so effective in regulating the South African environment are the Medicines and Related Substances Control Act, Act 101 of 1965, the Mental Health Care act, Act 17 of 2002, the National Health Act, Act 61 of 2003, The Medical Schemes Act, Act 131 of 1998 and others.

What has gone wrong with these acts?

The Mental Health Care Act, although ensuring the rights of psychiatric patients to deny treatment, is so confusing in its application, that there is little consistency in the application of the law, and thereby also denies patients with no insight into their condition at the time, any help and treatment. It places the burden mainly on the family to get assistance not considering the problems often present in families in patients with psychiatric problems. Thus denying patients the right to treatment as the families are often not willing to go through the process of getting patients involuntary care.

The Medicines and Related Substances Control Act, although ensuring a cost reduction in the prices of medicines, has not ensured trust in the market, especially the generic medicines market. Although generic medication is widely prescribed by doctors and purchased by patients, the market is heavily driven by funders and the only reason why patients often take generic medication is due to the lower cost. Regulation does little to incentivise patients and doctors to help build confidence in the market. The only selling point is price with very little comparison on efficiency of products, thus not building trust in this market sector. Doctors and patients are expected to find out for themselves if a product works or not. In patients with psychiatric

problems, this is of particular relevance as there is a large placebo response, and if the doctor and the patient do not have confidence in the treatment, it could lead to dissatisfaction and poor response of the medication. Is this regulation or control?

The section mandating patients on psychiatric medication to consult with a psychiatrist and even if they consult with a psychiatrist to see another psychiatrist after 6 months is so pedantic, that it had to be corrected by an amendment in the Mental Health Care Act that superseded the first act. Control rather than regulation.

The Medical Schemes Act, already promulgated in 1999, has up till now not yet been fully implemented with medication schemes interpreting Prescribed Minimum Benefits



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(PMB’s) benefits all differently, waiting periods varying from scheme to scheme, provision of ICD-10 diagnostic indiscriminately applied with little regard for patient confidentiality and cost still not contained. Yet again, control rather than regulation.

The National Health Act, already published in 2003, has still not been fully promulgated and already sections have been legally challenged. More control without regulation.

The more one tends to control, the more others tend to assist. This leads to conflict and dissatisfaction and ultimately no solution.

With the suggestions from the Department of Health and the Minister that regulation is needed to assist in solving the ever increasing cost of private health care and the conflict between the roleplayers in this sector, one can only hope that lessons from the past have been learned.

“Regulation” will assist in solving problems; “control” will leave us yet again without solutions. ◆

Of Pawns and Kings

and Mates ... e4 = P - K4



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INTRODUCTION

An attempt to explore the contribution that chess, like psychiatry, an art and a science, has made to research in psychology and to philosophical debate and reasoning.

A focus too on the practical value of a "mere game" in the development of the human potential.

"Chess is a sea where a gnat may drink and an elephant may battle."
(Indian proverb)

Psychological Studies

Psychological studies have for years tried to explain how the mind organises and retrieves information. Skills at chess are measurable and open to study. Hence chess has served as a test for theories of thinking and has been called the *Drosophila* of cognitive science.



Chess has helped provide insight into human thought processes and has been a vehicle for the implementation of artificial intelligence programmes. Binet (1894) observed blindfold chess players to investigate memory.

Rueben Fine (1956), psychoanalyst and chess-player held that chess was a substitute for war. In a Freudian analysis the king and queen were seen as parent figures and the minor pieces as phallic symbols. Adrian De Groot, Dutch psychologist, explored thought and choice in chess and marked orientation, exploration, investigation and proof as stages of choosing the next move.

Saariluoma (1995) used a cognitive approach to explore spatial relationships in chess and saw the board as a "mental space upon which several operators may be applied."

Benefits

Robert C. Ferguson cites numerous studies which show that chess improves:

- Cognitive Development
- Maths and science scores
- Organisational skills
- Memory, attention, concentration
- Reasoning
- Reading performance
- Self esteem
- Fantasy and imagination.

A Venezuelan study claims that chess develops new forms of thinking – an exercise that increases the intelligence quotient.

Recent research speculates that it is the growth of new synaptic connections that aids this process.

