Research is appearing that can help identify early cases of dementia or neurocognitive disorders.

For years researchers have focused on detecting dementia early in the hope of studying the disorders in its early phases. This would give doctors the ability to research early treatments that could either delay or stop the progression of dementia.

Studying the brain has always been difficult due to its inaccessibility. The more invasive the investigation, the more problematic, expensive and dangerous the procedure will be. Simple, cheap and non-invasive screening techniques are the best in detecting disease early.

Researchers have now, through careful observation, found that simple observations could yield the best results for detecting dementia early. Several scientific publications have now found that a simple smell test can detect dementia early. Patients with early dementia have an inability to smell for instance peanut butter.

The olfactory bulb is thought to be involved because smell loss occurs only in neurodegenerative conditions where there is olfactory pathology, such as Alzheimer’s and Parkinson’s disease.

Neurofibrillary tangles, features of Alzheimer’s Disease (AD), have been found in the olfactory bulb and tracts before the onset of symptoms, suggesting that olfactory deficits may be early markers of the disease.

AD symptoms in the entorhinal cortex, hippocampus and other temporal regions may limit ability to store and retrieve memories of smell and thereby to identify odours correctly.

Some studies suggest that the left nostril is affected earlier than the right nostril but other similar studies could not replicate this finding.

Early changes in the sense of humour in patients with fronto-temporal dementia also seem to be an early sign. Changes in retinal thickness seems to be an early sign of Alzheimer’s dementia.

All of these simple measures are non-invasive and the hope is that more tests would be developed and researched to enable the early detection of...
Depression is a disorder of the body as much as of the mind. The traditional understanding of pain and depression as separate conditions with overlapping symptoms has evolved through research into an understanding that pain and depression share pathophysiological mechanisms. These shared pathophysiological mechanisms include origins, mechanisms and neurotransmitters, resulting in shared treatments. In addition, pain and depression have a reciprocal relationship in that each heightens the severity of the other. Failure to eliminate the pain symptoms reduces the chances of full recovery from depression: it keeps depressed people from regaining full function in their personal and professional lives, and it raises the danger of suicide. Furthermore, the presence of a depressed mood increases the perception of the severity of, and contributes to distress associated with pain.

What is pain?
The International Association for the Study of Pain (IASP), defines pain as an emotional experience associated with actual or potential tissue damage. Pain is a multidimensional experience that includes discriminative, affective, motivational and cognitive components mediated by spinal, brainstem and cerebral functioning, modulated through forebrain mechanisms. Pain, as a sub-modality of somatic sensation, has been defined as a “complex constellation of unpleasant sensory, emotional and cognitive experiences provoked by real or perceived tissue damage and manifested by certain autonomic, psychological, and behavioural reactions”.

The severity of pain does not only bear a simple relationship to the degree of tissue damage. In addition to various genetic, epigenetic, and environmental factors, interpersonal variability in the engagement of emotional cortico-limbic circuitry by pain may explain why some patients develop chronic pain conditions and others do not. This is supported by consistent findings of chronic painful symptoms in stress-related mood and anxiety disorders, with up to 80% of patients with depression reporting comorbid pain conditions, while the presence of continuous pain increases the severity and frequency of depressive symptoms with up to four times. The co-morbidity of chronic pain and chronic depression makes it difficult to pinpoint the temporal and causal relationship, but the correlation between the two conditions is clear. People in pain who are also depressed become heavy consumers of medical services, even if they have no severe underlying illness. Pain slows recovery from depression, and depression makes pain more difficult to treat (e.g. due to lack of motivation for compliance). Both pain and depression exacerbates themselves through changing both brain function and behaviour.

Depression leads to isolation and isolation leads to further depression; pain causes fear of movement, and immobility creates the conditions for further pain. When depression is treated, the emotional impact of pain diminishes, while when pain is alleviated, so is much of the suffering that causes depression.

The biological pathways of pain
Pain processing typically involves transmission and modulation of noceptive signals along a predictable pathway. Cutaneous nociceptors are an extremely heterogeneous group of neurons. These nociceptors are generally electrically silent and transmit all-or-none action potentials only when stimulated by external noxious stimuli (temperature extremes, intense pressure, or chemicals). However, nociceptor activity alone does not lead to the perception of pain. Peripheral information is needed to reach higher cortical centres and depends on the frequency of action potentials in primary afferents, temporal summation of pre- and postsynaptic signals, and central influences. Acute pain is divided into fast pain (which is sharp, easily localisable and does not cause much emotional anguish) and slow pain (which is burning, aching, throbbing and triggers autonomic and emotional reactions). Fast pain is mediated by small lightly myelinated Aδ (A-delta class)-fibres, while slow pain is mediated by unmyelinated C-fibres which are stimulated by noceptive chemicals (serotonin, substance P, prostaglandins) released after the damage.

