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In This Issue:

Editorial

Editor - Vicki Evans

This edition sees many articles from local sources as well as overseas contributors. The first article is from the United States and discusses the special considerations when caring for the growing population of elderly trauma patients, focusing on their aged-related changes and co-morbid conditions.

From the UK, this paper describes apathy as a significant neuro-behavioural condition following TBI. Documented as common amongst many neurological disorders, apathy is still not well understood, nor described in the literature. A multidisciplinary team approach is required with an emphasis on the importance of being motivated for maximum recovery.

From Japan, implementation and education strategies for the disposal of material from patients undergoing Temozolomide treatment is described, with special note to exposure from the oral antineoplastic agents.

Anti-N-methyl-D-aspartic acid (anti-NMDA) Receptor Encephalitis is a devastating, complex disease that is eloquently described here. Like most things neuro, early recognition and rehabilitation is essential.

There are two articles relating to Parkinson's Disease, describing the important role that PD Nurse Specialists play in caring for these patients. Discussion around freezing-of-gait and future treatments for PD is also presented.

The sinking skin flap is a refreshing article describing a complication following decompressive craniectomy in which the skin flap sinks down causing direct pressure on the cortex and hence, neurological symptoms.

Finally, the process of implementation of a falls system is described. There are many relevant outcomes from this paper that can be implemented across all areas.

Our Guest Editorial captures the essence of neuroscience nursing—the teaching of others in a caring and compassionate manner in which the future of the specialty can flourish.

Cheers, *Vicki*

Guest Editorial

Ashleigh Tracey

I am honoured to be a part of ANNA's guest editorial for October. Being a neuroscience ICU nurse for the past ten years, I have had the pleasure of working with some of the most talented and knowledgeable nurses, who have endlessly supported the growth of new nurses entering the unit because in Neuroscience you don't just sit on the sideline!

Walking into the Neuroscience ICU on my first day as a 20yr old new graduate was a daunting, terrifying yet exhilarating experience. I was preceptored with a CNS who I admire to this day. Her clinical knowledge surpassed even the most confident of neurosurgical registrar and who I watched skillfully manage the most complex neurosurgical patients with a beautiful sense of ease and control, whilst having the ability to guide and support families at every turn. For her, not only was the patient her priority, but so too was my learning opportunities. I had the opportunity to practice and refine assessment techniques, practical skills and the general nursing 'tricks of the trade' needed to competently and safely manage neuroscience patients. Whilst I strongly believe classroom learning is the underpinning of strong theories and concepts, for me as a new neuroscience nurse, watching my colleagues' practice at the bedside and to have been allowed to learn in a safe, non-threatening clinical environment has resonated the strongest with me, and why I advocate this for all nurses entering this highly specialised and intricate field.

Clinical teaching and educational opportunities however, can prove difficult in some situations and are not without their challenges. Floor plan layouts including single rooms, increased staffing demands and increased patient acuity all play a role in limiting opportunities for clinical bedside teaching. There is an increased pressure placed on nurses due to overwork and stress, which is further compounded by the need to maintain clinical standards and patient safety.

A preceptorship model we are currently using, pairs new nurses with skilled experienced neuroscience nurses and allows both nurses

to work the same 12 hour day and night shifts, from day one. This allows for continuity when requiring a resource person and provides the new nurses with an opportunity to practise and develop assessment skills and techniques in a safe, familiar environment. Added to this, practical application of skills assists with the consolidation of knowledge learnt at university and allows for constructive feedback and critical reflection, all essential components in improving professional development.

Neuroscience Intensive Care is an environment rich in learning and professional growth, and where the disciplines of critical care and neuroscience naturally co-exist. Whilst we need to maintain our focus on developing nurses to encompass both specialties, we need to encourage the professional qualification of neuroscience to ensure the continual development of highly skilled neuroscience nurses.

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Trauma and the Elderly: The Real Picture of Their Health May be Blurred

Mary L. King

Abstract

The growing elderly population is impacting health care world-wide. Studies have shown that people over age 65 years have an increased risk of death, even with injuries that may be considered 'minor'. Overall survival rates in the elderly due to trauma are improving with advances in medical care; however, the incidence of co-morbidities are high in this age group thereby increasing lengths of stay, risk of complications, and the disproportionate amount of medical resources when compared to younger patients. While prompt and aggressive treatment of the elderly trauma patient decreases mortality, clinicians need to be aware that age-related insufficient physical reserves may mask underlying conditions and shock states that are difficult to identify. This article will discuss the hazards related to the elderly trauma patient and special considerations for health care professionals.

Keywords: *elderly, trauma, base deficit, physical reserves*

Introduction

Aging

The natural progression of aging is an expected decline in multiple body systems. As these changes occur slowly over time, they may not be evident until the elderly person becomes ill or injured. Decreases in brain mass, cardiopulmonary function, gastric motility, renal function, and impaired blood flow are among the major systems affected with age. Degeneration of joints, decreased senses of sight, smell, taste, and hearing, as well as neuropathies, loss of skin elasticity/turgor, and an approximate 15 to 30% loss of total body fat contribute to the development of complications with traumatic injury (Clement, Tennant, & Muwanga, 2010).

In the United States (U.S.) in 2006, the number of elderly (age 65 years and older) was approximately 37 million, or about 12% of the overall population. By the year 2020, the number of elderly is expected to double (Weber, Jablonski, & Penrod, 2010). Data from the World Health Organization (WHO) mirrors that of the U.S. showing a global increase in the elderly population with injuries related to motor vehicle accidents, violence, and falls as the leading causes of death (Sheetz, 2010). Among the elderly involved in motor vehicle crashes (MVC), blunt chest injury is a frequent pattern of injury. In addition to this, the elderly also suffer a significant number of potentially fatal conditions such as spinal cord and thorax injuries or intracranial hemorrhage (Sheetz, 2010).

In patients over age 65 years with head injury, a computed tomography scan (CT scan) is recommended to improve diagnosis and outcomes (Weber, Jablonski, & Penrod, 2010). Osteoporosis and degenerative joint disease predispose the elderly to fractures, specifically cervical spine fractures. Plain films are usually not adequate for identifying a cervical fracture in the elderly due to pre-existing degenerative changes in the vertebrae that may mask high cervical (C1, C2) fractures. A CT scan of the head and neck provide better visualisation of the bony and soft tissue structures making diagnosis more accurate (Weber, Jablonski, & Penrod, 2010).

There is no simple formula to determine the rate of deterioration in the elderly population. Each person is unique in their aging process and their response to illness or injury. The functional status of older adults contributes to their ability to survive an injury. Elders with high functional ability may have more physical reserves or resiliency to recover from an inju-

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ry that is not a reflection of their chronological age (Min, Ubhayakar, Saliba, Kelley-Quon, Morley, Hiatt, Cryer, & Rillou, 2011). Nurses are faced with the challenges associated with caring for the elderly population and must be aware that 'normal' is not always the true picture of the elderly patients' health. Physiologic age-related changes result in a decreased ability to effectively compensate for effects on body systems caused by the trauma; thus the laboratory values and vital signs in the elderly patient may reflect seemingly normal values when the underlying picture is that of low oxygenation, poor cardiac output, and an overall unstable condition (Sheetz, 2010).

Blurred Data

The under-detection of trauma in the elderly may be related to the use of trauma guidelines that may not accurately identify the extent of injury resulting in an inappropriate transport of the elderly patient to a non-trauma center. Standard Advanced Trauma Life Support (ATLS) protocols pertain to the elderly adult, but with precautions. This is of particular concern to nurses in the emergency department who may receive a "stable" elderly patient only to have the patient quickly deteriorate on arrival due to their injuries and co-morbid conditions. The elderly with diabetes, for example, may have neuropathies and sensory deficits that affect accurate assessment (Neville, Nemtsey, Manasrah, Bricker, & Brant, 2011).

The elderly patient is very vulnerable to respiratory compromise. One or two rib fractures may have significant implications and healthcare providers should have a high suspicion for chest injury with the so-called 'minor' falls. Half of the elderly population who experience a ground-level fall will suffer rib fractures resulting in respiratory distress that is complicated by the effects of aging as evidenced by a loss of alveolar volume and atrophy of the chest wall and muscles (Farhat, Velanovich, Falvo, Horst, & Swartz, 2012). These elderly patients need early intervention and airway management is critical. If the patient is not able to take deep breaths, has low oxygen saturations (≤ 90%), early intubation should be considered. The consequence of intubating an elderly patient with co-morbidities of heart failure or chronic obstructive pulmonary disease (COPD) may be the inability to wean from mechanical ventilation or extubation (Farhat, et al., 2012).

Blood pressure is one of the parameters under consideration with regard to the elderly population. In general, a systolic blood pressure (SBP) value of less than 90 mmHg is considered hypotensive in a normal adult. Recent discussions are suggesting that a higher SBP of 110 - 117 mmHg may be a better definition of hypotension for patients over age 65 years (Oyetunji, Chang, Crompton, Greene, Efron, Haut, et al., 2011). One study found that one-third of the elderly patients with normal blood pressure and considered 'stable' died within 24 hours of cardiac arrest due to age-related cardiac insufficiency as a result of hypo-perfusion and higher peripheral vascular resistance (Oyetunji et al., 2011).

This age-related insufficiency in cardiac output and hypo-perfusion results in a lower heart rate and masks signs and symptoms of metabolic deficiencies thereby making shock more difficult to diagnose (Chang, Tsai, Su, Huang, Chang, Tsai, 2008). Research has demonstrated a tendency to under-compensate in shock states in the elderly resulting in insufficient oxygen delivery and 'oxygen debt' resulting in higher mortality and poor outcomes (Chang et al., 2008).

Elderly trauma patients with higher peripheral vascular resistance and lower cardiac output had significantly higher mortality rates than younger adults or the elderly patient with higher peripheral vascular resistance without a lower cardiac output. The uninjured elderly patient is able to compensate and remain stable at rest, but the addition of trauma greatly complicates the picture as their physiologic reserves are drained and their ability to tolerate the stress of the injury is depleted (Oyetunji et al., 2011).

The usual markers, arterial base deficit and serum lactate levels, are helpful in determining the degree of shock in the elderly and if the resuscitation efforts were adequate. Base deficit is the amount of base (in mmol) that is needed to bring a liter of whole arterial blood to a pH of 7.40 and is considered a standard assessment of metabolic acidosis. Hypotension and hemorrhage is common in trauma patients and the degree of metabolic acidosis on arrival to the emergency department (ED) often provides a means to predict outcomes (Chawla, Nader, Nelson, Govindji, Wilson, Szlyk, et al., 2010). Base deficit is also considered a marker for severity of illness. When oxygen delivery is inadequate

and the body converts to anaerobic metabolism the result is an excess amount of lactate and hydrogen ions which combine to create lactic acid in the extracellular fluid. Monitoring serum lactate levels during resuscitation may be helpful in determining the effectiveness of the interventions as decreasing lactate levels are predictive of improved survival rates (Wilson, Grande, & Hoyt, 2013). Since hypoperfusion is often imperceptible in the elderly, examining serum markers of tissue oxygen deficit (lactic acid and base deficit) can help to detect underlying problems. Lactic acid and base deficit are among the measures employed to predict mortality in the elderly patient with blunt trauma but who appears stable with a 'normal' blood pressure (London, 2011).

Although the range of normal values may be the same for the elderly patient as in the younger patient, the mortality rates are higher. For example, the elderly trauma patient with a base deficit over -10 had a mortality rate of 80% and those with base deficits in the -6 to -9 or moderate range had a 60% mortality rate (Chang et al., 2008). An elevated serum lactate level is a factor in occult hypo-perfusion with the clearance rate directly associated with mortality as the elderly patient is less able to compensate. A lactic acid level greater than 22 mg/dl (or greater than 2.4 mmol/L) for longer than 12 hours is also associated with high mortality (Chang et al., 2008). Thus it is important to remember that specificity is more important than sensitivity; if the serum markers are positive, the test is likely an accurate depiction of the patients' condition. However, if the serum markers are negative, it may only indicate the elderly patient is in an early phase of decline and immediate interventions are required (London, 2011).

Changes in the tissues and organs of an elderly patient can mask injuries that may be more evident in the younger patient. For example, thinner eardrums and a decline in hearing may prevent timely diagnosis of a ruptured eardrum. Changes in membranes within the eye and diminished vision may delay identification of injuries to the elderly persons' eyes (Catananti & Gambassi, 2010). Abdominal pain may not be felt as acutely due to an age-related decrease in sensitivity of pain receptors. The elderly patient may have a reduction in the acute nociceptive sensation from the deep abdominal structures. Clinicians may recognise and treat an underlying shock, but damage to the intestinal wall or organs may not be evident for a long time and will be more life-threatening (Catananti & Gambassi, 2010).

The elderly patient is more likely to be on multiple medications (polypharmacy) especially those that are prescribed to treat cardiopulmonary conditions. Medications such as beta-blockers have the potential to delay rapid identification of problems as they may cover up a loss of physical reserves or hypoperfusion making diagnosis and treatment more complicated. Often the elderly patient is on an anticoagulant medication, i.e. warfarin or aspirin that increases their risk of haemorrhage resulting in a necessary blood transfusion which further increases morbidity and mortality (Soles & Tornetta, 2011).

Pain management is a challenge as the elderly patient is often sensitive to opioids and due to age-related decline in renal function, clearance from the kidneys is slowed. A common caveat for treating pain in the elderly is 'start low and go slow' to mitigate potential complications of respiratory depression and over-sedation. Elderly patients can be safely treated with opioid medications as long as health care providers order appropriately for age and physical condition (Weber, Jablonski, & Penrod, 2010).

Elderly Scoring

Research has been aimed at identifying the vulnerable elderly patients in order to focus interventions to reduce complications and promote better use of resources. The strongest predictors of survival have shown to be age, sex, co-morbid conditions, severity of injury, and vital signs. One tool that has been used is the Injury Severity Scoring (ISS) that measures the overall severity of injury in multiple body areas; however, it should be noted that some trauma associations have not recommended the ISS as a triage tool as the full extent of the patients' injuries are often unknown (Min et al., 2011).

Another study used the Vulnerable Elders Survey-13 (VES-13) as a determinant of complications and mortality in the injured elderly. This survey has been validated in the uninjured elderly population to predict the risk of functional decline and death in acute care and ambulatory settings. One of the benefits of this survey is ease-of-use. A non-clinical person, i.e. caregiver can answer the survey questions via telephone, if necessary. The

survey may be completed by a medical or nursing professional, or a non-licensed staff member without prior knowledge of the persons' pre-existing conditions. The VES-13 is based on age and functional status and uses a point-scoring system for age, activities of daily living (ADLs), common physical tasks, and self-rated health status (Min et al., 2011). This survey defines disability when the individual requires help or is unable to perform the activity due to health reasons. Among the items included in the survey are shopping, light housework, managing money, lifting 10 pounds (4.5 kg), reaching above shoulder level, and writing. The self-rated health of the individual is compared with other persons of similar age. The study using the VES-13 with injured elderly patients found a potential use existed to predict complications in the hospitalised elderly adult suffering from traumatic injuries when used with the ISS (Min et al., 2011).