Chess has this impact because it provides:

- Exposure to problems
- Immediate punishment and rewards

• Competition that fosters interest and promotes mental alertness

- Novelty

Chess also requires and develops:

- Determination to win
- Purposefulness
- Persistence
- Risk taking
- The ability to analyse.

Hence chess is part of the school curricula in 30 countries.

The social impact of chess has been demonstrated in the movie "Knights of the South Bronx" – chess was the means to improve the lives of inner city children and rescue them from gangs and drugs.

“The way he plays chess demonstrates a man’s whole nature.”
(Stanley Ellen)

Philosophy

The Tao of chess shows how chess reveals underlying truths that may be adapted from the chessboard to every aspect of life. Searching for the truth in a position with energy and determination develops thought processes that become habitual.

Siitonen and Pihlstrom (1998) explore the relationship between chess and philosophy. They examine how chess may shed light on features of philosophical problem-analysis and argumentation.

Thought patterns and reasoning procedures typical of chess seem to merge into those practised in philosophy. Philosophical insights inspired by the practice of chess may transform views about complex human phenomena including ethical reasoning and philosophical argumentation.

Therapeutic value

Games may be used to create a therapeutic alliance and to serve as a diagnostic and assessment tool. Gaines et al. explored the use of chess as a means of improving object relationships in narcissistic teenagers. It helped deal with interpersonal and school relationships.

Numerous anecdotal reports record the role of chess in therapy as a means of probing deeper in search of the absolute truth and as a

diversion. Chess is seen as an outlet for competition, a means to overcome life’s obstacles, a means to gain mastery over one’s impulses. It provides for the inhibition and sublimation of aggression.

A cautionary note ...

Chess may become an addiction/obsession. The Vladimir Nabokov novel *Zaschita Luzhina* reflects on the descent from obsession to delusion to suicide in a chess grandmaster who becomes insane.

Fischer-Spassky, Reykjavik, Iceland July 1972 encapsulated the passions of the Cold War – a World Championship game was marked by paranoia and psychological warfare as the battlefield extended beyond the chessboard.

“The chess master must have courage, a killer instinct, stamina and arrogance.”
(Harry Evans)

In the words of that unknown Indian philosopher “chess is an art and a science ... that heals the mind ... and teaches the angry man to restrain his passion.”

Author’s note: This article was originally presented as a poster at the 10th International Conference on Philosophy, Psychiatry and Psychology at Sun City in August 2007. ◆

[References on request](#)

“There is no other game so esteemed, so profound and so venerable as chess. In the realm of play, it stands alone in dignity.”
(Ely Culbertson)



Mood Symptoms

during the menopausal transition



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Menopause is a natural process that occurs in women's lives as part of normal aging. Despite the normality of this event, most women view menopause as a period that will be associated with mood swings, hot flushes and a general decrease in their quality of life. In this article I will review the literature on the association between the menopausal transition and mood symptoms, explore the possible etiology of these disorders and discuss the different management strategies for mood disorders during this period.

What is menopause?

Menopause is defined by the World Health Organization and the Stages of Reproductive Aging Workshop (STRAW) working group as the permanent cessation of menstrual periods that occurs naturally or is induced by surgery, chemotherapy, or radiation (NIH State of Science Panel on Menopause, 2005). Natural menopause is recognized after 12 consecutive months without menstrual periods that are not associated with a physiologic (e.g., lactation) or pathologic cause. Menopausal transition often begins with variations in length of the menstrual cycle. The hormonal changes during the menopausal transition can span several years.