Pain modulation occurs at both spinal cord level, and centrally. Peripheral nociceptive neurons synapse in the dorsal root ganglia where interneurons cause inhibitory/excitatory modulation. Fast and slow synaptic transmission are enhanced in large part by glutamate and peptides (e.g. substance P, CGRP).
Of particular importance to pain perception is the plasticity in synaptic strength (i.e. the ability to enhance homosynaptic as well as heterosynaptic connections) between primary efferents and the relay and interneurons they drive, presynaptic and postsynaptic modulation by descending facilitatory and inhibitory pathways in the spinal cord, and the efferent aspects of nociceptor function activated by strong GABAergic/glycinergic depolarisation of presynaptic terminals leading to the dorsal root reflex.\textsuperscript{14,15}

Secondary spinal projection neurons then transmit the information to two areas of the brainstem – the rostral ventral medulla and periaqueductal gray matter - where they are further modulated and relayed to the thalamus.\textsuperscript{16} All efferent neurons end in the thalamus where they synapse with three sets of neurons: those projecting to the somatosensory cortex ("where is the pain?") , the limbic area ("how do I feel about the pain?") and the frontal cortex ("what am I going to do about the pain?").

There is a significant overlap between pathways involved in mood and pain regulation – especially the serotonergic (5HT)/norepinephrine (NE) pathways which is central to the gated control theory.\textsuperscript{17-19} Both 5-HT and NE have ascending pathways from the brainstem to the cerebral cortex and limbic areas where they mediate many emotional and physical functions. The descending 5-HT and NE pathways in the spinal cord, modulate and inhibit ascending pain signals. Therefore, increasing the availability of 5-HT and NE, may promote central pain inhibition.\textsuperscript{20}

Converging lines of evidence now also suggest that the pathophysiology of pain is mediated to a substantial degree via allostatic neuroadaptations in reward- and stress-related brain circuits.

Acute pain activates dopamine (DA) transmission in the brain’s reward and motivational centres, whereas prolonged periods of pain produce the opposite effect (within system adaptation) clinically manifested by anhedonia and diminished motivational/incentive salience of natural reinforcers, i.e. the reward deficiency state (RD). Allostatic adjustment (processes that attempt to normalise the stress on the system) to excessive dopaminergic transmission in response to recurrent pain leads to a between system adaption involving the central and basolateral amygdala nuclei, the bed nucleus of the stria terminalis, the lateral tegmental noradrenergic nuclei of the brain stem, and the hippocampus. This leads to massive surges of corticotropin-releasing factor (CRF), NE, glutamate and dynorphin - leading to the anti-reward state (AR).

Recurrent pain or ongoing pain may contribute surges of these stress-related chemicals – presenting not only with ongoing pain, but also with symptoms of depression which include poor motivation, apathy, and anhedonia. Glutamatergic sensitisation promotes overlearning of the motivational salience of pain, analgesia and cues that predict the onset or severity of pain so that pain is constantly perceived to be worse than expected (i.e. “catastrophising”). These effects result in an unstable positive feedback loop wherein the combined reward RD and AR model (CReAM) drives further enhancement of pain and thus contributes to progressive worsening of the clinical condition and an end-stage outcomes of chronic, intractable pain (see Figure 2).\textsuperscript{5}

In chronic pain states, inflammatory factors and sensitised receptors in the skin are thought to cause an abnormal increase in the transmission of nociceptive signals from the periphery as well as either a lack of inhibition or increased excitation, or both, at the spinal cord, brainstem or cortical levels, called “central sensitisation”.\textsuperscript{15,21} In addition, according to the CReAM model, biopsychosocial variables modulating brain reward, motivation and stress functions can interact in a “downward spiral” fashion to exacerbate the intensity, chronicity and comorbidities of chronic pain syndromes - amplifying the aversive physical and emotional aspects of pain. Such processes may further contribute to treatment resistance (to current pharmacotherapies) in chronic pain.