Other studies with elderly trauma patients (age 65 years or older) found the incidence of complications was similar to younger trauma patients, but noted a trend in patients age 45 years or older and an increased risk of complications and mortality (Adams et al., 2012). When the researchers examined lengths of stay, end-organ failure, coagulopathies, and incidence of infectious complications there was a distinct difference between those patients younger than age 45 years and patients age 45 years and older. Interestingly, infectious complications seemed to peak around age 65 years, then decline; however urinary tract infections (UTI) increased in the patients who are 74-85 years old. In this age group it is difficult to diagnose infection due to atypical presentation such as confusion as a presenting symptom instead of fever (Adams et al., 2012).

Healthcare Considerations

Given the multiple studies on elderly trauma patients that demonstrate higher complication and mortality rates the questions arise whether the elderly patient should be as aggressively treated as the younger (less than age 45 years) population. Research on elderly trauma patients has suggested that use of the ISS should be considered for pre-existing disease processes. When complications are anticipated, the elderly trauma patient should receive aggressive care as there is evidence that a reasonable recovery is possible (Bar-Or, Salottolo, Orlando, Mains, Bourg, & Offner, et al., 2013).

Invasive monitoring devices may help to diagnose occult hypoperfusion states/shock and reduce end-organ failure. The use of pulmonary artery catheters (PACs) has decreased in the past few years; however, in the elderly trauma patient hemodynamic monitoring allows for direct measurement of cardiac function (London, 2011). Evidence has shown that outcomes are improved with a targeted approach to optimizing cardiac function and oxygen consumption. Early interventions, i.e. admission to an intensive care unit (ICU), goal-directed resuscitation, and accurate monitoring are recommended to improve survival rates (London, 2011).

Summary

Care of the elderly trauma patient is a challenge given the age-related changes and the interaction of their co-morbid conditions and medications with their injuries. Medical and nursing interventions aimed at resuscitation and stabilization of the elderly patient should be implemented aggressively and in a similar manner to younger patients. Evaluation of laboratory values, specifically lactic acid and base deficit are critical in the early stages of resuscitation and may assist the healthcare team to more accurately determine the patient's condition. What may be considered 'normal' vital signs and laboratory values in a younger patient are not necessarily 'normal' when the patient is age 65 years or older. Physical reserves are easily depleted and changes in pain perception, cardiac function, vision, and hearing may be further complicated with the addition of medications such as beta-blockers or an anticoagulant, i.e. warfarin. Elderly trauma patients have unique needs that require expert knowledge and skills to improve outcomes. An awareness of the subtly in which the elderly trauma patient presents and the motivation to 'dig deeper' when reviewing laboratory and radiology results, and accurate (and frequent) reassessment will offer the elderly patient the best chance for a meaningful recovery. The elderly trauma patient may present with a stable, but blurred picture of health but with an understanding of the special considerations for the elderly trauma patient the healthcare team can bring that picture into focus.

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Apathy a common feature in many neurological conditions: care and management

Mary E. Braine

Abstract

Apathy is associated with many neurological conditions including traumatic brain injury (TBI), stroke, Parkinson's disease and dementia and is primarily a dysfunction of the frontal-subcortical circuit. Despite being common, it is often under recognised, not viewed as problematic, or misdiagnosed as depression. Various definitions of apathy are offered in the literature along with various assessment tools and prevalence amongst various neurological conditions. This paper discusses the diagnosis criteria along with the importance of an accurate and timely assessment in aiding healthcare practitioners in caring for patients with apathy, and can be a useful starting point for rehabilitative efforts. Apathy can be profoundly disabling for both the patient and their caregivers, and is associated with a number of adverse outcomes such as a negative impact on daily life and quality of life, potential for poorer rehabilitation outcomes and increased burden on caregivers. Areas for further research and development are also presented.

Keywords: *Apathy, assessment, goal directed behaviours, traumatic brain injury, Parkinson's disease, stroke, Alzheimer's disease.*

Introduction

Apathy is an important, debilitating and distressing behavioural symptom that occurs in many neurological conditions and neuropsychiatric disorders. Despite its frequency, apathy is often unrecognised, untreated and misdiagnosed and not viewed as a problem. Apathy may also occur in association with a variety of other clinical problems and may complicate both assessment and treatment (Marin, 1990). Apathy is a significant and common neurobehavioural sequela following traumatic brain injury (Kant, Duffy & Pivovarnik, 1998) and is a common feature of subcortical pathologies such as Parkinson's disease, Huntington's chorea and Progressive Supranuclear Palsy (PSP). It is also a common behavioural disturbance following stroke (Mayo, Fellows, Scott. Cameron & Wood-Dauphinee, 2009) and in neurodegenerative pathologies such as dementia (Mendez, Lauterbach & Sampson, 2008). Despite apathy being prominent in many neurological and psychiatric disorders, there is a paucity of nursing literature on the care and management for a patient with apathy. This is surprising not only because of its high prevalence in many neurological conditions but because of the negative impact on both the patient and their families and carers. This paper will discuss the various definitions and criteria of apathy, describe assessment tools and review associated neurological disorders. The adverse effects on both the patient and the family are provided along with various treatment options.

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Definition and diagnostic criteria

The term 'apathy' according to English language dictionaries is defined as a lack of interest or concern. The word apathy consists of the prefix 'a' meaning without and 'pathos' the Greek word for passion. Apathy is therefore often described as a lack of emotion or interest. Nurses who come across apathetic patients may describe them as 'lacking a spark' or 'drive'. However, consensus on its definition in the field is still absent.

Two researchers, Robert Marin and Sergio Starkstein, have led the work in suggesting a diagnostic criterion to define this condition. The work of the American psychiatrist Robert Marin in the early 1990's initially led the interest, describing it as a syndrome of "...diminished motivation and goal directed behaviour, not attributed to diminished level of consciousness, general cognitive impairment or emotional distress.…therefore a state of primary motivational impairment" (Marin,

p.12:1990). According to Marin, Biedrzycki & Firinciogullari, (1991) apathy is a disorder of drive and motivation, and characterised by three main domains:

- diminished goal-directed behaviours lack of interest or concern for social and personal activities
- lack of responsiveness to positive and negative events (emotional indifference)

Marin emphasised that the lack of motivation is primary, and not secondary to intellectual impairment, emotional distress (e.g., depression), or impaired consciousness (e.g., delirium). Other researchers have conceptualised apathy as an absence of responsiveness to stimuli (external or internal) and characterised by a lack of self-initiated action (Stuss, van Reekum & Murphy, 2000). Whilst Levy & Dubios (2006) conceptualised apathy is a "… quantitative reduction of self-generated voluntary and purposeful behaviours" (Levy & Dubois p.916:2006). The Australian psychiatrist Sergio Starkstein (2000) modified and organised apathy into a standardised set of diagnostic criteria and specified the following core features: diminished motivation, initiative and interest and blunting of emotions. This criterion has recently been modified by a task force involving the French Association for Biological Psychiatry, the European Psychiatric Association, and the European Alzheimer's Disease Consortium in 2008 and resulted in a revised consensus criteria on the diagnostic criteria for apathy (DCA) in Parkinson's and Alzheimer's disease (Robert, Onyike, Leentjens, Dujardin, Aalten & Sarkstein, 2009) see Table 1. However, to date there still remains no consensus on the diagnostic criteria for apathy as a syndrome, and is mostly defined as a disorder of motivation.

Despite the DCA offered by Robert et al., (2009), international documentations only refer to apathy as a symptom of several conditions. The International Classification of Diseases ICD-10 (WHO, 2010) refers to apathy under symptoms and signs involving emotional state (R45.3 2010). More specifically the ICD-10 Classification of Mental and Behavioural Disorders (1992) make several references to the apathy as a symptom of several disorders; dementia, personality disorders, acute intoxication from substances e.g. sedative-hypnotic drugs and schizophrenia. In the updated American Diagnostic and Statistical Manual of Mental Disorders Fifth Edition (DSM-V-TR, 2013) apathy is only referred to under the newly named entity Mild and Major neurocognitive disorder (NCD).

Several experts within the field also argue that apathy sits on a continuum of disorders of decreased motivation with apathy being the less severe manifestation though to abulia and ending with akinetic mutism, the most severe form (Marin 1990; Marin & Wilkosz

For a diagnosis of apathy the patient should fulfil the criteria A,B,C and D
A Loss of or diminished motivation
B Presence for at least one symptom in at least two or three domains for a period of four week Domain B1 Lack of self-initiated behaviours Loss of environmental stimulated behaviours Domain B2 Loss of spontaneous ideas and curiosity Loss of environmental stimulated ideas Domain B3 Loss of spontaneous emotions Lack of emotional responsively to positive or negative events
C These symptoms A-B cause clinically significant impairment in social, occupational or other important functioning
D These symptoms A-B are not explained or due to physical disability, diminished levels of consciousness or direct physiological effects of substances (e.g., medication)

Table 1: *(Above) Diagnostic criterion for apathy*. *(Robert et al., 2009)*

Mild	Degrees of severity of apathy	Severe
Apathy	Abulia	Akinetic mutism
• Uninterested in their surroundings • Unmotivated and lacking in enthusiasm and spontaneity in day-to-day activities such as chores and activities of daily living • Diminished emotional reactivity, and diminished initiative • Lack of ability to decide	• From the Greek '*a- boule'* meaning 'without will' • The inability to initiate behaviour lack of will or initiative and can be seen as a disorder of diminished motivation (DDM). • Often used interchangeably with apathy but is regarded as the most extreme form of apathy. Characterised by the inability to communicate, initiate, and self- regulate purposeful behaviours	• A state of avolition in which the patient has no desire to think, speak or move. • Total absence of spontaneous behaviour and speech, occurring in the presence of preserved visual tracking

Table 2: *(Above)* Apathy – degree of severity and symptoms

2005) see Table 2.

Assessment

A good starting point in the assessment process, when caring for patients who might be vulnerable to apathy, is for the nurse to consider some key questions that may identify the need for a more detailed assessment such as:

- Is there a loss of interest or enthusiasm in the world around him/her?
- Does he/she lack motivation for starting new activities? E.g. are they content just to sit in their chair or lie in their bed?
- Is he/she more difficult to engage in conversation or in doing activities of daily living?
- Has their appearance changed e.g. have they lost interest in their appearance?
- Has the patient become indifferent and "cold" emotionally to events that would normally give pleasure or cause for concern?

It is also important to rule out causative factors in the initial assessment process by carrying out a detailed patient history which includes the pre-morbid personality and behaviours, and a thorough medical examination to determine whether the apathy is as a result of psychosocial, medical or pharmacological causative factors. A number of medical conditions may present with an apathy-like syndrome for example hyperthyroidism in the elderly (Lahey 1937). In addition some medications have also been implicated in apathy and need to be ruled out. The commonly prescribed antidepressants selective serotonin reuptake inhibitors (SSRI's) have been reported to affect the balance between serotonin and dopamine levels in the brain resulting in SRRI-induced apathy or as sometimes referred to SSRI- induced indifference (Settle 1998; Wongpakaran, van Reekum, Wongpakaran & Clarke, 2007; Sansone & Sansone 2010; Zahodne, Bernal-Pacheco & Okun, 2012). The effect of SSRI's exposure on the risk for apathy has not been well studied. This adverse effect is insidious in its onset, and dose-dependent i.e. the higher the dose the increased change of apathy. However, it can be completely resolved with a dose reduction or discontinuation of the SSRI (Sansone & Sansone 2010). Given that SRRI's are commonly-prescribed antidepressants in patients with Parkinson's Disease, in which the prevalence of depression is reported to vary from 2.7-90% (Rejners, Ehrt, Weber, Aarsland & Leentjens, 2008), this is an importance consideration in the assessment process. Healthcare professionals need to be aware, when caring for neuroscience patients who are depressed and prescribed SSRI, of this unwanted clinical side effect, and educate the patients as well as their families to the effect. Other notable medications which can induce an apathetic state include neuroleptics such as haloperidol and chlorpromazine. Neuroleptics are known to cause serious disturbance of the signalling mechanism in the frontal limbic system and consequentially a 'blunting of emotional arousal'.

Major Depression Disorder	Apathy Syndrome
• Presence of five of nine diagnostic symptoms with a minimum duration of 2 weeks • Depressed mood e.g. sadness • Loss of interest or feeling of pleasure • Changes in weight or appetite • Insomnia or hypersomnia • Psychomotor retardation or agitation • Loss of energy or fatigue • Feelings of worthlessness/guilt • Trouble making decisions/thinking or concentrating • Recurrent thoughts of death or suicide, or a suicide attempt	• Loss of interest in daily life, situation or stimuli that would normally be motivating • Loss of motivation • Lacking initiation and ability to sustain activities • Reduced emotional engagement and flattened affect • Blunted emotions (response to emotional situations poor or short lived) • Indifference

Table 3: *(Above)* Differences in the symptoms of depression and apathy symptoms (American Association of Psychiatry (DSM-5) 2013; Levy et al., 1998; Levy and Dubois, 2006)

It is important to understand that the assessment process may be complicated by the overlap between the symptoms of apathy and depression as this may cause diagnostic confusion (Levy, Cummings, Fairbanks, Masterman, Miller & Craig, 1998). Levy et al (1998) also suggest that much of this overlap is compounded by the use of depression scales which contain apathy items. It is important to understand that apathy and depression may coexist, and that apathy is related to, but different from major depression from the viewpoint of its causes and treatment. There is also evidence that separate brain circuits are involved in apathy and depression, adding to the view that the two are different neuropsychiatric disorders. Table 3 illustrates the main differences between the two.

Despite the recent consensus criteria for diagnosis there is no gold standard to assess apathy. However, a number of assessment tools have been developed specifically to rate the severity of apathy, and there are some instruments that assess apathy as part of a broader behavioural symptom assessment see Table 4. Due to the unavailability of structured instruments to diagnose apathy these severity assessment tools have been used to diagnose apathy using arbitrary cut off scores on the various severity rating scales. Indeed most definitions of apathy have been derived from severity assessment scores and have helped facilitate and promote the research into apathy over recent years.

Marin (1991) was the first to develop a specific scale, the Apathy Evaluation Scale (AES), as a method for measuring apathy resulting from brain-related pathology. Starkstein, Mayberg, Preziosi, Andrezejewski, Leiguarda & Robinson, (1992) later developed an abridged and modified version of the AES and publishing the Apathy Scale (AS). Several other scales have been developed since then most have been validated in Alzheimer's disease and Parkinson's disease. Given that in the acute stage of injury or illness neurological patients are often identified as being at risk of developing depression and apathy it would seem pertinent for healthcare professionals to assess for these conditions. Early identification may minimise the consequences of misdiagnosis, under treatment or overlooking apathy and aid in the caring of the patient with apathy.