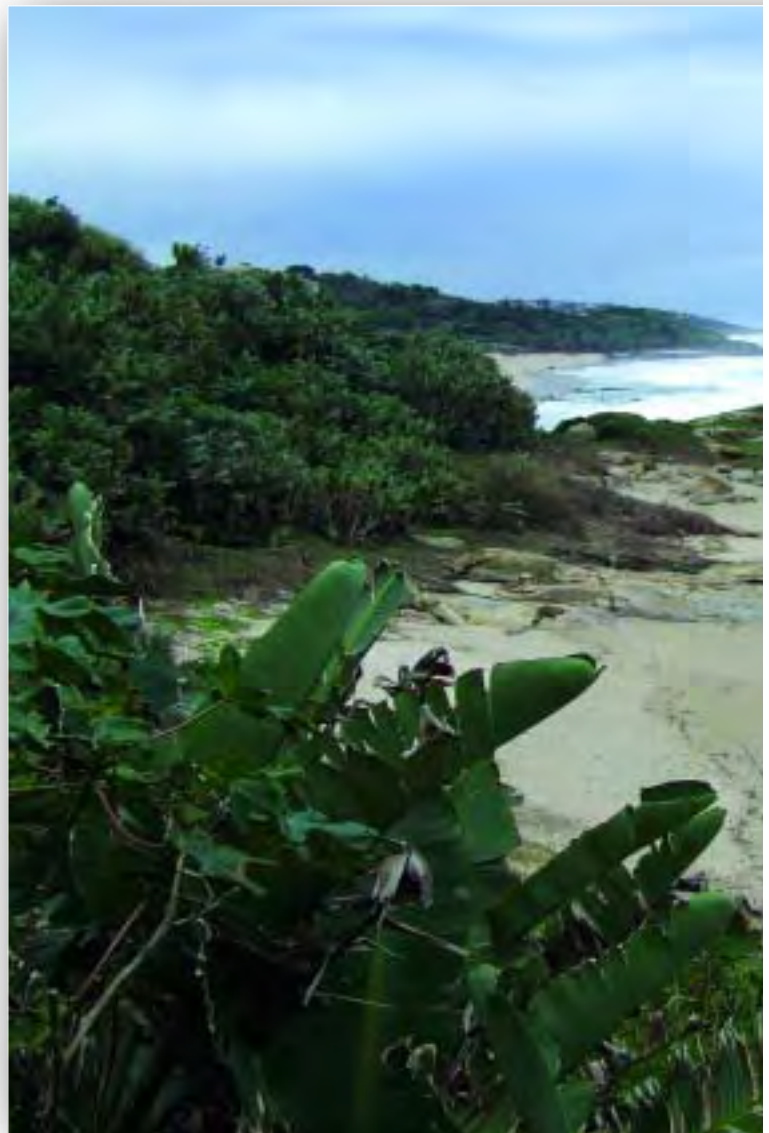
Perimenopause is not formally defined by the STRAW criteria, however it is a term that is frequently clinically used. It is defined as the period immediately prior to menopause (when the biological and clinical features of approaching menopause begin) and the first year after menopause (NIH State of Science Panel on Menopause, 2005). Thus, perimenopause includes the menopausal transition and overlaps the first 12 months of postmenopause. Perimenopause may be diagnosed even pre-symptomatically if FSH is above 25 IU/L and estrogen less than 40 pg/mL when drawn during the early follicular phase. During this time, there are fluctuations in the levels of gonadal steroids, with initial increase, then marked decrease in estradiol levels. Progesterone, another ovarian androgen, also decreases. (Aloysi et al 2006)

Is menopause a time of increased risk for mood symptoms?

Several longitudinal studies that follow women across the menopausal transition indicate that

risk for significant depressive symptoms increases during the menopausal transition and then decreases in the early postmenopause (Avis et al 1994, Freeman et al 2004). In the largest community-based study to date, the Study of Women's Health Across the Nation (SWAN) (Bromberger et al 2001) reported that women in early perimenopause had a higher rate of persistent mood symptoms than premenopausal women.

Freeman and colleagues (Freeman et al 2004) identified an increased risk for significant depression (defined by elevated CES-D scores and the Primary Care Evaluation of Mental Disorders [PRIME-MD]) during the perimenopause compared with the premenopause or postmenopause. Moreover, this



association remained after adjusting for several variables, including past history of depression, severe premenstrual syndrome, poor sleep, and hot flashes.

Schmidt and colleagues, (Schmidt et al 2004) prospectively followed 29 women with regular menstrual cycles (mean follow-up, 5 years) until they experienced 6 months of amenorrhea. The study revealed that the 24-month period surrounding the menopause carried a 14-fold increased risk for depression, as compared to both reproductive years and post menopausal period.

Several other studies confirm that, unlike perimenopause, postmenopause is not associated with an increased risk for developing depression. (Bromberger et al 2001, Kaufert et al 1992, Woods et al 2002)

What are the risk factors for developing depression during the menopausal transition?