Neuroimaging has become an increasingly important and popular means of studying how the brain perceives and processes chronic pain. Various neuroimaging modalities, such as positron emission tomograph, encephalography, magnetoencephalography, single-photon-emission...
computed tomography, magnetic resonance imaging (MRI), and functional MRI, have contributed to elucidating many of the neural correlates regarding factors well known to modulate the experience of pain, including attention, anticipation, empathy, placebo, meditation, fear/anxiety and reward.\textsuperscript{22}

Evidence from functional MRI of the brain confirmed that depressive symptoms are related to the cerebral processing of pain and indicated an overlap in the areas for pain processing and sensation, and major depressive-related alterations in the brain.\textsuperscript{23} Pain processing has been associated with involvement of primary and secondary somatosensory cortex, thalamus, insular cortex, amygdala, anterior cingulate cortex, and the prefrontal cortex. Major depressive disorder (MDD) often exhibited lateral and medial frontal hypo- and hyper-metabolism, and metabolic changes in limbic regions such as insula and amygdala.\textsuperscript{24} This activation of the prefrontal cortices might reflect an underlying prefrontal psychopathology in depression. Negative affective states therefore clearly influence pain processing in terms of augmented pain experience.\textsuperscript{25} It is therefore crucial that both mood and pain should be treated simultaneously.

The optimal management of patients suffering from these two hurtful conditions

Patients with chronic pain can be challenging to manage and historically providers have relied on opiates to treat pain. However, recent studies have brought into question the safety and efficacy of chronic opiate therapy in the non-cancer population. Nonsteroidal anti-inflammatory drugs (NSAIDs) affect varying degrees of pain modulation through the inhibition of prostaglandin (PG) synthesis. NSAIDs have been shown to be effective in the treatment of chronic lower back pain, as well as chronic osteoarthritis. A recent meta-analysis have also demonstrated the opioid-sparing effect (20-30%) of the addition of an NSAID to a pain management regimen.\textsuperscript{26}

Almost every drug used in psychiatry can also serve as a pain medication.\textsuperscript{27-30} Relieving anxiety, fatigue, depression, or insomnia with mood stabilisers, benzodiazepines, or anticonvulsants will also ease any related pain. The most versatile of all psychiatric drugs, the antidepressants have an analgesic effect that may be at least partly independent of their effect on depression since it seems to occur at a lower dose. Other strategies include the use of anticonvulsants (e.g. carbamazepine, gabapentin, and pregabalin), topical agents (e.g. lidocaine and capsaicin), cannabinoids, botulinum toxin, and non-pharmacological strategies such as physical therapy, progressive muscle relaxation, hypnosis, meditation, and cognitive and behavioural therapies (CBT). CBT teaches patients how to avoid fearful anticipation, banish discouraging thoughts, and adjust everyday routines to ward off physical and emotional suffering due to chronic pain. See Table 1 (overleaf on page 6).
Depression has long been associated with pain. Although it was once thought that people with pain were somehow "denying" their emotional disorder and converting it into bodily pain, evidence now suggest that somatic complaints are the way some people become depressed. For a substantial number of people, possibly up to half of depression sufferers, bodily pain is the way depression presents itself. Failure to eliminate the pain symptoms reduces the chances of full recovery. Persistent pain typically keeps depressed people from regaining full function in the personal and professional lives, and it raises the danger of suicide. The goal of treatment is not just comfort or the absence of symptoms but restoring the capacity to lead a productive life.