Neurobiology

Although the neurobiological basis of apathy is not fully understood, research has shown several neuronal networks that are associated with apathy. These networks form parallel segregated specific circuits connecting selected cortical areas in the frontal lobes with the basal ganglia and thalamus (Levy & Dubois, 2006; Bonelli & Cummings, 2007). These circuits are also referred to as the prefrontal cortical - basal ganglia functional axis (Levy & Dubois, 2006). According to Bonelli & Cummings (2007) three main frontal-subcortical circuits are implicated in apathy these include:

- dorsolateral prefrontal circuit allows the organisation of information to facilitate a response,
- anterior cingulate circuit is required for motivated behaviour,
- orbitofrontal circuit allows the integration of limbic and emotional infor-

Assessment Scale	***Authors***	***Application in clinical practice***
Apathy Evaluation Scale (AES) 18-item Multiple rater sources AES-I (informant) AES-S (self-rated) AES-C (clinician)	Marin et al. (1991)	The reliability and validity of this scale in different patient groups has been extensively evaluated including; Parkinson's disease, Alzheimer's disease, Stroke & Traumatic Brain Injury.
The Apathy Scale (Starkstein Apathy Scale (SAS)) 14-item Scored by the patient's relative or caregiver	Starkstein et al. (1992)	Generally reliable and valid instrument, especially for use in patients with Parkinson's disease, Alzheimer's disease or Stroke
Apathy Inventory (AI) Caregiver and patient-based versions	Robert et al. (2002)	Specifically designed and validated to assess apathy in Parkinson's disease. High internal consistency, item reliability, and inter-rater reliability in patient with cognitive impairment, Parkinson's disease, Alzheimer's disease (Robert *et al*. 2002)
Lille Apathy Rating Scale (LARS) Semi-structured patient interview with 33-item divided up into 9 domains. LARS-i caregiver version	Sockeel et al. (2006) Zahodne et al. (2009)	Validated in Parkinson's disease & has the ability to distinguish apathy from depression
Neuropsychiatric Inventory (NPI) 12 subscales structured interview conducted by the clinician. Developed to assess the presence and severity of behavioural specific domains; apathy being one of 10 Administered to caregivers	Cummings et al. (1994)	Widely used and developed to evaluate behavioural dysfunction including apathy in dementia patients
Frontal Systems Behaviour Scale (FrSBe) 46-item with three subscales: apathy (14 items), disinhibition (15 items), executive dysfunction (17 items) Versions include self-rating or rating by a caregiver	Grace and Malloy (2001)	A reliable and valid measure developed as a measure of behaviour associated with damage to the frontal systems of the brain.

Table 4: (Above) *Apathy measurement/assessment tools*

mation into behavioural responses.

Many researchers have focused on these fronto-subcortical circuits, given the extensive interconnection between basal ganglia and the prefrontal cortex to explain behavioural abnormalities such as apathy. It is the dysfunction of these circuits that is thought to give rise to apathy. Neurobiological history can provide some insight and understanding of the cingulate cortex and its role in motivation. In 1879 Pierre Broca, a French physician, surgeon, and neuroanatomist, applied the term "*limbic*" (from the Latin limbus for border) to the curved rim of the cortex which includes the cingulate and the parahippocampal gyri. This area of the brain and its role in our emotions was later elaborated by the American physician, James Papez in 1937 in the seminal paper titled 'A proposed mechanism of emotion'. Papez proposed that these limbic structures formed a circuit or emotional system between the hypothalamus and the cerebral cortex and became known as the Papez circuit, and was thought to be related to the regulation of emotion. Papez (1937) also noted that tumours pressing on the cingulate cortex produced "*loss of spontaneity in emotion, thought and activity*." The limbic lobe or circuit largest component is the cingulate cortex and functionally forms part of the limbic system. The anterior cingulate cortex (ACC), the frontal part of the cin-

gulate cortex, is also referred to as the cingulate circuit (Habib, 2004; Bonelli & Cummings, 2007), and is situated deep in the cerebrum and surrounds the corpus callosum. More recently studies using modern imaging techniques have demonstrated the involvement of the anterior cingulate cortex (ACC) in motivation and associated with apathy (Zamboni, Huey, Krueger, Nichelli & Grafman, 2008), and decreased spontaneous goal-directed.

Prevalence of apathy in neurological conditions

Despite being under diagnosed and untreated apathy is particularly prominent in neurological disorders that affect the frontal lobes, the basal ganglia, or the links between them. These include acquired brain injury, in particular Traumatic Brain Injury involving the frontal lobes, stroke, subcortical pathologies mainly affecting the basal ganglia, such as Parkinson's disease (PD), Huntington's chorea and Progressive Supranuclear Palsy (PSP), and dementias such as Alzheimer's disease, vascular dementia and fronto-temporal dementia.

Although there are no authoritative estimates of its frequency in the general population some idea can be gleaned by looking at its prevalence in specific neurological conditions. Studies have demonstrated that in subcortical disorders apathy results from the disruption of ascending and descending projections to and from the frontal lobes. It occurs in 20–70% of patients with Parkinson's disease (PD) (Pedersen, Alves, Larsen & Aarsland, 2009a; Oguru, Tachibana, Toda, Okuda & Oka, 2010), depending upon the instrument for assessment and the samples examined. According to available statistics, an estimated 6.3 million people have Parkinson's worldwide (Baker & Graham 2004). Thus the number of Parkinsonian patients with apathy could be between 4.41 and 1.26 million. Furthermore this figure is likely to increase given that the number of people over age 60 is increasing worldwide and unless a prevention or cure is identified, the number of people with PD will also grow substantially over the next 25 years. Apathy has been shown to be associated with or without depressive symptoms in PD and is associated with increased severity of depression (Pedersen, Larsen, Alves & Aarsland, 2009b).

Similarly, apathy is also prevalent and persistent in the dementia population in particular fronto-temporal dementia (FTD with reported prevalence rates ranging from 62 to 89% (Mendez et al., 2008). The prevalence of apathy in Alzheimer's disease ranges from 25 to 88% with a trend to increase with disease severity (Starkstein, Jorge, Mizrahi & Robinson, 2006). In light of the aging population worldwide, global estimates of Alzheimer's Disease, the commonest subtype of dementia, is generally estimated to increase from the estimated 35.6 million to 65.7million in 2030, and by 2050, to 115.4 million (World Alzheimer Disease 2009). Moreover within this population apathy is frequently seen as an early presentation and often described before the memory deficits become apparent (Bruen, McGeown, Shanks & Venneril, 2008). This raises the issue that apathy may be an early diagnostic indicator of the disease.

Apathy is one of the most prevalent neurobehavioral symptoms in Huntington's disease (HD), occurring in approximately 70% of the symptomatic HD population (Krishnamoorthy & Crauford, 2011) and occurs in 91% in Progressive Supranuclear Palsy (PSP) (Litvan, Mega, Cummings & Fairbanks, 1996).

Individuals suffering from acquired brain injury in particular stroke and traumatic brain injury commonly manifest apathy. In traumatic brain injury (TBI), varying prevalence rates are reported. Kant, Duffy & Pivovarnik, (1998) one of the first researchers to study apathy in the TBI population reported a rate of 71.08% using a self-report version of the AES, whereas Marin & Wilkosz (2005) reported that some 50% of adults suffering from TBI are affected with apathy. Similarly van Reekum, Stuss & Ostrander, (2005) reported rates of 61.4%, whereas Al-Adawi, Dorvio, Burke, Huynh, Jacob, Knight, Shah & Al-Hussaimi, (2004) Omanian study reported much lower rates of 20%. They attributed this to the sample being relatively older than survivors in other populations and the sample was small and heterogeneous with variations in duration and severity of TBI.

Stroke survivors are commonly described as having lost interest, seem unmotivated and unable to get going. Whilst the prevalence of depression post-stroke has been extensively studied few have studied apathy. Existing studies yields a varying prevalent rate of apathy post-stroke between 20% (Mayo et al., 2009) and 55% (Yamagata, Yamaguchi & Kobayashi, 2004). In Mayo and colleagues' (2009) Canadian longitudinal study the authors found that the extent of apathetic behaviour remained fairly stable over the first year after stroke. Similarly Santa, Sugimori,

Kusuda, Yamishita, Ibayashi & Iida, (2008) found 20% of patients with apathy by patient report but also suggested that apathy tends to occur more frequently in first time stroke patients who are older and more cognitively impaired. In Hama, Yamishita, Shigenobu et al., (2007) Japanese study a higher rate of apathy 40% was reported by patients, but a lower rate of 19 % was reported by structured interview of the caregiver. A more recent meta-analysis of 24 studies found that apathy occurs in 29.5 - 40.2 % of patients after stroke, and is typically associated with worse disability and enduring cognitive deficits (van Dalen et al. 2013). In stroke, apathy has been found to be associated with damage or reduced blood flow to prefrontal cortex and basal ganglia (Okada, Kobayashi, Yamagata, Takahashi & Yamaguchui, 1997). Other researchers have demonstrated reduced regional cerebral blood flow in the basal ganglia for the apathetic patient when compared with the non-apathetic patients (Onoda, Kuroda, Yamamoto, Abe, Oguro, Nagai, Bokura & Yamaguchi, 2011).

The variation in prevalence figures may in part be attributed to the utilisation of varying assessment measurements across the studies, sample sizes and study population. In addition studies that rely on the apathetic patient rating of their severity of apathy may underestimate their severity. Within neuroscience care this may be an important consideration as many stroke and TBI survivors lack cognitive abilities or insight to be able to recognise that they have feelings or behaviours that can be attributed to apathy.

Adverse effects

Several studies have documented the adverse consequences that apathy can have on interpersonal relationships, daily functioning and recovery outcomes. Apathy may also have a direct adverse impact on self-care leading to failure to recognise early symptoms of impending medical problems. For example in a prospective multi-centre study of 686 Alzheimer's disease suffers, the presence of apathy was significantly associated with poor nutritional status (Benoit, Andrieu & Lechowski, 2008). Apathy has also been shown, although limited, to impact negatively on medication adherence. In the American study involving 81 male patients with diabetes Padala, Desouza, Almeida et al., (2008), showed that 61% were apathetic and that they were less likely to adhere to exercise plan or insulin regimen. Although no studies have been conducted on the subject this may be of significance in the field of neuroscience, given that medication adherence is a crucial area of many neurological conditions such as in Parkinson's disease and is warrants investigation.

Apathy can interfere with a patient's ability to fulfil their full recovery potential and act as a barrier to participating in rehabilitation activities (Resnick, Zimmerman, Magaziner & Adelman, 1998). In patients with TBI apathy has also been observed to contribute to the unsatisfactory integration into home and community activities (Cattelani, Roberti & Lombardi, 2008). Whilst in post-stroke patients apathy is also reported to lead to less recovery in activities of daily living (ADL's) (Mikami, Ricardo, Moser, Jang & Robinson, 2013), and a predictor of poor functional recovery (Hama et al., 2007). In a more recent American study Harris and colleagues (2014) found that post-stroke apathy resulted in an increased chance of being admitted to a nursing home and scoring below the mean FIM score.

Apathy not only impacts negatively on the patient but also on their family and carers. Apathy can disrupt the quality of life for both patients and caregivers (van Reekum et al., 2005). Gerritsen et al. (2005) study found that apathy in nursing home residents, with relatively good cognition, was found to be associated with lower quality of life (QoL). However, in those with severe cognitive impairment apathetic behaviour was associated with high QoL. Apathy has also been found to be associated with increased caregiver burden and reported to be a significant source of caregiver stress. In a UK study by Leroi et al (2012) a significantly greater burden was seen in carers of PD participants with apathy when compared with carers of PD participants without such behavioural disturbances. In contrast, caregivers of those with Alzheimer's disease who suffer apathy found apathy to be less burdensome than more active behaviours such as executive dysfunction and disinhibition (Davis & Tremont 2007).

The full impact of apathy on families and carers is unknown. Additional research is especially warranted to evaluate the impact due to societal increases in both care giving and the prevalence of apathy in many neurological disorders.

Nursing Care

Once the reversible causes of apathy have been excluded, and the diagnosis of apathy has been made, interventions need to be con-

sidered to prevent any adverse consequences. Despite the increasing number of published paper on prevalence rates of apathy in many neurological conditions there is very little published literature on the treatment or management of apathy. Nurses can contribute to the management of apathy in a number of ways. By adopting an individualist approach to their management, centred on the individual behaviour that involves the family and caregiver, then the apathy potentially is treatable. Nurses' interventions should be tailored initially to the suspected causes of apathy, which have been identified via a detailed assessment. Furthermore regular assessment may be useful as a means of monitoring especially in rehabilitation.

Many of the non-pharmacological interventions are subject to a limited number of studies and most have been carried out in the dementia population. Brodaty & Burns (2012) argue that despite the lack of methodological rigor, non-pharmacological interventions in dementia patients have the potential to reduce apathy. They further argue in their systematic review that therapeutic activities, particularly those provided individually such as creative activities, cooking and gardening have the best available evidence. Critical to the implementation of any non-pharmacological approach for an apathetic patient is the development of an individualised plan that involves input from the multidisciplinary team.

As apathy may result in a lack of or failure to engage in goal direct-behaviour, motivation is a very important facet of the recovery and rehabilitation process. If apathy is believed to be related to a lack of motivation, factors influencing the appraisal of the situation, such as past experiences and beliefs and previous interests may help to predict individual strategies for engaging with the apathetic patient. This may involve providing external clues and prompting the patient. Exploring what the patient wants and why they want it, and explaining what might realistically be expected may assist in establishing attainable goals. Although the apathetic patient may not be able to initiate activities the nurse can help by initiating or suggesting an activity that the patient enjoys and encouraging them to participate. Nurses may, if available, want to consider group activities rather than activities that the patient is expected to do on their own. In addition offering choices to enhance self-determination and rewards frequently, especially early on, may help to sustain the behaviour. Interventions should also be specifically and appropriately geared to the limitations of the patient's condition.

Several studies have demonstrated the importance of the environment in the management of apathy, although this is mainly found in the literature on dementia care. Under stimulation or a lack of appropriate stimulation in such an environment may lead to boredom in older adults, particularly for those with dementia (van Reekum et al., 2005). Multisensory stimulation also referred to Snoezelen therapy has been shown to reduce apathy compared with activity therapy or no therapy in people with moderate to severe dementia (Verkaik et al., 2005). Holmes, Knights, Dean et al. (2006) demonstrated that live music had a positive engagement effect in dementia patients with apathy. The usefulness of stimulation such as multisensory stimulation and music in other neurological disorders is unknown. A systematic review by Lane-Brown & Tate (2009) evaluated the effectiveness of non-pharmacological interventions available in the TBI population and concluded that there was not robust evidence for the interventions reviewed.

There is very little evidence to guide the pharmacological management of apathy. Drijgers, Aalten, Winogrodzka, Verhey & Leentjens, (2009), in their systematic review concluded that there was insufficient evidence for the pharmacological treatment of apathy in patients with neurodegenerative disease. As the dopaminergic system is implicated in the pathophysiology of apathy this has increasingly been the target for modulation in the treatment of motivational deficit behaviours such as apathy.

Nurses need to acknowledge the role the family and carers can play in caring for the apathetic patient. By being aware of the impact of apathy on families and carers nurses can help to improve support and coping mechanisms especially in the in-patient phrase when much of the family and social adaptation occurs. Helping the family and caregiver understand their family members' behaviour, (i.e that they are not just being lazy or difficult) may prevent resentment and difficult interactions between themselves and the patient. Additionally, it may also help to alleviate caregiver frustration, stress and burden. Family and carer involvement may also be effective in reducing apathy and increase functioning and outcomes of the patient. Nurses can play a crucial role in involving

and educating family members and caregivers about apathy. This should include: providing information about their family members' condition and apathetic behaviour; involving the family and carers in multi-disciplinary team meetings, goal setting and decision-making; and targeting interventions that involve the family and carers.

The apathetic patient may depend more on their families and carers to provide care, resulting in a loss of skill and function in activities of daily living. Thus it is vitally important when planning care activities that the family or caregiver does not cause more apathy seeking active engagement, rather they promote physical and cognitive stimulation.

In summary apathy care and management requires a multidisciplinary approach that considers careful assessment, environmental modifications, and individual interventions that consider the apathetic person as well as their family and carers. Although non-pharmacological interventions may be of limited effectiveness they are low risk and should be a considered in the nursing management of apathy.