As with mood disorders in general, a previous episode of depression (including premenstrual depression or postpartum depression (Stewart et al 1993) is one of the strongest predictors of a mood disorder in the perimenopausal period. Other risk factors include longer duration of the peri-

menopause (defined by menstrual cycle irregularity), (Kronenberg 1990) presence of hot flashes (Kronenberg 1990, Joffe et al 2002) stressful life circumstances, complaints of poor health, history of smoking, disturbed sleep, reduced parity, and absence of a partner (Bromberger et al 2001)

What is the possible etiology of depression in the menopausal transitions?

Hormonal factors

The "estrogen withdrawal theory" proposes that the onset or worsening of mood symptoms in perimenopausal women result from a significant decline in peripheral concentrations of estradiol. However, estrogen levels increase during the early perimenopausal period and then drop again, which contradicts this theory (Burger et al 1999) Furthermore, no difference between estrogen levels have been found between women who develop depression and those who do not.

If one looks at the period of greatest risk for depression (ie perimenopause) this represents a period of greatest decline in estrogen, suggesting that sensitivity to changing rather than lower levels of estrogen may be the causative factor. This is supported by higher rates of depression in women who have undergone bilateral oophorectomy (surgical menopause) who experience abrupt changes in estrogen levels. The increased risk for perimenopausal depression associated with premenstrual and postpartum depression, both times of significant fluctuations in hormonal levels, also supports this theory.

Furthermore, placebo controlled trials of estradiol have shown improvement in depressive symptoms in perimenopausal (Joffe et al 2001, Odmark et al 2004, Soares et al 2003) but not in postmenopausal women (Grady et al 2003), suggests that mood disorders occurring in perimenopausal women are caused by changes in hormones rather than prolonged ovarian steroid deficiency.

Changes in testosterone may also play a role in the pathogenesis of mood symptoms. Depressive and anxiety symptoms, as well as decreased libido, have been described among postmenopausal women who present with decreased testosterone, particularly after oophorectomy. Testosterone supplementation has been shown to alleviate these symptoms in some women treated concurrently with estrogen. (Shifren et al 2000)

Hot Flashes

Hot flashes are the primary symptom of the menopausal transition. The prevalence varies from 35% to 50% in perimenopause, and from 30% to 80% in postmenopause.

Hot flashes are a manifestation of the dysregulation in the thermoregulatory



centre in the hypothalamus that occurs in the setting of ovarian failure and estrogen withdrawal. (Joffe et al 2002)

Several studies have shown a strong association between hot flushes and depression (Bromberger et al 2001, Joffe et al 2002) Some have suggested that depression during the menopausal transition is an indirect consequence of sleep disruption that occurs in association with hot flushes. Others have hypothesized that depression during the menopausal transition results from sensitivity to changes in estradiol in the brain and suggest that the association between perimenopausal depression and hot flushes indicates that both are markers of the brain's sensitivity to changing levels of reproductive hormones in some individuals (Rubinow et al 1998).

Psychosocial factors

"Empty-nest syndrome" (when children leave home) has been extensively used to characterize the psychosocial origin of depressive symptoms manifesting during the menopausal transition. The relative validity of this theory, however, is questionable, and it appears to be restricted to a subgroup of women who have children and who are overly engaged with them. (Dennerstein et al 2002)

Management of depression during the menopausal transition

Hormonal measures.

Randomized placebo-controlled trials of estrogen for perimenopausal depression indicate that it is an effective treatment in this population. (Soares et al 2001, Schmidt et al 2000). Estrogen works within the first month of its use, but there is no data on the duration of use required to obtain sustained antidepressant benefits from estrogens. Estrogen is not effective for depression in the postmenopausal woman.

The concomitant use of progesterone has not been specifically investigated, however, clinical evidence suggests that the use of progestogens may lead to increased irritability and fatigue (Soares et al 2003) and that cyclic use of progesterone deteriorates mood in those whose depression responds to estrogen (Odmarm et al 2004).

The use of hormonal therapy however, is not without risks. Unopposed estrogen, however, is contraindicated in any woman with an intact uterus and long term (> 4 years use) has been associated with an increased risk of cardiovascular disease and stroke.