### Summary

| Antidepressant medications | Medication Class | Starting Dosage | Titration | Maximum Dosage | Duration of Adequate Trial | Major Side Effects | Precautions | Other Benefits |
|----------------------------|-----------------|----------------|----------|----------------|---------------------------|-------------------|-------------|---------------|
| Secondary amine TCAs       | Nortriptyline   | 25 mg at bedtime | Increase by 25 mg daily every 3–7 d, as tolerated, until pain relief | 150 mg daily; if blood level of active drug and its metabolite is <100 ng/mL (mg/mL), continue titration with caution | 6–8 wk at maximum tolerated dosage | Sedation, dry mouth, blurred vision, weight gain, urinary retention | Cardiac disease, glaucoma, suicide risk, seizure disorder, concomitant use of tramadol | Improvement of depression, improvement of insomnia, low cost |
| Desipramine                |                 |                |          |                |                          |                   |             |               |
| SSNRIs                     | Duloxetine      | 30 mg once daily | Increase to 60 mg once daily after 1 wk | 60 mg twice daily | 4 wk | Nausea | Hepatic dysfunction, renal insufficiency, alcohol abuse, concomitant use of tramadol | Improvement of depression |
| Venlafaxine                | 37.5 mg once or twice daily | Increase by 75 mg each week, as tolerated until pain relief | 225 mg daily | 4–6 wk | Nausea | Concomitant use of tramadol, cardiac disease, withdrawal syndrome with abrupt discontinuation | Improvement of depression |
| Calcium channel α 2 -δ ligands | Gabapentin     | 100–300 mg at bedtime or 100–300 mg 3 times daily | Increase by 100–300 mg 3 times daily every 1–7 d, as tolerated, until pain relief | 3600 mg daily (1200 mg 3 times daily); reduce if impaired renal function | 3–8 wk for titration + 2 wk at maximum dose | Sedation, dizziness, peripheral oedema | Renal insufficiency | Improvement of sleep disturbance, no clinically significant drug interactions |
|                           | Pregabalin      | 50 mg 3 times daily or 75 mg twice daily | Increase to 300 mg daily after 3–7 d, then by 150 mg/d every 3–7 d, as tolerated, until pain relief | 600 mg daily (200 mg 3 times daily or 300 mg twice daily); reduce if impaired renal function | 4 wk | Sedation, dizziness, peripheral oedema | Renal insufficiency | Improvement of sleep disturbance, improvement of anxiety, no clinically significant drug interactions |
| Topical lidocaine patch    | 5% lidocaine    | Maximum of 3 patches daily for a maximum of 12 h | None needed | Maximum of 3 patches daily for a maximum of 12–18 h | 3 wk | Local erythema, rash | None | No systemic side effects |
| Opioid agonists            | Morphine, oxycodone, methadone, levorphanol | 10–15 mg morphine every 4 h or as needed (equianalgesic dosages should be used for other opioid analgesics) | After 1–2 wk, convert total daily dosage to long-acting opioid analgesic and continue short-acting medication as needed | No maximum dosage with careful titration; consider evaluation by pain specialist at relatively high dosages (eg, 120–180 mg morphine daily; equianalgesic dosages should be used for other opioid analgesics) | 4–6 wk | Nausea/vomiting, constipation, drowsiness, dizziness, seizures | History of substance abuse, suicide risk, driving impairment during treatment initiation | Rapid onset of analgesic Benefit |
|                           | Tramadol        | 50 mg once or twice daily | Increase by 50–100 mg daily in divided doses every 3–7 d, as tolerated, until pain relief | 400 mg daily (100 mg 4 times daily); in patients aged >75 y, 300 mg daily | 4 wk | Nausea/vomiting, constipation, drowsiness, dizziness, seizures | History of substance abuse, suicide risk, driving impairment during treatment initiation, seizure disorder, concomitant use of SSRI, SSNRI, TCA | Rapid onset of analgesic benefit |

### Table 1: Prescribing recommendations for first-line medications and for opioid agonists

Medication and dosage recommendations for pain management include:

- **Secondary amine TCAs** (Nortriptyline, Desipramine): Start with 25 mg at bedtime, increase by 25 mg daily every 3–7 days as tolerated, until pain relief is achieved.
  - Maximum dosage: 150 mg daily.
  - Duration of adequate trial: 6–8 weeks.
  - Major side effects: Sedation, dry mouth, blurred vision, weight gain, urinary retention.
  - Precautions: Cardiac disease, glaucoma, suicide risk, seizure disorder, concomitant use of tramadol.
  - Other benefits: Improvement of depression, improvement of insomnia, low cost.

- **SSNRIs** (Duloxetine, Venlafaxine): Start with 30 mg once daily, increase to 60 mg once daily after 1 week.
  - Maximum dosage: 60 mg twice daily.
  - Duration of adequate trial: 4 weeks.
  - Major side effects: Nausea.
  - Precautions: Hepatic dysfunction, renal insufficiency, alcohol abuse, concomitant use of tramadol.
  - Other benefits: Improvement of depression.