Conclusion

Apathy is a debilitating, under-recognised and under-studied multidimensional syndrome that affects many neurological conditions, and results in functional impairment among patients and causes stress among their caregivers. Despite the prevalence of apathy in varying neurological conditions there remains a lack of a clear definition and a consensus on diagnostic standards. A variety of assessment tools have been developed over the years but there is no gold standard by which to assess apathy. Early identification of patients at risk of developing apathy through timely and accurate assessment and the development of targeted interventions may help to prevent misdiagnosis, improve long-term outcomes and the quality of life for the patient and their carers. There is a paucity of research to guide healthcare professionals in the management and treatment of apathy. High quality, methodologically rigorous treatment studies of apathy is required along with studies that validate the use of an assessment scale across varying pathological conditions. Given the importance of motivation in achieving goals in rehabilitation and during the recovery process following illness and injury, apathy deserves more focused attention. Neuroscience nurses can play a major part in this process by early recognition that apathy is present in significant proportions in the neuroscience population, by participating in the assessment process and contributing to the patients' management through non-pharmacological interventions and education and involvement of the family and caregivers.

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The Louie Blundell Prize

This prize is in honour of our colleague Louie Blundell and will be awarded for the best neuroscience nursing paper by a student submitted to the Australasian Neuroscience Nurses Association (ANNA) for inclusion in the Australasian Journal of Neuroscience by the designated date each year. The monetary value of the prize is AUD$500.

Louie Blundell, was born in England, and although she wanted to be a nurse she had to wait until after World War II to start her training as a mature student in her late twenties. Later she and her family moved to Western Australia in 1959. She worked for a General Practice surgery in Perth until a move to the Eastern Goldfields in 1963. Subsequently, she worked at Southern Cross Hospital and then Meriden Hospital. During this time she undertook post basic education to maintain her currency of knowledge and practice, especially in coronary care.

Louie was also active in the community. She joined the Country Women's Association and over the years held branch, division and state executive positions until shortly before her death in 2007. She was especially involved in supporting the welfare of students at secondary school, serving on a high school hostel board for some time.

She felt strongly that education was important for women and was a strong supporter and advocate of the move of nursing education to the tertiary sector, of post graduate study in nursing and the development of nursing scholarship and research, strongly defending this view to others over the years.

For further details and criteria guidelines please visit the ANNA website at **www.anna.asn.au**

Analysing the Present Situation of Protection Against Exposure to Temozolomide on a Japanese Neurosurgical Ward

Tetsuya Isaji

Abstract

Temozolomide was approved as an oral alkylating agent against glioma in 2006 in Japan. The nurse gives Temozolomide through the stomach tube with medicine suspension method for patients with a disturbance of consciousness and a dysphagia. Exposure of antineoplastic agents has been a focus in recent years in Japan. Nurses use protection goods when they give Temozolomide; however, they did not protect themselves from exposure of the antineoplastic agent when they disposed of used materials and excrement. Therefore, there is the need to educate and evaluate protection materials.

Keywords: *Temozolomide, Antineoplastic agents, exposure, personal protective equipment, Japan.*

Background

Temozolomide was approved as an oral alkylating agent against glioma in 2006 in Japan. Seventeen patients with a glioma were prescribed Temozolomide at the Kizawa Memorial Hospital in 2012. Although Temozolomide is an oral antineoplastic agent, not all patients can take it by themselves. The nurse gives Temozolomide through the stomach tube via medicine a suspension method for patients with a disturbance of consciousness and a dysphagia. It was found that nurses suffered from alkylating agent due to giving Temozolomide from stomach tube. Exposure of antineoplastic agents has been a focus for the Japanese medical staff in recent years. Thus, many nurses have studied about exposure of antineoplastic agents in the hospital since 2010. Although nurses take care of exposure from injections of antineoplastic agents, it was found that they did not worry about exposure from the oral antineoplastic agents.

Objective

The purpose of this study was to analyse the present situation of preparation and giving Temozolomide and handling of excrement in the neurosurgical ward in order that nurses can give patients the prescribed treatment and fully understand the safety precautions for themselves when handling excrement.

Questions or comments about this article should be directed to Tetsuya Isaji, Certified Nurse Specialist Cancer Nursing, Kizawa Memorial Hospital, Gifu, Japan at t-isaji@kizawa-memorial-hospital.jp

Methodology

The present situation of handling and giving Temozolomide was understood. Best practice was researched and discussions were held with the pharmacist about the best way to safety handle the giving of Temozolomide and the consequences of handling patients' waste products afterwards.

Results

Nurses wore vinyl gown, surgical mask and nitrile gloves in medicine suspension method and giving Temozolomide from stomach tube (Fig. 1).

Figure 1: *(Left) Nurses use vinyl gown, surgical mask and nitrile glove for preparing and giving Temozolomide.*

In the medicine suspension method, nurses put Temozolomide in syringe and they used apple juice as a solution in order to keep the stability of Temozolomide (Fig.2). Nurses put the syringe in hot water. Then, they put the cap on the syringe because it is possible that the apple juice-melted Temozolomide leaks without the cap (Fig.3). After warming up the syringe, nurses stirred the Temozolomide solution by shaking the syringe. They then discarded the used syringe and other goods without any protection from exposure (Fig.4,5).

Figure 2: *(Above) Syringe in a medicine suspension method.*

Figure 3: *(Above) Using cap for preventing leaking.*

Figure 4: *(Above) discard with used goods.*

Figure 5: *(Above) nurses do away with used syringe without protection.*

When handling waste, nurses wore a vinyl apron without sleeve, surgical mask and vinyl glove. This is the same as for patients who do not take Temozolomide (see Figure 6). If the patients who take Temozolomide had a urethral catheter, nurses wore only vinyl gloves. Used bottles for collecting urine are washed with detergent. This is the same as other patients who do not take Temozolomide (see Figures 7 & 8). Nurses discarded used diapers without protection from exposure.

Discussion

Nurses in the neurosurgical ward used protection goods when they give Temozolomide. However, they did not protect themselves from exposure of the antineoplastic agent

Figure 6: *(Above) Wearing style when handling excrement.*

Figure 7: *(Above) Washing used bottle.*

Figure 8: *(Above) Storing washed bottles.*

when they disposed of used materials and excrement. The method of discarding used materials is very dangerous because nurses do not protect themselves. Therefore, it is possible that Temozolomide adheres to the nurses' hands. Nurses who wear vinyl aprons and vinyl groves for handling excrement can be exposed to urine containing Temozolomide easily. In addition, vinyl gloves cannot protect from antineoplastic agents. Also, the washed bottles are placed on newspaper to dry. The newspaper gets wet with Temozolomide-contaminated water.

Temozolomide is an alkylating agent. Alkylating agents act on DNA; thus, it has an affects on cancer cells but normal cells are also affected. Temozolomide is excreted in the urine within 8hours and metabolites of Temozolomide remain for over 24 hours (Baker et al.,1999). Approximately 75% of nurses in the neurosurgical ward are in the 20-39 year-age-group. This is important and safety must be considered due to the opportunity of pregnancy within this age group. There is a law to protect people from exposure of radiation but the Japanese government has not made a national policy against exposure of antineoplastic agents. Although the guideline was made by the Japanese Society of Hospital Pharmacists in 1992, the nurses have not been educated to this guideline. So, only few nurses know about exposure of antineoplastic agents. Hence, it is important to educate and protect the nurses from exposure. Therefore, we need to prepare and evaluate protection materials. This is an ongoing education initiative for the Japanese nurses.

Conclusion

After this study, all nurses now have a better understanding of the consequences of Temozolomide delivery and the precautions that must be taken when delivering this type of medication. For our staff, this is an ongoing education matter and there will be follow up evaluations of the content and protection methods available.

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Anti- N-methyl-D-aspartic acid Receptor Encephalitis: A Critical Literature Review and Implications for Neuroscience Nurses

Linda Nichols

Abstract
Anti- N-methyl-D-aspartic acid (anti-NMDA) Receptor Encephalitis is a devastating disease that is linked with alarming rates of misdiagnosis and clinical management conundrums. As a relatively new disease process the literature is limited to case studies and small case series, generally focusing on the natural history of the disease, clinical management and medical interventions. This has resulted in a dearth of literature related to the neuroscience nursing care of individuals diagnosed with anti-NMDA receptor encephalitis. Nurses play a vital role in caring for individuals diagnosed with anti-NMDA receptor encephalitis and thus it is vital that this role is explored.

Keywords: *anti-NMDA receptor encephalitis, paraneoplastic syndrome, autoimmune disease, neuroscience nurses.*

Let me introduce myself
You don't know me yet, but by the time you do
You will wish we had never met
I will take your memory
I will take your speech
I will make you see, hear, horrible things
You will no longer be able to eat
You will no longer be able to sleep
You will no longer be able to breathe
I will play with your heart, and just for fun
I may even make it stop
Your arms and legs will be at my command
And these are just a few of the things I can do
You will be mine, all mine
Let me introduce myself
My name is Anti-NDMA Receptor Encephalitis

Figure 1: *(Above) Poem by Nesrin Shaheen (2008).*

Introduction

This beautiful poem was created by Nesrin Shaheen. Her daughter Sonia suffered from anti-NMDA receptor encephalitis. It's a very good description of what this illness does to its sufferers.

Identified in 2005 and officially named in 2007, anti-NMDA receptor encephalitis is encephalitis associated with antibodies against the N-methyl-D-aspartic acid receptors and including psychotic manifestations, autonomic abnormalities and catatonia (Sansing et al., 2007). Whilst there has been limited opportunity to analyse anti- N-methyl-D-aspartic acid receptor encephalitis (anti-NMDA receptor encephalitis) from a historical perspective (Day, High, Cot, & Tang-Wai, 2011; Hughes et al., 2010), there has been noteworthy reference in the literature prior to this of similar descriptors of the disease process. First recognised as a paraneoplastic syndrome in young women with ovarian teratomas, anti-NMDA receptor encephalitis is a member of the neuro autoimmune syndromes that make antibodies against synaptic proteins in response to either a neoplasm or another potential trigger (Dalmau et al., 2008). This paper critically explores the literature focusing on the implications for neuroscience nurses, exploring the key neuroscience nursing and treatment interventions that require consideration when caring for individuals diagnosed with anti-NMDA receptor encephalitis.

Questions or comments about this article should be directed to Linda Nichols, Lecturer Unit Coordinator Neuroscience Nursing, School of Health Science, University of Tasmania at linda.nichols@utas.edu.au

Anatomy and Pathophysiology

Anti-NMDA receptor encephalitis is an autoimmune disease that results in the production of antibodies against the glutamate binding site of the NR1 subunit of N-methyl-D-aspartic acid (NMDA) receptors. The receptors are located throughout the central nervous system with concentrations in the forebrain, limbic system and hippocampus, which in some part explains the clinical features of the disease (Ferdinand & Michelle, 2012). The receptors are made up of two subunits;

NR1, which binds glycine and NR2, which binds glutamate (Dalmau, et al., 2008). The receptors are ligand-gated cation channels, which play a role in synaptic transmission and plasticity, acting as the cellular substrates for learning and memory (Chapman & Vause, 2011). For a receptor to function, both subunits must be bound, thus dysfunction of one leads to a failure of synaptic transmission (Verhelst et al., 2011). Transmission between the receptors is important and it is hypothesised that hypoactivity plays a role in the pathogenesis of schizophrenia with hyperactivity linked to conditions such as dementia (Goff & Coyle, 2001). In the case of anti-NMDA receptor encephalitis the body forms antibodies as an immune response or against neuroplastic cells contained within a tumour and in turn this is replicated with the receptor resulting in damage to the ion channels in the post-synaptic membrane (Day, et al., 2011). The antibodies decrease the number of cell surface NMDA receptors and NMDA receptor clusters in the postsynaptic dendrites, reducing neuronal signals (Chapman & Vause, 2011). However, in most cases the disease is reversible irrespective of the duration of symptoms, supporting an immune mediated neuronal dysfunction rather than irreversible degeneration of the neurons (Ferdinand & Michelle, 2012). Despite this reversibility inflammatory processes can lead to scaring and a prolonged or an incomplete recovery for individuals, particularly those who experience delays in diagnosis and subsequent treatment.

Diagnosis

Described by some as being “driven insane by your own immune system”, anti-NMDA receptor encephalitis is linked to an alarmingly high incidence of misdiagnosis and inappropriate admission to psychiatric units (Lebon et al., 2012; Maneta & Garcia, 2014; Turkdogan, Orengul, Zaimo, & Ekinci, 2014). Regularly misdiagnosed as a psychiatric presentation, there is a need to consider anti-NMDA receptor encephalitis as a differential diagnosis when individuals present with the constellation of acute onset psychiatric symptoms, autonomic dysfunction and altered muscle tone (Chapman & Vause, 2011). Rapid referral to psychiatric teams for assessment and diagnosis has long been criticised for its ethical abuses and the ability that psychiatry has to ultimately cancel all other medical interventions and investigations (Rutkowski & Gordon, 1994). This initial misdiagnosis and incarceration into psychiatric units has historically limited further investigation of a differential diagnosis and it is not uncommon for individuals to experience an exhaustive range of medical evaluations and investigations before a diagnosis is made. There are many published case studies and cases presented in the media describing young girls who were initially identified as psychiatric cases only to be diagnosed with anti-NMDA receptor encephalitis. Despite this, it is often invaluable to have ongoing psychiatric input as individuals will often present with a complex range of psychiatric symptoms and the management of these symptoms can improve recovery time.

Antibody testing can often be delayed, due to the limited number of pathology laboratories accredited in Australia to test for NMDA receptor assay and diagnostic delays. Cerebrospinal fluid (CSF) analysis is integral to diagnosing a multitude of cerebral nervous system conditions. Abnormal CSF analysis is reported in 80% of affected individuals (Dalmau, et al., 2008), presenting with increased oligoclonal bands or bands of immunoglobulins and leukocyte counts. Over 90% of individuals will demonstrate lymphocytic pleocytosis during the course of their illness (Consoli et al., 2012; Johnson, Henry, Fessler, & Dalmau, 2010). However, protein and glucose levels can present as normal, potentially delaying further analysis of the CSF. The diagnosis of anti-NMDA receptor encephalitis is confirmed with specific testing for antibodies to the NR1/NR2 heteromers of the NMDA receptor in both CSF and serum samples (Iizuka et al., 2008). Whilst serum antibodies to the NMDA receptor can be detected via a cell-based assay, the levels are typically lower than those in the CSF, with Vincent & Bien (2008) suggesting that CSF levels can be up to 10 times higher than serum levels.

Electroencephalograph presentation is usually slow non-specific dis-organised activity with occasional electrographic seizures. The rhythm change can occur relatively unilaterally (Day, et al., 2011). There is generally little evidence of limb paralysis and deep tendon reflexes are generally normal. Imaging, including computed tomography, plays a limited role in the diagnostic period with magnetic resonance imaging (MRI) generally presenting as normal on admission (Gable et al., 2009). However, during the course of the disease MRI may show meningeal enhancement, and periventricular white matter changes similar to demyelination (Hughes, et al., 2010; Tuzun et al., 2013). Once the diagno-

sis of anti-NMDA receptor encephalitis is suspected, a prompt and thorough investigation for neoplasm is vital, including imaging of the abdomen, chest and pelvis. Dalmau, Lancaster, Martinez-Hernandez, Rosenfeld & Balice-Gordon (2011) suggest that the younger an individual is, the less likely that a tumour will be detected. In the absence of an identified malignancy, authors such as Florance et al., (2009), suggest that periodic ultrasound of the abdomen should be continued for up to two years post diagnosis.