Furthermore, rapid re-emergence of menopausal symptoms have been reported in women who discontinue hormone replacement therapy (Kronenberg 1990). It is yet to be determined whether the discontinuation of hormone therapy will increase the risk of depression, particularly in those women who initiated the medication for treatment of menopause-associated depression.

Given these risks hormonal treatment is probably best reserved for women with depression and significant menopausal symptoms who have not responded to an antidepressant.

Antidepressant treatment

Extensive data exist regarding the efficacy of antidepressant therapy for the specific treatment of depression in both perimenopausal and postmenopausal women (Joffe et al 2001, Soares et al 2003). This has been demonstrated across several antidepressant classes including SSRI's, TCA's SNRI's and NaSSA's (Cohen et al 2005). As in other patients with depression, treatment studies suggest that relapse into depression is minimized if antidepressants are continued for 6 to 12 months of wellness.

Antidepressants have also shown utility in treating hot flushes, in patients both with and without mood symptoms. In particular, efficacy has been demonstrated for venlafaxine and paroxetine.

Given their efficacy and safety, antidepressants should be the first line of treatment for the perimenopausal woman with depression.

Non-pharmacologic interventions

Nonpharmacologic interventions such as specific psychotherapies have not been systematically evaluated as a treatment for depression during the menopausal transition. A few reports indicate that measures such as relaxation response training and exercise (Irvin et al 1996, Coope 1996) can help in preventing or ameliorating symptoms of depression, and these may be a useful adjunct to antidepressants.

Conclusion

The menopausal transition is a time of increased risk for mood disorders. Similar risk factors for the development of a depressive episode during the life cycle apply during this period. In addition, increased severity of menopausal symptoms, such as hot flushes, pose an additional risk. Antidepressants remain the treatment of choice for depression in this period, ideally with an agent that is effective in treating hot flushes as well. ◆

References on request

The Mark of Men (tal Health Care)

Then Cain said to the Lord, "I am being punished more than I can take! See, this day You have made me go away from the land. And I will be hidden from Your face, I will run away and move from place to place. And whoever finds me will kill me." So the Lord said to him, "Whoever kills Cain will be punished by Me seven times worse." And the Lord put a mark on Cain so that any one who found him would not kill him.

Genesis 4:13-15 (The New Life Bible)



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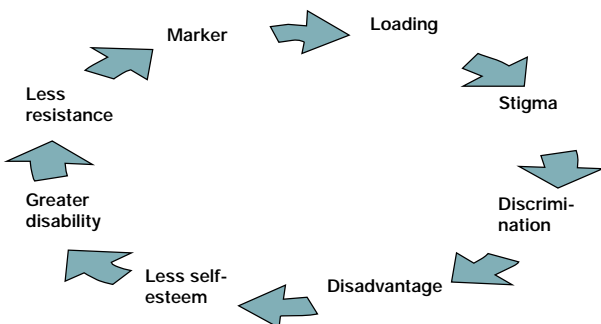
As Cain was ostracised and made to "go away", stigma continues to separate mental healthcare users from their peers and communities. But, unlike Cain, who was protected from death by the mark, stigma leaves them vulnerable, misunderstood and isolated.

Unsurprisingly the term stigma derives from the Greek stigma meaning "dot" or "brand" according to the Oxford Dictionary. It refers to the Greek practice of branding and disfiguring slaves as a way of easily identifying them as such and confirming their lower social status. The World Psychiatric Association in a publication on reducing stigma of Mental Illness mentions that the word "stigma" may mean, "to pierce, to make a hole". According to this publication stigma became to mean branding a criminal with a hot iron to mark infamy. During the late middle ages it took on the meaning of public defaming and branding of a criminal "SO THAT ALL COULD RECOGNISE HIM" (my emphasis). Stigma is linked to prejudice as the latter refers to an attitude of readiness to act in a positive or negative way towards the object of prejudice without examining whether justification for such behaviour exists. Focus groups revealed four dimensions of stigma. These dimensions are interpersonal stigmatisation, denying the mental healthcare user social roles and relationships. Stigma not only impacts social roles but also social benefits e.g. employment and housing. Structural stigma refers to structural imbalances built into legal regulations, health and other insurance as well as political decisions. The latter factor often impacts the human rights of mental healthcare users. Lastly stereotypical and largely negative portrayals of mental healthcare users in the media and entertainment arena entrenches the stigma within the community sphere, leading to social isolation and abandonment. Stigma creates an "us" and "them".