- **Calcium channel α 2 -δ ligands** (Gabapentin, Pregabalin): Start with 50 mg 3 times daily or 75 mg twice daily, increase to 300 mg daily after 3–7 days, then by 150 mg/d every 3–7 days as tolerated, until pain relief is achieved.
  - Maximum dosage: 3600 mg daily (1200 mg 3 times daily).
  - Duration of adequate trial: 3–8 weeks for titration + 2 weeks at maximum dose.
  - Major side effects: Sedation, dizziness, peripheral oedema.
  - Precautions: Renal insufficiency.
  - Other benefits: Improvement of sleep disturbance, no clinically significant drug interactions.

- **Topical lidocaine** (5% lidocaine patch): Maximum of 3 patches daily for a maximum of 12 hours.
  - Duration of adequate trial: 3 weeks.
  - Major side effects: Local erythema, rash.
  - Precautions: None.
  - Other benefits: No systemic side effects.

- **Opioid agonists** (Morphine, oxycodone, methadone, levorphanol): Start with 10–15 mg morphine every 4 hours or as needed (equianalgesic dosages should be used for other opioid analgesics).
  - Duration of adequate trial: 4–6 weeks.
  - Major side effects: Nausea/vomiting, constipation, drowsiness, dizziness, seizures.
  - Precautions: History of substance abuse, suicide risk, driving impairment during treatment initiation.
  - Other benefits: Rapid onset of analgesic Benefit.

- **Tramadol**: Start with 50 mg once or twice daily, increase by 50–100 mg daily in divided doses every 3–7 days as tolerated, until pain relief is achieved.
  - Maximum dosage: 400 mg daily (100 mg 4 times daily).
  - Duration of adequate trial: 4 weeks.
  - Major side effects: Nausea/vomiting, constipation, drowsiness, dizziness, seizures.
  - Precautions: History of substance abuse, suicide risk, driving impairment during treatment initiation, seizure disorder, concomitant use of SSRI, SSNRI, TCA.
  - Other benefits: Rapid onset of analgesic Benefit.
The DSM criteria for panic disorder (PD) include somatic symptoms (e.g. increased heart rate, dizziness) and cognitive symptoms (e.g. fear of dying). A further set of criteria emphasises worry about future panic attacks, also the consequences of having an attack (e.g. having a heart attack), and significant behavioural changes in response to the attacks (i.e. avoidance of any situation or place which could possibly trigger another attack).

Many individuals struggle with PD for up to 10 years before finding the correct treatment. As a result, they can often predict when an attack will occur, and then actively avoid triggers. This cycle of avoidance starts to impact more and more on what can be done or where someone can ‘safely’ go, without an attack happening, and this has an increasingly negative impact on functioning or freedom of movement. For example, if an attack happened even just once when out at a restaurant, the person begins to avoid any type of social situation which involves a public place or eating with a group.

Even if there are no more attacks, or they have far less severe symptoms· typically when medication has started to have an effect, there is still a cognitive cycle where worry about having another attack and worry about the possible consequences of such an attack (as mentioned in the opening paragraph of this article) becomes a defining feature of the disorder. This is sometimes called a ‘fear of fear’ cycle, and leads to more and more avoidance of potential triggers, based largely on the possibility of an attack· i.e. the ‘maybe’ more than the ‘likely.’

Typical catastrophic thoughts in PD are: fear of death or disability (I’m having a stroke/heart attack, I can’t breathe), fear of losing control or going crazy (I’m going to lose control and jump out of the car/window, I’m going to scream without being able to control it, I need to escape or I will definitely go insane), and fears of humiliation or embarrassment (people will think I’m stupid/weak/something is really wrong with me).

Often, people with panic disorder can recognize how unrealistic their fears or predictions are when they are in a calm state, and usually not in one of the situations or places they worry about. When they are in the middle of a panic attack, though, the thoughts “feel true” and other explanations for their symptoms are not considered. In addition, remembering how awful and how frightening the symptoms were, is often enough to lead to behavioural avoidance due to anticipation of what another attack would be like. Recall and anticipation also tends to occur in a biased manner (i.e. recalling and fearing the symptoms described previously, and not that they ’survived’ them or that the intensity and frequency of panic attacks has reduced in many cases), which reinforces the fear of another attack occurring, also that it would be catastrophic.