Phases of Anti-NMDA Receptor Encephalitis

Anti-NMDA receptor encephalitis differs from limbic encephalitis in that it normally develops in five distinct phases. In children and infants the symptoms can differ with the prominent presenting symptom being mood or behavioural changes including temper tantrums, disintegrative disorders and autistic like behaviours (Ben Achour et al., 2013; Creten et al., 2011; Tsutsui et al., 2012). This is followed by a period hyperactivity and irritability rather than psychosis, often delaying the diagnosis (Dalmau, et al., 2011). Whilst adults are more likely to present with psychological disturbances and children with neurological disturbances, the symptoms generally merge within a month to a similar presentation (Ingram & Robertson, 2013; Titulaer et al., 2013).

The first phase is the prodromal phase where up to 70% of individuals experience symptoms similar to those of a viral infection (Dalmau, et al., 2011). However, this prodromal phase may go unnoticed, as symptoms are often mild and non-specific including headache, malaise and fever. These symptoms generally last a week and can occur up to two weeks prior to the second phase (Ferdinand & Michelle, 2012). The second phase is the psychiatric phase, where individuals present with an acute onset of psychiatric manifestations with an estimated 68% of individuals exhibiting psychotic symptoms (Dalmau, et al., 2008; Gable, et al., 2009). Symptoms can include progressive confusion, agitation, hallucinations and suicidal ideation, fear, personality changes, mood disturbances, depression, twitching, paranoid delusions and violent episodes (Gable, et al., 2009; Sonn & Merritt, 2010). It is important to note that this phase can be a particularly difficult for parents and loved ones; there are often numerous presentations to hospital and psychiatric admissions and reports of parents struggling to manage increasingly difficult behaviours at home with limited support.

The non-responsive phase presents with cognitive decline, anorexia, incontinence, disorganised language progressing to linguistic regression and mutism, finally leading to catatonia (Gable, et al., 2009). Individuals will often present with their eyes open, yet unresponsive to visual threats, and absent or limited responses to painful stimuli (Chapman & Vause, 2011; Iizuka, et al., 2008). During this phase there is often evidence of increased muscle tone with dystonic and cataleptic posturing. An estimated 76% of individuals will experience seizures (Dalmau, et al., 2008), and seizure activity can progress to status epilepticus. The hyperkinetic phase can present as a separate phase or can overlap with the unresponsive phase. It presents as increased muscular activity resulting in excessive and abnormal movement of the limbs and body. Dalmau et al., (2008) suggests that up to 86% of individuals will experience dyskinesia with individuals presenting with increased restlessness, flexing of the limbs and facial twitches (Day, et al., 2011). Movements generally include the chorea-like rapid uncontrolled movements and athetosis or slower twisting movements. Orofacial dyskinesia is identified as a key feature, with individuals exhibiting lip smacking and tongue twitching (Dalmau, et al., 2008). An estimated 69-89% of individuals will also experience dysautonomia, with autonomic instability evidenced by fluctuation in temperature, blood pressure, heart rate, hyper-salivation and hypoventilation (Chapman & Vause, 2011; Gable, et al., 2009). During the course of the disease more that 60% of individuals will experience hypoventilation and will require respiratory support (Dalmau, et al., 2008). (Ingram & Robertson, 2013) purport that up to 75% of individuals require admission to an intensive care unit at some point during the trajectory of the disease for respiratory support (Lim et al., 2013).

With treatment, individuals will enter a gradual recovery phase however this may include relapses, with up to 20-25% of individuals experiencing at least one clinical relapse (Dalmau, et al., 2011). Recovery may take up to three years and it is a multistage process that usually occurs in the reverse of the symptom presentation. Social behaviour and executive functions are the last to normalise and recall of the trajectory of the illness is normally poor as persistent amnesia is a characteristic feature of anti-NMDA receptor encephalitis (Dalmau, et al., 2008).

Incidence and Aetiology

The exact incidence of anti-NMDA receptor encephalitis remains difficult to identify. When it was first identified in 2005, four women with ovarian teratomas were described. By 2007 when the NMDA antibodies were detected over 420 cases were known (Dalmau, et al., 2011), and the number of cases continue to grow suggesting that anti-NMDA receptor encephalitis is perhaps not such a rare disease. Gable, Sheriff, Dalmau, Tilley & Glaser (2012) suggest that anti-NMDA receptor encephalitis occurs four times more frequently than viral encephalitis. Whilst anti-NMDA receptor encephalitis affects both genders, there is a predominate predilection for females. This is shown in Dalmau et al., (2011) case series of over 400 individuals identifying that 80% of those diagnosed were female. The estimated age range spans from 8 months to over 80 years (Armangue et al., 2013; Day, et al., 2011; Wong-Kisiel et al., 2010) with 65% of cases occurring in individuals aged 18 years or younger (Dalmau, et al., 2008; Gable, et al., 2012). Individuals generally present with an unremarkable medical and psychiatric history (Mann et al., 2012) and no history of cigarette, alcohol, or drug use (Barry et al., 2011). Individuals are often not sexually active and have no history of oral or genital herpes (Hegarty & Mikli, 2013).

Anti-NMDA receptor encephalitis is generally associated with ovarian teratomas that almost always contain central nervous system tissue (Day, et al., 2011). However, anti-NMDA receptor encephalitis has also been associated with breast cancer, neuroblastomas and Hodgkin's lymphoma (Lebas, Husson, Didelot, Honnorat, & Tardieu, 2010; Zandi et al., 2009). Within the male population, anti-NMDA receptor encephalitis has been linked to a number of malignancies including testicular seminoma and lung cancer (Dalmau, et al., 2008) although it is estimated that less than 5% of male individuals will present with an underlying malignancy (Finne Lenoir et al., 2013). Dalmau et al., (2008) and Gable et al., (2009) suggest a predilection in Hispanics, African Americans, Asian and Pacific Islanders, thereby linking with an increased incidence of ovarian teratomas in individuals with dark skin pigmentation. However, it is suggested that up to 40% of individuals do not present with an identifiable tumour (Dalmau, et al., 2008). Whilst there have been some reports in the literature linking anti-NMDA receptor encephalitis to vaccinations and non-specific systemic infections, (Dalmau, et al., 2011) suggest that these events only act as an adjuvant of the autoimmune response in individuals with a racial or genetic predisposition to autoimmune diseases.

Treatment and Interventions

The care of individuals diagnosed with anti-NMDA receptor encephalitis is complex and challenging and the clinical presentation presents a conundrum in terms of what type of ward setting is most beneficial and the safest option for individuals. Individuals generally require hospitalisation for several months, followed by long-term physical and cognitive rehabilitation. The complex neuro-psychiatric presentation may lead to the consideration of a psychiatric unit as a potential admission option. However, psychiatric units are limited in their ability to facilitate medical interventions and to facilitate regular monitoring of individuals in terms of autonomic instability. Likewise, whilst a neurology setting may be considered appropriate in terms of managing seizures, these units are often ill-equipped to manage the behavioural and psychiatric needs of individuals diagnosed with anti-NMDA receptor encephalitis (Chapman & Vause, 2011). Neurosurgical units are often considered the best option for the acute management of individuals diagnosed with anti-NMDA receptor encephalitis (Lim, et al., 2013). Neurosurgical wards, particularly those that specialise in traumatic brain injuries, are often staffed at higher levels and can provide the constant supervision together with an environment that is usually secure and organised to accommodate confused individuals. More importantly, the nursing staff are competent in managing conditions such as post traumatic amnesia and autonomic dysfunction. The symptoms of autonomic dysfunction can interfere with rehabilitation due to their unpredictability and physical nature that adversely affect progress. Treatment is focused on a symptomatic approach and symptoms are treated independently as there is no specific treatment for the underlying cause.

Initially treatment is focused on the identification and removal of any tumour. However this stage can often be delayed due to precedence to treat psychiatric symptoms. The surgical removal of malignancies, particularly ovarian teratomas, is associated with improved outcomes and a reduction in the severity and duration of symptoms (Ferdinand & Michelle, 2012; Uchino et al., 2011). The use of steroidal and immunosuppressive

therapy has proved vital in the management of symptoms, although there have been no large studies or randomised control trials that have evaluated the management and treatment effect on anti-NMDA receptor encephalitis (Hegarty & Mikli, 2013; Ingram & Robertson, 2013). Treatment decisions are generally based on personal preferences and clinical symptoms rather than antibody titers (Titulaer, et al., 2013).

The first line of treatment is usually a high dose five-day course of intravenous methylprednisolone and or intravenous immunoglobulin therapy. As a plasma product derived from the immunoglobulin antibodies, intravenous immunoglobulin slows the body's natural immune response and binds the overactive antibodies, in this case the N1 subunits of the NMDA receptors (Pham, Daniel-Johnson, Stotler, Stephens, & Schwartz, 2011). Individuals generally begin to demonstrate improvement with 48 hours of commencing immunoglobulin therapy. However this may involve individuals experiencing a progression in reverse order through each of the clinical phases (Day, et al., 2011). Plasma exchange or plasmapheresis is an alternative option to immunoglobulin therapy, where the offending antibodies can be removed from circulation in an attempt to temper the disease process. The use of plasma exchange can be limited in cases of autonomic instability, or in children and uncooperative individuals (Dalmau, et al., 2011). Whilst plasma exchange can remove the offending antibodies, the success is dependent on adjunct treatments that are focused on the suppression of the immune system, including treatment with rituximab.

Immunotherapy is generally considered as either an adjunct or second line therapy. It is considered when there has been a delayed diagnosis or limited response to other interventions, and for those without an identifiable malignancy (Ferdinand & Michelle, 2012; Lim, et al., 2013). Immunosuppressive treatment options include rituximab, azathioprine and cyclophosphamide. Rituximab (or Mabthera as it is commonly known) is a therapeutic monoclonal antibody that is used to treat common forms of Non-Hodgkins lymphoma (NHL) and is often the drug of choice as it has demonstrated positive results in the treatment of multiple autoimmune diseases. Rituximab and immunoglobulin therapy are often used in combination due to their synergistic effects and are associated with long-term remission for a number of disease processes (Ahmed, Spigelman, Cavacini, & Posner, 2006). Immunotherapy is associated with fewer relapses when used for anti-NMDA receptor encephalitis (Gabilondo et al., 2011; Titulaer, et al., 2013). Treatment is generally continued until there has been clinical improvement and a reduction in antibody levels. However treatment can be extended for up to 1 year to prevent clinical relapses (Dalmau, et al., 2011; Ferdinand & Michelle, 2012).

Symptomatic treatment includes seizure management, where multiple agents may be required, with some individuals requiring benzodiazepine infusions for refractory seizures (Maramattom, Philip, & Sundaram, 2010). Seizure activity can often be a presenting symptom or can develop during the early stages of the disease (Dalmau, et al., 2011). Seizure activity can be difficult to distinguish from hyperkinetic movements and this has resulted in both an under recognition of seizure activity for some individuals, or in contrast, an unnecessary escalation of anti-convulsant medications for others (Bayreuther et al., 2009). The use of continuous video electroencephalography is recommended as it allows for simultaneous video recording of clinical manifestations to be correlated with electroencephalography activity. This approach, and clear descriptions of behaviours by staff, can ensure that there is a clear differentiation between seizure activity and abnormal movements.

Psychiatric drugs do not stop the development or progression of the disease. However they do play an important role in reducing the psychiatric symptoms. All features of psychosis should be managed in the conventional manner, as treatment will often unmask other symptoms that can then be treated (Ferdinand & Michelle, 2012). Supportive treatment is vital as individuals often respond to treatment environments such as those provided for post traumatic amnesia, including a darkened room, minimal distractions and routine sleep wake cycles. Individuals often require physical and chemical restraint to maintain their own safety, particularly during periods of violence, self-harm and agitation (Zehry, Matrunola, & Hyde, 2011). Sedation reduces elevated sympathetic tone and the relaxed state often facilitates easier management of blood pressure and intracranial pressure. It is vital that oxygen saturations are monitored at all times. It is also important that nurses support families when making this decision and that hospital policies and protocols are followed at all times. Ongoing reas-

surance to family members is vital as the symptoms that individuals can experience can be distressing.

Neuro-rehabilitation consultants play a vital role in facilitating the recovery of individuals post anti-NMDA receptor encephalitis. Individuals respond to similar medications and care regimes used for post traumatic amnesia. When managing agitation, it is important to consider non-pharmacological approaches. Individuals should be nursed in a low stimulation environment, and provided with ongoing reorientation and reassurance. Targeted strategies should also be employed to address short-term memory impairment (Chapman & Vause, 2011). This can include interactive white boards and clear care plans. Initial goals of treatment include increasing individuals' responses to external stimuli including pain, touch, light and verbal stimulation. Following this, there is a focus on channeling excess energy into functional activities that will help in reestablishing a normal routine again. More than 85% of individuals will still be experiencing significant psychiatric symptoms when discharged (Kayser, Kohler, & Dalmau, 2010), thus making family and caregiver education a high priority.

Individuals also face the challenges of impaired executive functioning including reduced impulse control, loss of inhibitions, poor attention spans, disorganisation and impaired planning skills (Chapman & Vause, 2011). Given the high incidence of frontal lobe dysfunction associated with anti-NMDA receptor encephalitis, long-term rehabilitations, particularly specialised neuro rehabilitation, is central to improved outcomes. As individuals pass through the initial phase of anti-NMDA encephalitis they often begin to exhibit a range of symptoms, including a range of functional changes. These changes can affect an individual's thinking; memory and reasoning skills; language skills including communication, understanding and expression and sensations may be altered including taste, touch and smell. Perhaps one of the most difficult challenges is when an individual's emotions are affected resulting in anxiety, depression, aggressive and or socially inappropriate behaviour.

The establishment of a sleep-wake cycle is vital to assisting individuals to re-orientate and still have the energy to undertake activities of daily living. Nursing staff should have clear care plans in terms of nocturnal activities and noise and stimulation should be kept to a minimum. Activities of daily living and care interventions, such as taking observations, can be difficult to undertake as they can increase an individual's agitation and anxiety, triggering and elevating behaviours and autonomic instability. Thus careful planning and alternative options often need to be considered, which also includes regular toileting regimes to assist in regaining continence. The majority of individuals will also require nutritional support, and coupled with this is the need to maintain glycaemic control. Hyperglycaemia leads to hypercatabolic state and muscle protein breakdown. Tight glycaemic control has become a part of the routine management of individuals diagnosed with anti-NMDA receptor encephalitis.

Recovery and rehabilitation can also involve speech therapy for both language and communication assistance as well as for swallowing assistance. This is because a significant proportion of individuals will develop swallowing issues at some stage during the course of the disease, and will require enteral feeding due to decreased level of consciousness and/or reduced motor control. Individuals require close monitoring due to the impulsive nature of the disease and activities should be broken down into smaller tasks to assist in maintaining attention. Interventions need to be focused on reducing the risk of generalized physical deconditioning, and where possible, maintaining independence with activities of daily living. This requires a multidisciplinary approach including nurses, speech therapists, occupational therapists and physiotherapists. At all times individuals need to be nursed in a safe environment that includes bed rails, padding and floor mats to protect individuals during the hyperkinetic phase. Central cardiac monitoring also needs to be in place to monitor for respiratory distress and supported by regular observations. When available, a 1:1 nursing staff ratio is also suggested.