Shame is the heart of stigma.

Sartorius (2000) described an operational model of the "vicious cycle of stigmatisation".

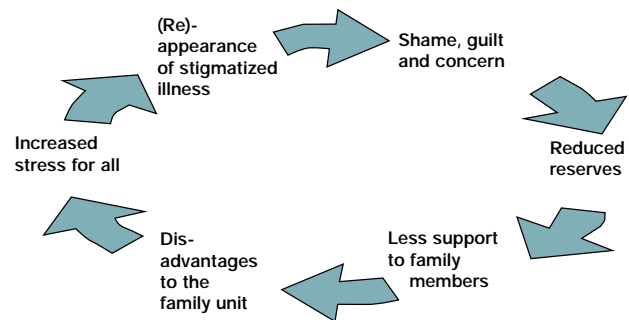
For the individual patient, the following cycle exist:



A marker or visible abnormality allows for the identification of a person as belonging to a specific group. This may be behavioral symptoms, extra pyramidal side effects or being seen at the psychiatric practice/clinic in the context of psychiatric disorders. The marker becomes

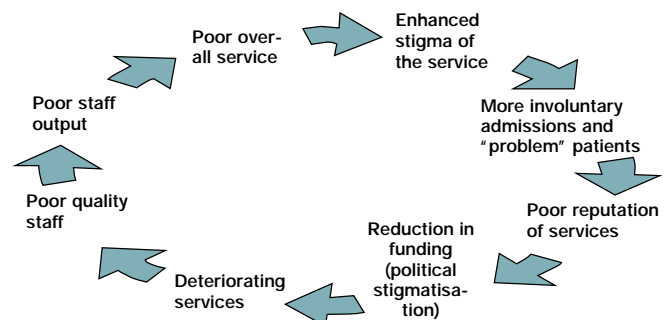
loaded with negative contents leading to the formation of stigma. Because of prejudice, the stigma leads to negative discrimination, leading to disadvantage including but not limited to poor healthcare, lack of access to social benefits and social isolation. This leads to a decrease in self-esteem and self-efficacy, enhancing the perceived disability. The person so stigmatised, offers less resistance to the offending illness and adverse circumstances and thus enhances the "marker" with predictable results.

Sartorius went on to describe a cycle of stigmatisation of the family:



This cycle is epitomised by a quote by a member of an anti-stigma focus group in India: "My parents support me but we can't tell any of our neighbours (about my diagnosis of schizophrenia). It would hurt my sister's chances of being married".

Sartorius is even of the opinion that stigma may affect mental healthcare workers!



Stigma is the "basal cell carcinoma" of mental health, overtly visible to the naked eye but covertly destroying the psyche of mental healthcare users, providers and the community. It impinges on the human rights of those affected and marks them as different and undesirable.

Fighting stigma ought to be the part of good clinical practice for every mental healthcare practitioner. It takes energy, courage, vision and leadership. It strives for equality, compassion and respect, even for those other than "us".

It should be the true mark of men. ◆

Geriatric Depression



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INTRODUCTION

The aged segment of the population has grown over the last decade. Research in the area of geriatric psychiatry is playing catch-up. Depression, occurring more commonly than dementia and delirium¹ has an increased risk of mortality from suicide and medical illness². Clinically significant depression increases in older people with age³. Despite effective treatment options and treatment responsiveness that parallels younger cohorts², the prognosis of geriatric depression is still guarded¹.

Psychiatrists must become skilled in managing the organic basis⁴ of late-onset depression and any medical co-morbidity⁵ for this to change.

The complexity of the clinical picture

The diagnostic process in geriatric depression is a complex one. Of the 1-4% of the elderly that have major depression, 40% have recurrence⁵ from earlier depressions. The 30% with late-onset major depression have specific deficits in tasks of attention and executive function⁵. More commonly, a 'subsyndromal depression'⁶ that presents with somatic concerns instead of sadness, poor subjective memory, late-onset neurotic symptoms,² apathy⁷ and poor motivation³, a negative view of self, the world and the future is seen.