A further component of the cognitive model of panic, is that the person becomes hyperaware and hypervigilant around physical sensations or changes. Even subtle symptoms are noticed, then interpreted catastrophically (i.e. this will definitely become another attack, what if I don’t get through this one! It’s going to be worse!). This type of thought leads to activation of the fight/flight system, which entails a release of adrenalin and this feeling is then again interpreted catastrophically, leading to further release of adrenalin. In this way, the thoughts of catastrophe are reinforced, and the person has further reason to fear even thinking about a place or context that could trigger another attack, and certainly wants to avoid going into such a situation at all.

This is a catch 22 situation - thoughts such as “am I feeling any symptoms?” and checking for any sort of symptom, tend to bring on the very thing the person fears. It’s ironic that one has to think about something to unthink it- for example, if I said that you could win a large sum of money if you DIDN’T think about, say, pink elephants, the immediate cognitive response would likely be “don’t think pink elephants... don’t think pink elephants... don’t think pink elephants!”
Cognitive behaviour therapy (CBT) for panic disorder targets three main areas: the physical symptoms and how to desensitise to them, the catastrophic thoughts which occur before and after an attack, as well as the worry/anticipatory anxiety (fear of fear cycle), and the behavioural symptoms such as avoidance of activities, places and people where a panic attack may be triggered (agoraphobia). The rest of this article will elaborate on the latter two components - how they are inter-linked and how to treat them. A case study (used with permission, and identifying details changed) will illustrate a typical example of how this occurs.

T, a 32 year old woman who had been a stay home mom since the birth of her second child, presented with severe panic symptoms and agoraphobia for crowds of any kind. On taking a full history, the main points relevant to this article were: the first panic attack happened 2 years earlier, when T had been rushing to a mall to buy food for her children, aged 1 and 2 at the time. She had not eaten that day, there was heavy traffic on the road, and the children were both in the car with her, in her recall- “very niggly.” She recalled thinking that she had to get to the mall very quickly as the children were starving and would start to scream soon, also that she was a bad mother for having run out of supplies (her husband travelled frequently, so she was handling the home, both children, and without help. Her mother was away visiting her other daughter, abroad, for 2 months).

Regarding family history, her father had a moderate degree of generalised anxiety, no one else in the family had panic, and she had not had worry or other features of generalised anxiety in the past. There was no other anxiety (such as social phobia) or depression present, though she had started to worry about the effect on her children and these thoughts were beginning to consume her. She had only partly responded to an SSRI (she was very sensitive to side-effects, and when someone has panic they are hyperaware of even slight unusual or uncomfortable physical symptoms, which highlights the experience of any side-effects).

We began with psycho-education (explaining what panic is, why it is happening, and reality testing - that it is not a threat to survival or risk for insanity). This is actually the start of cognitive restructuring, as knowledge definitely is power. T felt much more comfortable knowing that she was not at ‘real’ (life-threatening) risk during a panic attack, but still feared the actual sensations of an attack, and had some thoughts around “what if the next one is worse and I can’t stop it.” The next intervention (interoceptive deconditioning) entailed desensitising to her specific symptoms, and this was followed by addressing her worry/anticipatory thoughts and reality testing the validity of each component making up a set of beliefs - about what the symptoms meant, whether there was any basis for a future attack being worse (for no reason other than a ‘maybe,’ unfounded in factual evidence). Once these parts were complete we devised a detailed ‘avoidance hierarchy’, listing each variation of crowd and place where anxiety symptoms could be triggered, and then working through the beliefs and fears around each one. For example - “if I am in a shopping mall, I won’t be able to get to the exit before I faint/start screaming/go crazy” and “people will think I’m a drug addict and will not help me” were reality tested by asking questions such as: have you been able to speak/move when having a panic attack in the past? Who noticed? How did they react? What did they think was happening to you?” In this way, T was able to actively confront the fears, in a safe setting, and come up with alternate beliefs that she could keep in mind before going into the actual situation.

The thoughts and beliefs underlying the avoidance must be addressed one by one, for exposure to be successful. This allows for an opportunity to change the self-talk which would occur during a feared situation, also to rehearse being in the situation but in a safer way. Done enough times, the person would start to desensitise to the fear thoughts, which

This case illustrates how important it is to address every fear belief there could be, before going into the actual exposure. This is done collaboratively and respectfully, never demeaning the client’s fear even if it is irrational in nature.
is what happened here. T took her mother with her for the first few exposures, and her mother reminded her of the reality tested thoughts - e.g. “remember, these feelings are real but the alarm is false”; and “you managed to go into shops for many years without feeling anxious, so anxiety is not inherent in you but a learned reaction.” Her mother was coached in a session before the exposure, also in what not to say or do - i.e. not to distract or to end the exposure when the first symptom of discomfort began.