Prognosis and Outcomes

The prognosis and outcome for individuals is generally improved when there has been a recognised malignancy and diagnosis has been prompt. Mortality rates range from 4-10% with the average time to death 3.5 months (Dalmau, et al., 2011; Gable, et al., 2009). Post diagnosis mortality is linked to events including sepsis, cardiac arrest, seizure activity and respiratory distress. Morbidity rates are variable and the recovery process can take many years. In Dalmau, et al., (2008) series of 100 individuals, close to 50

made a full recovery, 28 experienced mild long-term deficits and 18 experienced severe deficits. Of these deficits, frontal lobe dysfunction was the most prominent with 85% of individuals in this, and subsequent series, experiencing some degree of dysfunction (Dalmau, et al., 2008; Finke et al., 2012). The goals of treatment are to improve concentration, judgment and problem-solving abilities. At this level, individuals are involved in their care and goal setting and activities can be focused around tasks of daily living such as accessing phone numbers, shopping and returning to their former employment or study. As individuals progress, their level of responsibility increases and in most cases they are able to return home with only minimal supervision.

Conclusion

The management of individuals diagnosed with anti-NMDA receptor encephalitis remains unclear. As there are few guidelines, care and interventions are currently focused on medical interventions with limited literature to support the role that neuroscience nurses play in people with this condition. This paper demonstrated the vital role that neuroscience nurses do play in caring for individuals diagnosed with anti-NMDA receptor encephalitis. It is vital that neuroscience nurses understand the natural history, available treatment options and interventions required for individuals diagnosed with anti-NMDA receptor encephalitis.

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The Tomorrow: Advanced Treatments in Parkinson's Disease Does Not Necessarily Equate to Treatments in Advanced Parkinson's Disease

David Shek-Yan Tsui

Abstract
A person with Parkinson's disease (PD) can have variations in the severity of Parkinsonian symptoms; this is dependent on the stage of their medication cycle. Variations in dopamine plasma levels cause erratic changes in severity of symptoms, known as 'motor fluctuations'. Apomorphine infusion, Duodopa® and Deep brain stimulation are the three current advanced treatments for people with PD. The goal of therapy is to replicate the normal function of the neurotransmitter dopamine by providing continuous stimulation in an effort to minimise motor fluctuations. Although these advanced treatment options seem like the ideal solutions for people with PD, awareness and knowledge is relative low amongst the neuroscience community.

Advanced PD treatments have the potential to improve the quality of life in people who are battling this chronic and complex disease. The aim of the presentation is to increase awareness amongst neurosciences nurses of modern treatments available to people with PD, enabling neuroscience nurse to advocate for their patients. This presentation will also highlight the role of the Parkinson's Disease Nurse Specialist in the assessment, diagnosis, management and ongoing follow-up of these patients who require specialised expertise.

Keywords: *Parkinson's disease, tremor, deep brain stimulation, dopaminergic, movement disorders, Parkinson's disease nurse specialist, motor fluctuations, "OFF" period, "ON" period, dyskinesia.*

Traditional Thinking Versus Contemporary Thinking

Parkinson's Disease (PD) was first described by James Parkinson in 1817 in the paper 'An essay on the Shaking Palsy'. He was able to document some key features of PD which are still relevant to this day. Some of the key features he described include the chronic degenerative nature of the disease, the predominance of unilateral symptoms, tremors and the latency in tremors, akinesia and bradykinesia, rigidity, stooped posture, postural instability, gait disturbance and freezing of gait and even non-motor symptoms of the disease such as sleep disturbance, speech disturbance and constipation (Parkinson, 1817 & 2002).

The traditional understanding of the pathophysiology of PD begins with the destruction of dopamine producing neurons in the substantia nigra located in the midbrain. These structures are part of the basal ganglia which is responsible for initiation and controlling voluntary movements. The reduced production and action of dopamine lead to reduced neuro-transmission and stimulation to the motor cortex causing motor dysfunction (McCance & Heuther, 2006).

However, the more contemporary thinking which is beginning to be more wildly accepted is known as the Braak's Hypothesis. The Braak's hypothesis proposes that there are 6 stages of PD. Stages 1 and 2 are the premotor or prodromal phase of PD which suggests the pathophysiology of PD begins in the gut causing gastrointestinal symptoms commonly manifesting as long standing constipation years before the diagnosis of PD. The progression of the pathophysiology into the medulla causes the autonomic dysfunctions such as temperature imbalance, postural hypotension, urinary and erectile dysfunction. Further progression to the pons causes the sleep disturbances such as REM sleep behaviour disorders, insomnia and day time somnolence (Braak, Del Tredici, Rüb, de Vos, Jansen Steur & Braak, 2003).

Stages 3 and 4 are classed as the motor or clinical phase of PD when the midbrain is affected causing the manifestations of the motor symptoms such as tremor, rigidity, akinesia and postural imbalance which allows for the clinical diagnosis of PD to be made (Braak et. al. 2003).

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Stages 5 and 6 are caused by further progression of the pathophysiology into the forebrain and the rest of the cortex which leads to the cognitive impairments such as dementia, hallucinations and psychosis and can exacerbate pre-existing anxiety and depression (Braak et. al. 2003).

The 'OFF' & 'ON' Phenomenon and Dyskinesias

People with Parkinson's disease may have different phases that their body is in depending on the stage of their medication cycle. This is commonly known as motor fluctuations. In their "OFF" period or pre-medicated state, the person may have typical motor symptoms of PD and in their "ON" period or their medicated state, the same person may well be functioning close to the level of a normal person with minimal interruptions from their Parkinson's symptoms due to having optimised dopaminergic plasma levels. There is also a third state when the dopaminergic plasma levels peak and break through the therapeutic threshold of the "ON" state causing excessive chorea-like movements. These movements are involuntary, twisting or writhing movements which can affect any part of the body. This is known as peak-dose dyskinesia (Frabbrini, Brotchie, Grandas, Nomoto & Goetz, 2007).

Contemporary Concepts of Treatments in PD – Continuous Dopaminergic Stimulation

The control and balance of motor fluctuations within the therapeutic threshold is dependent on accurate titration of the individualised medication regimen, strict compliance and accurate timing of medication administration. However, when the symptoms are refractory to these strategies, advanced treatments in PD are considered (Hayes, Fung, Kimber, & O'Sullivan, 2010). The common misperception of advanced treatments for PD is that they are usually reserved for patients in their later or advanced stages of the disease process. However, these advanced treatments can be implemented in the earlier stages of PD and patients can benefit from earlier interventions.

The concept behind the advanced treatments in PD is referred to as 'continuous dopaminergic stimulation' where medication or stimulation is administered in a constant fashion to provide stability and continuity to prevent the sudden and pulsatile fluctuations in dopaminergic plasma levels (Nutt, 2007).

Contemporary Treatments

Despite the advancement in technology and the development of advanced treatments, some of the older treatments used in PD are still currently in use today. Oral Levodopa, in the form of Sinemet® (Levodopa/Carbidopa) or Madopar® (Levodopa/Benserazide) continue to be the gold standard treatment in PD since the 1960's (Hornykiewicz, 2010). The introduction of dopamine agonists such as Sifrol® and Cabaser® (Cabergoline) and enzyme inhibitors such as Comtan® (Entacapone) and Azilect® (Rasagiline) have assisted in maintaining and/or prolonging 'ON' periods and better management of PD symptoms (Hayes et. al. 2010).

The development of combination drugs such as Stalevo® (Levodopa/Carbidopa/ Entacapone) and once daily extended release tablets such as Sifrol ER® (Pramipexole Extended Release) have enhanced simplicity and convenience in the patient's often complex medication regimen (Hayes et. al. 2010).

Delayed gastric emptying in patients with PD often leads to sporadic absorption of oral PD medications which can worsen motor fluctuations. As a result, more contemporary treatments attempt to bypass the gastrointestinal tract. The Neupro® (Rotigitine) patch, for example, is a form of dopamine agonist made into a daily 24 hour patch and is the only transdermal treatment currently available in the management of PD (Hayes et. al. 2010).

The Advanced Treatments in PD

Currently there are three advanced treatments in Parkinson's Disease based upon the concepts of providing stable and continuous dopaminergic stimulation to prevent the sudden and unexpected fluctuations in dopaminergic plasma levels. These treatments include Apomorphine, Duodopa® and Deep Brain Stimulation (DBS) (Hayes et. al. 2010).

Apomorphine is a subcutaneous injection and is the only injectable treatment available to manage PD. Apomorphine is a dopamine agonist that works to stimulate the dopamine receptors in the substantia nigra, more specifically the D1 and D2 receptors. It can be given as an intermittent rescue injection using a heparin/insulin syringe or via continuous subcutaneous infusion. Although it is derived from the morphine molecule it has no narcotic, analgesic or addictive properties. The dilution and infusion rates on the pump are pre-set and individualized to each patient

by a neurologist and commonly in conjunction with a Parkinson's Disease Nurse Specialist (PDNS) (Hayes et. al. 2010). .

Duodopa® is an intestinal gel of Levodopa/ Carbidopa (20mg/5mg/ml) enclosed in a 100 ml cassette delivered via an infusion pump for the treatment of Parkinson's Disease. The Duodopa® pump infuses the medication directly into the duodenum or jejunum via a Percutaneous Endoscopic Gastrostomy (PEG) tube with a Percutaneous Endoscopic Jejunostomy (PEJ) threaded through it. The effect is superior to oral medications because the absorption of the medication is optimal in the duodenum or jejunum. The infusion rates on the pump are also pre-set and titrated individually for each patient (Hayes et. al. 2010).

Deep brain stimulation is a neurosurgical option in the treatment of Parkinson's Disease. The treatment involves stereotactic insertion of electrodes into target sites in the brain, usually the subthalamic nucleus or globus pallidus, and connecting the electrodes to a pacemaker to control the abnormal firing of neurons by delivering steady and continuous electrical impulses to the target sites (Hayes et. al. 2010).

The Role of the Parkinson's Disease Nurse Specialist

With the development of advanced treatments in PD, there is growing complexity and subspecialisation of these treatments. Nursing assessments and interventions are vital in the initiation, implementation and ongoing management of advanced treatments in PD. Pre and post treatment assessments including the use of the Unified Parkinson's Disease Rating Scale (UPDRS), cognitive assessments, quality of life assessments and other psychosocial assessments require the expertise, detailed attention and dedicated time of a specialised PDNS. With the thoroughness of the nursing assessment, the PDNS becomes the ideal person to determine and profile which patient is appropriate for which advanced treatment based on treatment goals, lifestyle wants or needs and comorbidities.

The PDNS is also in the perfect position to be the patient's point of contact and liaison with the neurologist for medical and psychosocial support and also for follow up purposes. These may include issues such as troubleshooting device malfunctions, side effects monitoring and coordinating with other allied health professionals of the multidisciplinary team to manage the multifaceted aspects of PD.

The Future of the Parkinson's Disease Nurse Specialist Role

With the growing development of technology and advanced treatments, there is a growing need for the expertise of Parkinson's Disease Nurse Specialists. However, the majority of PDNS in New South Wales are funded only at a part time capacity and majority by pharmaceutical companies and/or research grants and therefore ongoing funding cannot always be guaranteed. Formal education programs or courses are in the development process but still in preliminary stages and there is a need for accreditation programs to ensure that PDNS are practicing according to a standardised and evidence based guidelines.

An audit performed by the World Health Organisation (WHO) in collaboration with European Parkinson's Disease Association (EPDA) and International Council of Nurses (ICN) revealed similar responses from current PDNS practicing in numerous different countries. They conclude with a consensus view that there is a lack and a need for uniformity in formal education courses and established career pathways for the development of a PDNS. A good model for the Australian PDNS to follow would be to examine the role of the PDNS in the United Kingdom of which the role was first described in the early 1990's (European Parkinson's Disease Association, 2009).

Conclusion

The field of Parkinson's Disease and Movement Disorders are growing in complexity and specialty as technology and the development of advanced treatments become more prominent and accessible. The role of the PDNS and the multidisciplinary team are a vital part of this process and there is a need for more stable government funding and accredited education programs for the development of the role of the PDNS.

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Defying the Odds of Gravity: The Case of the Sinking Skin Flap

Rhiannon Carey

Abstract
For many years, neurosurgeons have performed decompressive craniectomies for the relief of intracranial hypertension. It involves temporarily removing a large segment of skull in order to accommodate for the rising pressure inside the head. Many studies have shown that the procedure is effective in reducing intracranial pressure (ICP) and improving cerebral blood supply and the flow of cerebrospinal fluid (CSF). Nurses are aware that all surgical procedures carry a degree of risk of complications to occur. When thinking about craniectomy, the most common complication would probably be infection.

However, what of the sinking skin flap? The Sinking Skin Flap Syndrome (SSFS) is a complication from decompressive craniectomy characterised by symptoms such as headache, seizures, motor deficits and paralysis. It occurs when the skin covering the segment of missing skull literally sinks down onto the brain tissue and causes direct pressure on the cortex. Theories for why this condition occurs are endless; however studies have shown that the neurological symptoms associated with SSFS vastly improve when a cranioplasty is performed.

Neuroscience nurses are constantly on the lookout for infection and preventing injury to the unprotected brain tissue when their patient has had a decompressive craniectomy. However, how aware are they about the complication of the sinking skin flap and its' potential to cause neurological deterioration? This literature review will explore the case of the sinking skin flap and highlight the need for nurses to be more aware of its' potential impact on an individual's recovery.

Keywords: *sinking skin flap syndrome, decompressive craniectomy, syndrome of the trephined, cranioplasty, complications.*

Introduction

The decompressive craniectomy was first carried out in approximately 4000BC, when the pre-Colombian Incas performed trepanation on infants that were epileptic (Kakar, Nagaria & Kirkpatrick, 2009). The word "trepanation" originates from the Greek word "Trypanon", meaning "to bore". The concept of trepanation has been the interest of brain surgeons since the early 1860's (Finger & Clower, 2001).

Craniectomies were first performed using a piece of equipment called a Trephine; "a cylindrical or crown saw used in the removal of a disc or bone" (Gadde, Dross & Spina, 2012, pg 213). The first trephined skulls were discovered in Peru in the 1860's and studied by a man named Paul Broca, the same man that discovered the area of speech in the brain. He theorised that by the look of the scar tissue surrounding the holes in these skulls, each of the people to whom they belonged had lived considerably past the time that the holes were actually made (Gross, 1999). As a member of the Paris Surgical Society, Mr Broca brought his theory forward. Unfortunately, other members of the Society could not comprehend that such primitive man could trephine the skull of another living human being and not cause death in the process. Overall, it took seven years for Broca's theory to be proved (Gross, 1999).

Alongside Paul Broca was a man named Victor Horsley, who also took interest in the concept of trephining. Whilst Broca's interest in the concept was life-long, Horsley's interest was more of a passing craze. Either way, the contribution of theories put forth by Horsley was well regarded and, between the two men, the surgical procedure of craniectomy came about (Gross, 1999).

Case Study

Mrs V (pseudonym) is an 89 year old female

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who has an 11 year affiliation with the neurosurgical department of a large teaching hospital. Mrs V was first admitted in 2002 with a large intracerebral haemorrhage in her right fronto-parietal lobe. Treatment for this involved a craniectomy and a lengthy stay in hospital, followed by a cranioplasty later the same year.