Complicating the clinical picture is mild cognitive impairment in as high as 60% of patients³. This memory dysfunction persists even after response to antidepressant treatment⁷, highlighting the role of the ageing brain, especially mesial temporal lobe dysfunction⁸. Although psychosocial factors trigger depressive symptoms, both loss and cardiovascular risk factors have been found to be independent predictors of depression in the elderly⁹. It is thought that vascular disease disrupts mood regulation circuits in the brain, which decreases its ability to respond to stressful events¹⁰.

Late-onset depression: psychiatric or medical morbidity?

Depression affects one in 7, dementia, one in 17 and delirium, one in 25¹. Ischaemic small vessel disease has been implicated in the pathogenesis of late-onset depression⁷, despite the fact that opinion is divided on the concept of 'vascular depression'. Imaging studies consistently show deep white matter lesions in depressed patients with cerebrovascular risk factors like hypertension and cardiac disease⁸. These white matter changes pre-date the development of depressive symptoms¹². Not everything is that clearly linked. Apathy in the presence of cognitive decline is consistent with frontal lobe dysfunction⁷, yet the incidence of depression in patients with fronto-temporal dementia is inexplicably low.

One key lies in the association between cardiovascular disease and late-onset depression. Consistent with its link to other vascular disorders, the angiotensin II receptor, vascular type 1 (AGTR1) genotype is associated with treatment outcome in late-onset depression¹⁴. Cardiac morbidity and mortality post major depression is attributed to ischemic heart disease¹⁰. High plasma homocysteine and triglyceride levels have been found in depressed older men¹³. In fact, major depression in older age predicts first cardiac events¹⁴.

The role played by degenerative changes is also not clearly defined. Structural and functional links between depression and dementia of the Alzheimer's type are well documented despite the absence of genetic history or apolipoprotein E¹⁶. Imaging studies have consistently reported a decreased volume of the frontal and temporal lobes, ventricular enlargement, sulcal widening and decreased volume of the hippocampus and caudate nucleus⁸. Regional cerebral blood flow in the left anterior temporal and left anterior frontal regions is also impaired⁴. Depressive symptoms occur in patients with stroke, Parkinson's disease and other degenerative neurological conditions¹.

Implications for management

As with any other psychiatric condition the principles of management of geriatric depression include; making an accurate diagnosis, excluding other treatable conditions like delirium and selecting effective treatment regimes. The difference stems from the medical co-morbidity which requires treatment options not usually regarded as part of the psychiatrist's toolkit e.g. medications that halt vascular damage.

Evaluating and controlling cardiovascular risk factors is an

integral part of the management of late-onset depression¹⁰. Assessment must include a physical examination², a full neurological examination, mental state examination and a MMSE. Adverse outcomes in late-life depression have been seen in patients with neurological signs consistent with subcortical-frontal dysfunction. Although the Geriatric Depression Rating Scale¹ is used as a screening instrument, symptoms like diminished self-care, increased irritability, social withdrawal and psychomotor retardation are often missed. Anaemia, hypothyroidism, hypokalaemia, hyponatremia, vitamin B12 and folate deficiency, the use of medications like steroids, anticholinergics, benzodiazepines, cimetidine, antibiotics, analgesics and antihypertensives must be excluded as causes for depression.

The evidence base for the management of late-onset depression is increasing although recommendations are still extrapolated from studies using non-geriatric patients. Specifically, antidepressants have been shown to effectively reduce suicide risk in the elderly¹⁸ and improve cardiac recovery after cardiac events. Anxiety in the elderly is rarely present alone¹⁶ and its detection is vital to effectively managing depression. Frontal sub-cortical damage is associated with treatment resistance¹⁹.

Age-related pharmacodynamic changes do affect treatment response. The higher fat percentage, less volume fat of distribution, muscle mass, and total body water in the elderly causes a higher concentration of liposoluble drugs in the brain. Decreased plasma protein levels cause diminished drug binding and increased levels of free active drug. Decreased size of both the liver and kidneys promotes drug accumulation and delays drug elimination. As the brain ages, alterations in receptor reactivity can affect neurotransmitter levels and target tissue responsiveness and reduction of dopamine levels increase the risk of extrapyramidal symptoms from neuroleptics, SSRI's and other antidepressants.