T was able to wait out the anxiety symptoms, without catastrophising them, and was very optimistic about her progress. She worked through the full hierarchy with her mother, some items alone, and then did an exposure on a Saturday morning at month-end, when the mall was very busy, and did have some thoughts around “this is too much, I’m trapped, there are too many people around me.” She had gone to the mall with the toddlers, but no adult to assist. This led to her leaving the mall, but she sat in her car for a few minutes, reality tested - anyone would feel pressured with a busy mall and two busy toddlers, and these symptoms are actually quite understandable in this situation. She then returned to the mall and walked through for a half hour, not pressuring herself by being ‘trapped’ in a checkout line, but remaining in the mall nonetheless. Subsequently she built up a ‘mastery’ database of experiences in her mind, to such an extent that when she redid this exposure she was able to reality test on the spot and was able to remain in the mall as well as complete her shopping, while riding out the symptoms until they faded away.

T has returned for booster sessions, initially every 3 months and then annually, but she is now the one reality testing and reporting her successes, rather than needing the therapist to do this function for or with her.

This case illustrates how important it is to address every fear belief there could be, before going into the actual exposure. This is done collaboratively and respectfully, never demeaning the client’s fear even if it is irrational in nature. The fear of fear cycle can be broken in this way, and this is a form of imaginal exposure, long before the person actually gets into the actual situation. To quote Shakespeare (as one of many who have been cited as the originator of these words): “There is nothing either good or bad, but thinking (interpretation) makes it so.”

References available on request.
Vocational Rehabilitation, Work Hardening and Case Management for Clients with Mental Health Challenges

With the World Health Organisation predicting that depression will be the second highest cause of morbidity in the world by 2020, employers cannot afford to bury their heads in the sand and hope for the best. Some companies have also established Employee Assistance Programmes (EAPs) to support employees dealing with issues that impact on mental health. To improve the efficacy of these programmes, appropriate linkages between EAPs and other interventions are important.

Mental health related disability is rapidly increasing with depression emerging as the most common mental disorder in the workplace. “Many people with mental health problems fear that, no matter how good a recovery they have made, their symptoms will be made worse by going back to work.”

Treatment is available to alleviate symptoms and functional impacts; however, it is not specifically aimed at returning people to work.

Work plays a significant role in how people identify themselves. Successful return to work is associated with significant psychological and rehabilitative benefits which include improved mental health, opportunities to enhance self esteem and improved quality of life.

The Challenge
Psychiatric disorders as a cause of occupational disability are under recognised and under-treated worldwide. In South Africa there has been an alarming increase in applications for medical disability on psychiatric grounds, related in many cases to socio-political changes, and psychiatric disorders have taken over from musculoskeletal conditions, particularly lower back pain, as the leading cause of disability.

As occupational therapists working in acute psychiatry, our services are restricted mostly to in-hospital assessment and treatment or for as long as medical aids provide funding. Clients who apply for temporary disability often remain untreated during the waiting period and temporary disability leave, as they already have depleted their medical aid benefits and may only receive a portion of their salary or nothing at all.

This becomes a barrier to return to work and recovery as clients do not access therapy aimed at returning them to work, but rather access treatment to manage their acute symptoms, which include psychotropic medications, psychotherapy, and psychological support. These treatments can be effective in helping to alleviate symptoms and functional impacts, but are not, however, specifically aimed at preparing the person to return to work. The dilemma is that the longer people stay away from work due to disability/impairment, the harder it gets to return to work.

Return to Work Rates

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A review of the current literature suggests a direct relationship between an early referral to rehabilitation and a successful return to work programme.

The longer a person is off sick, the more difficult it becomes for them to return to work and the less likely it is that they will return to work at all. In part, this is because many people with mental health problems fear that, no matter how good a recovery they have made, their symptoms will be made worse by going back to work. This is especially so for those who believe that work has either caused their health problem or made it worse.

The Role of Cognition
The disability and burden associated with major depression comes only in part from its affective symptoms; cognitive dysfunctions associated with depression play a crucial role. Elementary and more complex cognitive processes are equally affected during depression. It is important to recognise the cognitive dysfunction, which may include: poor verbal and visual short and long term memory, impaired executive function, impaired psychomotor skills and poor attention.