In 2010 Mrs V was readmitted with complications of hydrocephalus, for which a Ventriculo-Peritoneal (VP) shunt was inserted. At the same time, a reconstructive cranioplasty was performed. Then late in 2013 Mrs V was readmitted for a third time due to an infected shin, which spread to her cranioplasty site and infected the bone flap. Mrs V underwent a third craniectomy to remove the infected bone. Due to the infection testing positive for Methicillin-Resistant Staphylococcus Aureus (MRSA), performing a cranioplasty was not appropriate at this time. Instead, Mrs V was commenced on antibiotics, fitted with a protective helmet and transferred back to her nursing home.

In early 2014, Mrs V was readmitted to the neurosurgical department for a fourth time. The handover from her nursing home was that she had developed a left-sided weakness approximately 6 weeks prior, which was worsening over time. On initial examination, Mrs V was lying in a supine position and was able to spontaneously move both her left arm and leg. She had full flexion in both the knee and elbow. However, when trying to sit Mrs V up, she immediately developed a left hemiparesis. Mrs V was incapable of holding herself up. All that could be elicited from Mrs V's left side was a response to painful stimuli. Mrs V scored a Glasgow Coma Scale (GCS) of 14/15 and had a systolic blood pressure of 110mm/Hg. She was noted to have an extensive cardiac history and was currently taking warfarin. A Computed Tomography (CT) scan was immediately undertaken. The result of the scan showed slight mid-line shift however, the cause for concern was the area where the bone flap had previously been removed. A two-degree compression of Mrs V's right hemisphere had occurred due to the skin flap sinking down onto it
(see Figure 1).

Mrs V was diagnosed with "Sinking Skin Flap Syndrome", otherwise known as the "Syndrome of the Trephined". Mrs V was to be managed lying flat, as per her surgeon's orders. She was allowed to sit for meals; however as soon as the left-sided paralysis began to appear, she was to lie flat again thus relieving pressure and lessening the hemiplegic

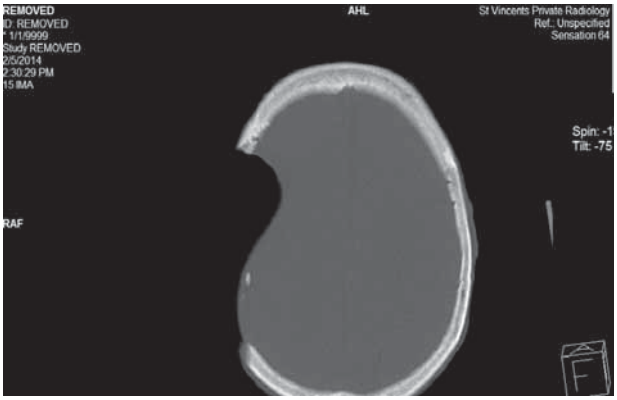

Figure 1: *(Above) Compression of the right cerebral hemisphere, due to the skin flap sinking inward. (Compliments of St Vincent's Public Hospital CMMI Melbourne).*

symptoms. A cranioplasty was the required treatment as this would allow relief from the compression on the brain tissue. A synthetic bone plate was ordered, antibiotics were commenced and the warfarin was ceased. Surgery was planned for 1 week's time.
Post surgery, Mrs V showed significant improvement. Her left-sided paralysis was beginning to resolve and she was capable of performing activities of daily living that she was unable to do prior to the cranioplasty. A

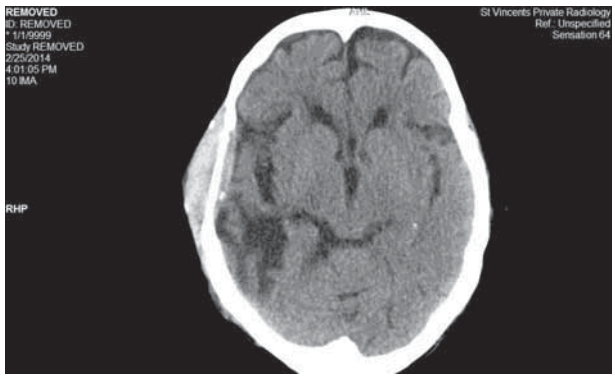

Figure 2: *(Above) Post cranioplasty- a synthetic bone plate now resides where the missing bone once was. Swelling on the outer edge of the plate is noted. (Compliments of St Vincent's Public Hospital CMMI Melbourne).*

CT (see Figure 2) showed that Mrs V's brain tissue was much less compressed and the midline shift had resolved. Despite there being some swelling and slightly more gliotic, non-functioning tissue than was present before the surgery, Mrs V recovered well and was able to transfer back to her nursing home.

Discussion

The Syndrome of the Trephined is a delayed complication of a craniectomy. Generally, it will take place more than 8 weeks post the

procedure (Staykov, Huttner, Kollmer, Ganslandt, Doerfler, Schwab & Kohrmann, 2010). There are a number of symptoms one may experience with this condition and quite often they may go unnoticed. In many instances, symptoms may be mistaken for those related to the underlying brain insult for which the craniectomy was first indicated. Such symptoms include dizziness, headache, irritability, seizures, depression, behavioural changes and focal deficits (Staykov et al., 2010). It has been shown that the symptoms often worsen when a person is upright and ease off slightly when a person lies flat. It has been suggested that the Syndrome of the Trephined is more common in stroke victims, however due to the fact that craniectomies are mainly performed on individuals who have had a stroke or endured a traumatic brain injury, it is difficult to know if it would occur in those with other conditions for which craniectomy is not indicated (Staykov et al., 2010).

Syndrome of the Trephined occurs because the atmospheric pressure exceeds intracranial pressure (Sarov, Guichard, Chibarro, Guettard, Godin, Yelnik, George, Bousser & Vahedi, 2009). Increased pressure outside the skull creates a force that pushes down on the skin flap post craniectomy, causing it to collapse down onto the underlying brain tissue. Whether an individual experiences symptoms depends on the amount of pressure being exerted (Sarov et al., 2009).

Research suggests two consistent theories supporting why a sinking skin flap can cause neurological deficits. Kemmling, Duning, Lemcke, Niederstadt, Minnerup, Wersching & Marziniak (2010), suggest that when an area of brain tissue becomes compressed, the amount of blood flow to that area is restricted. This leads to a decrease in oxygen supply and less blood entering the venous system, hence a reduction in neurological function. Gadde, Dross & Spina (2012), support the notion that defects in the skull create a siphoning or turbulent effect on the flow of CSF. The change in CSF flow then affects the amount of blood supply to the area, leading to a decrease in neurological functioning. Symptoms of a sinking skin flap can be aggravated by CSF diversion, dehydration and/or changes in position (Sarov et al, 2009).

There are minimal statistics available indicating the incidence and prevalence of the Syndrome of the Trephined, but what is available appears to be low. A study carried out by Yang, Wen, Shen, Li, Lou, Liu & Zhan (2008), followed 108 patients who underwent a craniectomy post head injury. Of those, 14 (13%) were later diagnosed with the Syndrome of the Trephined. The study found that older patients and those with more severe head trauma were at a higher risk for developing the condition. A second study conducted by Sarov et al., (2009), analysed 27 patients undergoing a craniectomy post hemispheric infarction. 3 (11%) of those were later established to have sinking skin flaps.

It is important to understand the symptoms of a sinking skin flap because, if left untreated, it can go on to cause “Paradoxical Herniation”. This condition is described when the brain herniates down through the tentorial notch as a consequence of extremely low pressures inside the skull (Staykov et al., 2010). Management of herniating brain tissue is generally achieved by draining excess CSF, hyperventilating the patient and administering Mannitol - all indicators for a high pressure state. However, paradoxical herniation is a low pressure state, therefore management would involve preventing CSF drainage, hydrating the patient and lying the patient down to try and normalise the pressures (Gadde et al., 2012).

Ultimately, the only way to treat the Syndrome of the Trephined is to perform a cranioplasty. Conservative management alone has been found to be largely ineffective (Bhat, Kirmani, Nizami, Kumar & Wani, 2011). Mokri (2010) proposes that the symptoms of the Syndrome of the Trephined will completely resolve after a cranioplasty is performed. Trnovec, Halatsche, Behnke-Mursche & Mursche (2009), agree that neurological improvement is frequently observed after bone flap reimplantation occurs. Furthermore, increased cerebral blood flow post cranioplasty suggests that lack of blood flow may be an underlying mechanism for why a sinking skin flap causes neurological deterioration (Sarov et al., 2009).

The Syndrome of the Trephined has a large negative impact on patients who are trying to rehabilitate after a craniectomy. Many patients do not regain full physical and/or cognitive function after the procedure, which in the long-term, creates problems with the psychosocial impact of recovery (Hossain-Ibrahim, Tarnaris & Wasserberg, 2011). The challenge of rehabilitation post craniectomy is hard enough, without having to endure the effects of a sinking skin flap. Archavlis & Nievas (2011), report that the rehabilitation of someone who has suffered a sinking skin flap is significantly prolonged because they must undergo periods of

immobility which potentially could lead to increased rates of thrombolytic events and pulmonary infection. A lack of knowledge of the Syndrome of the Trephined by healthcare workers means that the symptoms of the condition often go unnoticed (Kemmling et al., 2010). If awareness of the condition can be raised, patients may be treated more quickly and more accurately, minimising the delay in their rehabilitation (Janzen, Kruger & Honeybul, 2012).

Conclusion

The Syndrome of the Trephined is a delayed complication of craniectomy, generally occurring more than eight weeks post procedure. The condition is largely characterised by headache, dizziness, mood or behavioural disturbance and focal deficits. Changing position, particularly into an upright position, appears to make the symptoms worse. It is also suggested that there is a higher rate of incidence in the older patient and those with larger craniectomy sites. The neurological deficits associated with a sinking skin flap are a result of poor blood flow and oxygen supply to the craniectomy site and the only way to treat the deficits is to perform a cranioplasty. The Syndrome of the Trephined is not well known or understood; therefore the delays in recovery associated with the condition are extensive. Prevention of such setbacks in patient rehabilitation is to raise awareness of the complication of the sinking skin flap and ultimately improve care.

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Falls Prevention Strategies among acute Neurosurgical in-patients: a Best Practice Implementation Project

Kylie M Wright

Abstract
Patient falls are a significant problem and one of the most common adverse events experienced in hospitals. Falls are considered an indicator of the quality of nursing care that requires uncompromising attention and they have been recognised by the Australian Commission on Safety and Quality in Health Care as a National Standard.

This project aimed to conduct an audit of in-hospital falls-prevention practices, to implement evidence-based best practice recommendations and to increase staff compliance within an acute neurosurgical unit. The Joanna Briggs Institute's Practical Application of Clinical Evidence System and Getting Research into Practice audit tool for promoting change in healthcare practice was used. A baseline audit was conducted measuring eight best practice recommendations, followed by the implementation of targeted strategies and follow-up audits.

The baseline audit revealed large gaps between current practice and best practice and overall performance was poor. Barriers for implementation of best practice falls prevention strategies were identified by the project team and numerous strategies were implemented. There were improved outcomes in the follow-up audits.

The findings showed how audits may be used to promote best practice in healthcare and that focussed education and provision of relevant resources can have an immediate impact on clinical practice. By the end of the project, attitudes to falls prevention had been 'transformed' from passive acceptance of falls to active engagement in falls prevention. Future audits are planned to ensure sustainability.

Keywords: *falls prevention; best practice; acute neurosurgical inpatients*

Background

Patient falls are a significant problem and one of the most common adverse events experienced in hospitals. Evidence-based best practice guidelines for preventing falls are available and provide specific information for Australian hospital settings. Most hospitals have fall prevention policies that include the use of falls risk assessment tools. However, despite access to these resources, many preventable falls continue to occur in Australian hospitals.

Patients admitted to hospital often have changes in physical or cognitive condition, and when combined with unfamiliar surroundings present a high risk of falling. Injuries resulting from falls can range from minor bruising to serious injuries such as intracranial haemorrhages, fractures and in some cases can lead to permanent disability or death. Falls rates in Australian acute care settings are reported to range from 2-5% per 1000 patient separations (Kannus, Khan, and Lord, 2006), with one health service area reporting more than 22,000 falls resulting in patient harm in a one year period, representing a rate of 2.5 falls per 1000 patient separations, with a higher rate in public hospitals (3.3) than in private hospitals (1.3) (Australian Institute of Health and Welfare , 2012).

The impact of falls on patients and the added costs to the healthcare system are significant. Patients may experience decreased physical activity related to the fear of further falls (Suzuki, Sonoda, Misawa, Saitoh, Shimizu, and Kotake, 2005), decreased falls self-efficacy (the belief that one can independently ambulate without falling), a diminished sense of dignity (Rapport, Hanks, Millis, and Deshpande, 1998), an increased length of hospital stay, a reduced quality of life, and emotional distress (Department of Health and Aging,

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2013). Furthermore, the cost to the community and increase on the demand for health services for falls-related injuries is considerable. It has been predicted that unless effective preventative strategies are utilised, the cost attributable to falls-related injury will increase three-fold to $1375million per annum by 2051 (Department of Health and Aging, 2013).

Multiple risk factors that can contribute to in-hospital falls have been identified in the literature and include patient characteristics, staff behaviour and the hospital environment (Stern and Jayasekara, 2009). Patient risk factors include advanced age, muscle weakness, gait or balance problems, visual impairment, altered bowel or bladder elimination patterns, dizziness or vertigo, depression, cognitive deficits, impaired activities of daily living, use of psychotropic medications and a history of falls (Hendrich, Bender, and Nyhuis, 2003; Rubenstein and Josephson, 2002; Oliver, Britton, Seed, Martin, and Hopper, 1997; Leipzig, Cumming, and Tinetti, 1999; Tinetti and Kumar, 2010). Neurosurgical patient groups experience many of these identified risk factors, with falls rates of 4-12 per 1000 bed days in patients over the age of 65 years and more than 40% of patients with specific clinical neurological problems experiencing one or more falls during their hospital admission (Australian Commission on Safety and Quality in Health Care, 2009).

A large number of interventions for preventing in-hospital falls have been recommended in the literature. These include early detection and treatment of conditions such as incontinence, eyesight problems and delirium, reviewing medication regimes, providing safe non-slip footwear to patients and modifying the environment with things such as handrails and non-slip flooring (Stern and Jayasekara, 2009). Current literature recommends a comprehensive and multi-factorial methodology to falls prevention, involving the use of risk assessment tools and targeted interventions (Stern and Jayasekara, 2009; Australian Commission on Safety and Quality in Health Care, 2009; Cameron, Gillespie, Robertson, Murray, Hill, Cumming, et al, 2012; Oliver, Healy, and Haines,2010).

This project aimed to conduct an audit of in-hospital falls prevention practices, to implement evidence-based best practice and assess the effects of these strategies at minimising in-hospital falls. The project was undertaken in the neurosurgical unit of a large 855 bed public, tertiary referral hospital and major trauma centre in Sydney, Australia. The hospital has a strong commitment to teaching and research across a wide range of disciplines and serves between 1.3 and 1.4 million people in the South West of Sydney, with the most culturally diverse population in the state,39% of people from non-English speaking backgrounds (NESBs).The hospital has well established policy directives on falls prevention and management for inpatients including a falls risk assessment tool embedded into the electronic medical record, patient/carer falls brochures in six different languages, established 'falls champions' in clinical areas, patient falls risk assessment guidelines and prevention strategies endorsed by the New South Wales (NSW) Clinical Excellence Commission (CEC). Despite a culture of falls prevention awareness and established resources, the 2012 falls rates for the neurosurgical unit was 6.44 per 1000 beds (Liverpool Hospital. Incident Management System).