Selection of an appropriate agent is the anchor of any treatment regime. SSRI's like escitalopram and paroxetine¹, bupropion, mirtazapine, and venlafaxine¹ have all shown efficacy and safety in the elderly. Sertraline is relatively safe and effective in treating depression in the patient with ischemic heart disease¹⁶. For the severe or psychotic depression, atypical antipsychotics like risperidone, quetiapine and olanzapine have proved effective. Melancholic depression responds well to ECT¹. The elderly are susceptible to iatrogenic complications like the lowering of sodium levels that can occur with an SSRI¹. Studies

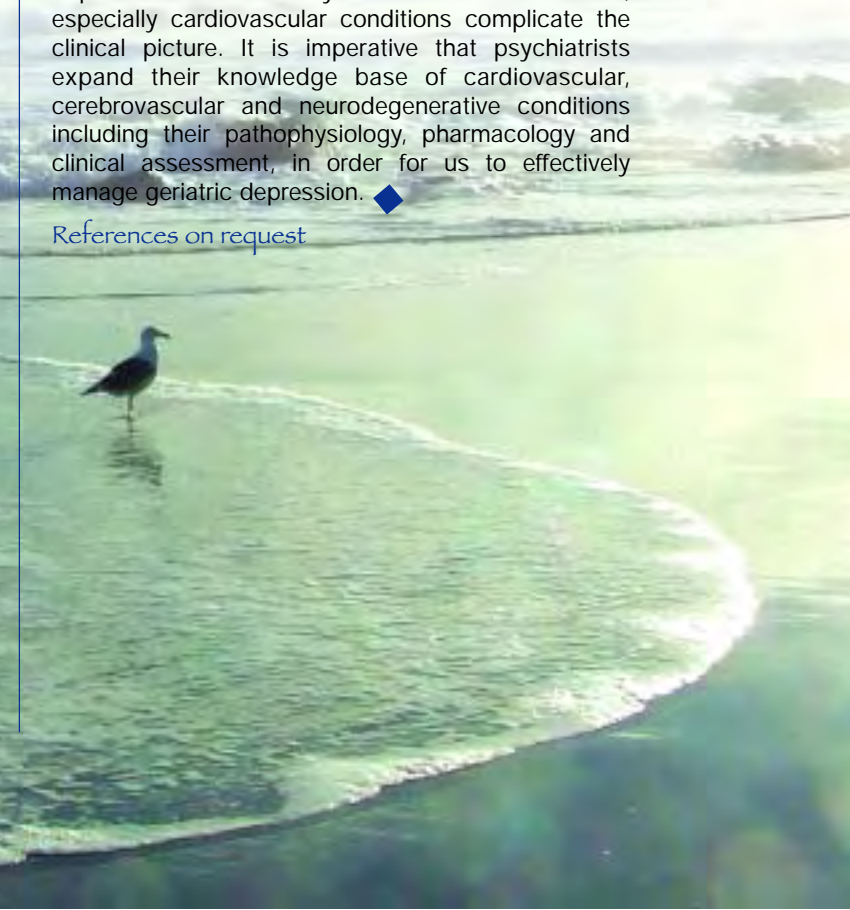
have also demonstrated the increased risk of falls, tremors, extra-pyramidal symptoms, somnolence, and delirium.

Despite cognitive changes, psychotherapy is an important treatment option in geriatric depression and improves treatment outcome. Many geriatric patients have religious and spiritual beliefs that form part of their coping skills especially when there is loss. Factors associated with lower rates of complicated bereavement include the reliance on prayer as a means of coping.

Conclusion

The prevalence of geriatric depression is increasing as the aged segment of the population expands and grows older. It has a negative impact on quality of life, adds significantly to disability from physical disorder and is the leading cause of suicide in older people. It is also an independent predictor of mortality. The high incidence of cognitive impairment highlights the role of the ageing brain, either from neurodegenerative or vascular changes or both, in the pathogenesis of depression. Co-morbidity with medical conditions, especially cardiovascular conditions complicate the clinical picture. It is imperative that psychiatrists expand their knowledge base of cardiovascular, cerebrovascular and neurodegenerative conditions including their pathophysiology, pharmacology and clinical assessment, in order for us to effectively manage geriatric depression. ♦

References on request



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