Persistent cognitive dysfunction can decrease coping capacities, influence therapeutic response and compliance, impact on risk of relapse, and limit daily functioning and quality of life.

Cognitive dysfunctions are significant predictors for therapeutic response, daily function and also play a prominent role in residual symptoms.

Cognition therefore, becomes a key target in the treatment of depression. Interventions aimed at improving cognition decreases symptoms but also the disability associated with depression.

There is a consistent relationship between cognitive dysfunction and difficulties with employability.

Bridging the Gap
Work has many health benefits and physical and mental health is generally improved through work. People also recover from sickness quicker and are at less risk of long term illness and incapacity when they are at work. Because of these benefits, sick and disabled people are encouraged to return to, or remain in, work if their health condition permits it. As occupational therapists working in acute psychiatry, we have an obligation to guide our clients to either retain or explore alternative employment, as we are fully aware of the benefits of being employed.

Assisting in swift and timeous return to work is therefore an important part in recovery. It thus makes sense to engage suitable clients in return to work activities as soon as possible that will contribute to return to work.

Presenting Dysfunction
- Any client that gets booked off work for longer than 2 weeks
- Employees identified as poor work performers
- Clients that present with cognitive impairments:
  - Decreased ability to concentrate and pay attention.
  - Difficulty in completing a task
  - Difficulty pacing tasks
  - Memory difficulties
  - Lack of current skill set
  - Difficulty with decision making
  - Difficulty meeting reasonable deadlines
  - Lack of daily routine
- Clients that present with emotional and social impairments:
  - Lack of self esteem and self confidence
  - Avoid social interaction and work place activities
  - Poor interpersonal relationships at work
Why Occupational Therapy?

- Occupational therapists are very well suited to address issues in the workplace
- Occupational therapists are trained in the pathology of mental illness and its impact on functioning
- Occupational therapy services increase the probability of returning to work in good health and with better work and role functioning
- Occupational therapy services (work skills training) increase chances of integration into chosen work environment
- Occupational therapy services result in a reduction in work-loss days and have a 75.5% probability of being more cost-effective than treatment as usual alone

Services: Cognitive Work Hardening, FCE’s and Case Management

Cognitive work hardening is a highly structured, goal orientated, individualised treatment programme designed to maximise the individuals’ ability to return to work. It aims to improve the client’s work performance skills to enable the safe and productive return to the work force. It includes analysis of the client’s job, understanding the impact of the environment and recommending reasonable accommodations/modifications to job tasks.

A functional capacity evaluation (FCE) involves obtaining, interpreting and documenting data about the functional status of the individual. The purpose of the assessment is to identify the client’s abilities and limitations and to match or adapt these to the requirements of the job. Data is obtained through a structured interview, standardised and non-standardised assessments, questionnaires and collateral information. Sometimes a work visit is needed.

Case management involves coordinating the return to work process and liaising with all stakeholders. It also involves working collaboratively with the employee and employer to identify and address work-related functional limitations or restrictions prior to and during a work trial. The ideal outcome is to find appropriate reasonable accommodations to assist the employee to return to work safely and timeously. Reasonable accommodations may include an alternative position in the current workplace, managing workload more effectively, graded return to work in terms of hours or workload, working flexi-time, etc.

Benefits of Early Return to Work

Client benefits:
- Greater sense of purpose and identity
- Having a sense of security and stability
- Retaining full earning capacity
- Avoiding dependence on a disability benefit
- Greater sense of well-being
- Assisting in preventing relapse
- Sense of belonging
- Faster recovery
- Maintaining a productive mind set
- Staying on a regular work schedule

Insurer benefits:
- Initial investment may prevent long term disability leading to
  - Financial benefits
  - Less paperwork
- Less claims or claims for a shorter period
- Anticipating and controlling hidden costs

Employer benefits:
- Retain skilled employees
- Avoiding cost of retraining, recruiting or hiring of new staff
- Positive workplace culture
- Increased productivity
- Improved ability to manage a disability claim and any restrictions
- Anticipating and controlling hidden costs

Conclusion

“This (cognitive effects of depression) is one of the reasons why its vital to examine how depression is managed in the workplace and what procedures are in place to ensure that affected employees are encouraged to and supported in seeking treatment” - Casey Chambers, Director SADAG.

References available on request.