Not only are patient falls one of the most common adverse events experienced in hospitals, but such incidences are considered an indicator of the quality of nursing care that require uncompromising attention. The Australian Commission on Safety and Quality in Health Care (ACSQHC) has recognised the prevention of falls and harm from falls as a National Standard (Australian Commission on Safety and Quality in Health Care,. 2012). In brief, this standard requires that health service organisations have governance structures and systems in place to reduce falls and minimise harm from falls; that patients on presentation, during admission and when clinically indicated are screened for risk of a fall and the potential to be harmed from falls; and that patients and carers are informed of the identified risks from falls and are engaged in the development of a falls prevention plan. Overall, the aim of this standard is to reduce the incidence, or number, of patient falls and minimise harm from falls when they occur, hence in-patient falls prevention is very much a priority of modern Australian healthcare.

Objectives

This project aimed to conduct an audit of in-hospital falls prevention practices, to implement evidence-based best practice and assess the effects of these strategies at minimising in-hospital falls in a neurosurgical unit in a large tertiary hospital. The overall purpose of the project was to increase staff compliance with falls prevention best-practice to prevent in -hospital falls amongst at-risk patients. Objectives included:

- To improve the local practice of completing patient falls risk assessments appropriately, accurately and in a timely manner.

- To ensure health care professionals have been educated regarding falls assessment and prevention strategies and targeted interventions have been implemented.

- To ensure patient and family education regarding falls is conducted.

An evidence-based practice approach underpins the entire implementation project.

Methods

The project used the Joanna Briggs Institute (JBI) Practical Application of Clinical Evidence System (PACES). JBI PACES is an online tool for health professionals and/or researchers to use for collection and comparison of data and to conduct efficient audits in small or large healthcare settings. PACES has been designed to facilitate the use of audits to implement evidence based health practice and includes the Getting Research into Practice (GRIP) framework that may be used to help identify factors underpinning gaps between practice and best practice and strategies to overcome them. The project involved three phases as follows.

Phase 1—Baseline audit

A baseline audit of in-hospital falls prevention practices was conducted. A multidisciplinary team of key stakeholders was formed to support the work of this project. The lead author of this article, is the neurosurgical clinical nurse consultant at the hospital where the project was implemented, and led the project as part of the JBI Clinical Fellowship program (Joanna Briggs Institute, Adelaide, South Australia). Involvement of the project team was in varying capacities of support, data collection, data entry and/or participation. Patients/ consumers were also inclusive of the team at a participatory level.

The objectives of the baseline audit were to establish the size and nature of the gap between practice and best practice in falls prevention strategies on the neurosurgical unit. The JBI best practice recommendations, related to falls assessment and preventative interventions, are based on a structured search of the literature and selected evidence-based health care databases. Eight criteria based on these recommendations were audited throughout this project. The eight identified criteria for data collection were divided into assessment, education and intervention categories and were measured as follows:

Assessment

1. Fall risk assessment is done upon admission.

This criterion was considered met if the case notes/electronic medical record (EMR) showed a risk assessment completed within four hours of admission.

2. Fall risk assessment is done upon transfer

This criterion was considered met if the case notes/EMR for patients that have been transferred (intra-hospital transfer) show a risk assessment completed within four hours of transfer.

3. Reassessment occurs when there is a change in condition or following a fall.

This criterion was considered met if the case notes/EMR for patients who have had a change in clinical condition (that affects their falls risk status) or experienced a fall, included a reassessment performed within four hours of this event.

4. Patients who have experienced a fall are considered at high risk for future falls

This criterion was considered met if it was documented in the case notes/EMR for patients who have had a history of falls, are assessed as high risk for future falls according to the risk assessment.

5. Fall risk assessment is done accurately using a falls assessment tool

This criterion was considered met if the case notes suggest the fall risk assessment was done accurately. If the accuracy of the risk assessment is not clear from the notes, then the patient can be visited to determine the accuracy of the assessment.

Education

6. Healthcare professionals have received education regarding falls assessment and prevention strategies

This criterion was considered met if staff members in the participating wards report that they have received education in the last two years. Question: “Have you received education regarding falls assessment and prevention strategies in the last two years?” This is by convenience sampling. Sample: 30 healthcare staff.

7. Patient and family education is carried out for patients at risk of falls

This criterion was considered met if from the case notes, for patients at risk of falls, there is documentation that patient and family education has occurred.

Intervention

8. Targeted interventions are implemented according to risk factors

This criterion was considered met if it is documented in the case notes for patients assessed as at risk, that there has been implementation of targeted interventions to address every identified risk factor.

The baseline audit was conducted over a 4 week period. To assess the compliance of each audit criterion, the case notes/EMR of 30 neurosurgical inpatients were examined. To assess compliance for the audit criteria regarding staff education, 30 clinicians working on the neurosurgical ward were interviewed by the project lead.

Phase 2 – GRIP (Getting Research Into Practice) Strategy

The objectives for the second phase of the project were to gain an understanding of the barriers underpinning gaps between practice and best practice found in the baseline audit and to implement tailored strategies to close gaps and address barriers. Using the PACES program, baseline audit results were collected for analysis and discussion by the project team and proposed strategies for improving compliance of falls prevention best practice were identified.

Open communication and engagement with all stakeholders was maintained and welcomed at all times throughout the project and provided the platform to suggest and discuss strategies for improvement. Via a series of face-to-face meetings using practice development principles and e-mail correspondence between the project team, each best practice criterion was reviewed and strategies for improved compliance were discussed. Furthermore, potential barriers and strategies to overcome such barriers, as well as resources required to implement change strategies were identified, discussed and formally documented.

A GRIP report matrix was generated which kept the project team informed, as well as provided a means of gathering and recording their opinions and clearly outlining the implementation plan and the team involvement. As described further in the results section, a major strategy identified to close the gap between practice and best practice was educating clinicians on best practice falls prevention strategies. This education, along with other strategies, was implemented during Phase 2 of the project which was conducted over a 4 month period.

Phase 3—Follow-up audits- Cycle 1 & Cycle 2

The objectives of the follow-up audits were to assess whether there had been improvement in compliance with best practice; to establish if improvements, if any, had been sustained, and identify remaining areas where further improvements are required. Cycle 1 and Cycle 2 post-implementation audits and collection of data were repeated using the same eight criteria defined in Phase 1. There were no variations to the topic, the criteria, the sample size, the characteristics or location of the project during the follow-up cycles.

The follow-up data was entered into the PACES program and data analysis comparing follow-up results with those of the baseline audit were undertaken to examine any change in compliance rates. Phase 3 was conducted over a 4 week period on both occasions.

The project received formal approval by the South Western Sydney Local Health District Human Research and Ethics Committee (NSW).

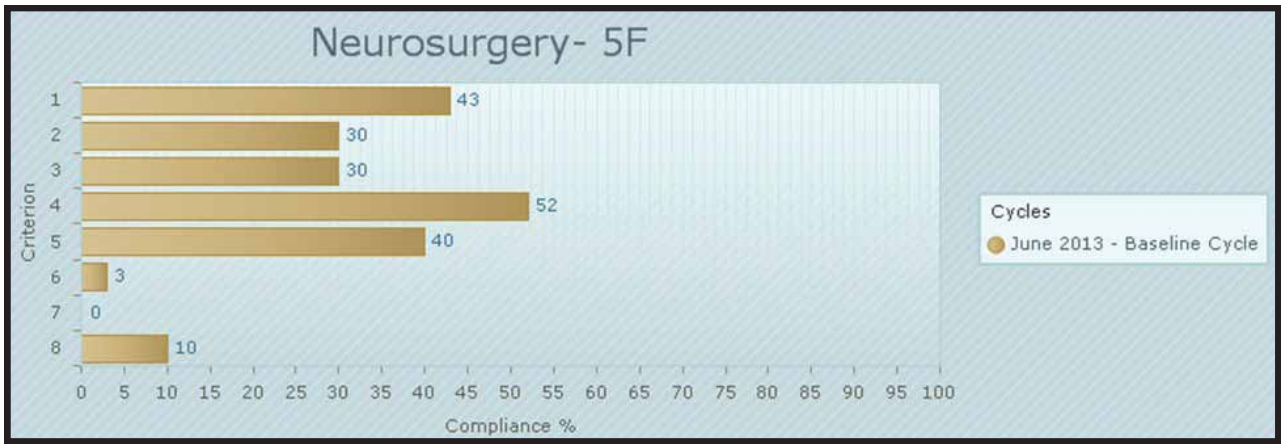

Figure 1: *(Above) Baseline audit results for the neurosurgical ward*

Results—Baseline Audit

The percentages for compliance with each audit criterion in the baseline audit for the neurosurgical ward are shown in Figure 1. The best baseline performance was found for criterion 4, which measured that patients who had experienced a fall were considered at high risk for future falls. In 52% of cases there was evidence of this practice.

Performance emerged as very poor in the baseline audit for the remaining criteria. Only 30% of occasions showed evidence that the fall risk assessment was done upon patient transfer (Criterion 2) and/or when there was a change in condition or following a fall (Criterion 3). There was evidence that only three patients had targeted interventions implemented according to risk factors (Criterion 8), and one healthcare professional, a medical officer, had received education regarding falls assessment and prevention strategies, resulting in very low compliance of 3% (Criterion 6). Of most concern, was that upon review of 30 patient medical records there was no documented evidence that patient and family education was carried out for patients at risk of falls (Criterion 7).

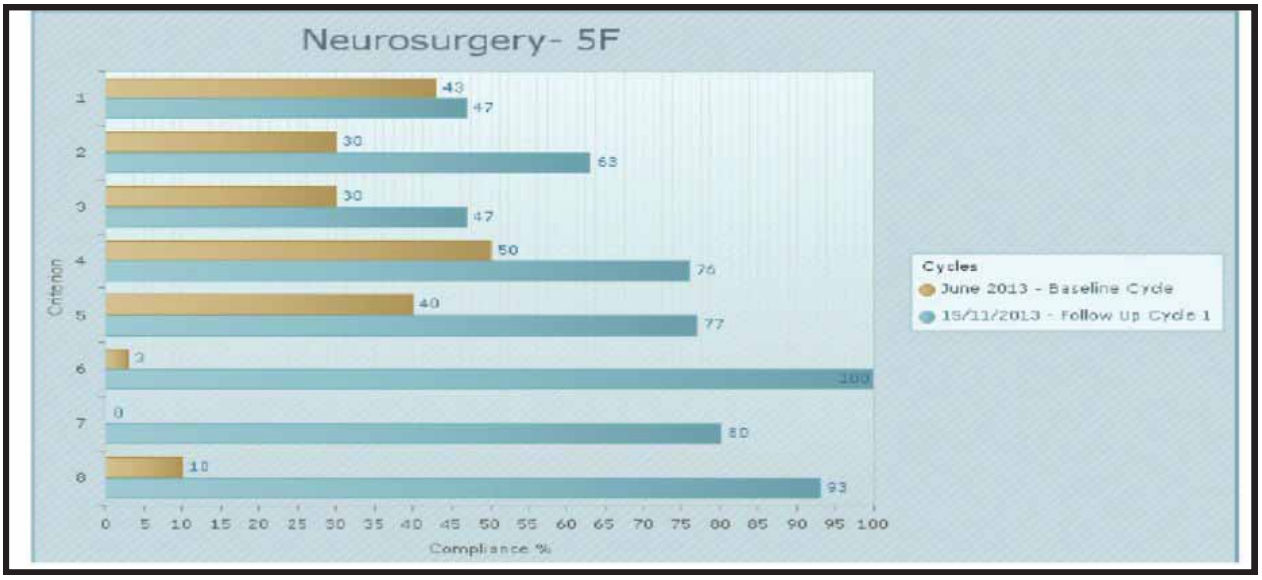

Figure 2: *(Above) Baseline and Follow-up Cycle 1 audit results for the neurosurgical ward.*

GRIP Strategy

Table 1 shows the barriers to best practice falls prevention strategies that emerged from the project team discussions of the Phase 1 results. It also shows resources identified as being required to implement the strategies and the outcomes.

Providing education to the clinicians was selected by the project team as the highest priority and the most feasible strategy to implement as a means of bringing best practice falls prevention strategies more in line with actual practice. A working party was formed, led by the project leader, to collaboratively develop, distribute and deliver a 'Falls Prevention and Management' education package incorporating best practice strategies and practical application.

To further build upon falls prevention awareness, an overview of falls risk factors, impact of falls, and falls incidents (case studies) that had occurred over the past 2 years were presented to nursing staff via formal presentations and analysis of falls incident reports were discussed at the ward level. The project team also engaged with clinicians and enabled essential practice changes to the documentation of targeted falls prevention strategies via implementation of a Falls Risk Assessment Management Plan (FRAMP) Form (Clinical Excellence Commission, 2013).

In addition, the hospital had patient/carer falls brochures in six different languages, however they needed updating and were very low on stock. The project leader sourced, copied and distributed the NSW CEC endorsed falls educational materials and engaged consumer representatives regarding appropriateness of this material. As a result, multi-lingual patient/carer falls education material encompassing 17 different falls related topics were made available to the bedside clinicians. The 'Falls Prevention and Management' education package informed staff of the range and location of educational material. Clinicians were encouraged to distribute such material to patients and families as a strategy to provide educa-

Barriers	Strategies
Staff nurses' lack of awareness and understanding of the extent of the 'problem' of inpatient falls and its importance in relation to the National Standards.	Overview of falls risk factors, impact of falls, falls incidents over the past 2 years (analysis of SAC1 & SAC 2 incidences) to be presented formally to nursing staff. Analysis of falls incident reports at ward level – present & communicate analysis to core ward groups and discuss findings. Build awareness by—Displaying falls rates "running tally", Commencement of ward "measles chart" Participate in interactive discussion & open communication
Staff nurses lack awareness and understanding of the evidence base for falls prevention strategies among in-patients	Build/promote awareness by—Education sessions/handouts; Signs in clinical areas; Distribution of baseline audit results; Focus groups; Environmental checklists Identify and engage ward based Falls Champions Promote awareness of JBI Best Practice Implementation Project- initiate as a regular agenda item on all ward based meetings. Participate in interactive discussion & open communication
No existing standardised hospital wide falls prevention education package	Engage hospital Falls Reference group & Falls Prevention Committee and collaborate with key stakeholders for input into education session content. Develop, distribute and deliver a 'Falls Prevention and Management' education package incorporating—Falls risk assessment; Management strategies; Post fall management including a case study to highlight each section. Gain Executive support for mandatory nursing attendance at educational session. Engage key stakeholders to deliver education package
A culture of 'weekly' falls risk assessments were embedded into clinical practice due to an existing weekly "falls score compliance" audit occurring routinely on Wednesdays.	Reinforce via education session situations when the falls risk assessments should be completed and re-assessed and in what time frame including use of the over-ride option Perform an audit from the EMR looking at compliance of falls risk assessments on all days of the week (not just Wednesdays), and analysis of times the assessments are being completed. Provide feedback on appropriate completion of falls risk assessments and use of the over-ride option.
Existing method of "flagging" high risk falls patients was often invisible and ineffective	Implementation of 'green' initiatives including—High risk fall sign above bed; Inserts to place in bedside folders; Post fall stickers to be placed in patient records Documentation of falls risk assessment score on electronic handover Falls assessment score included in all clinical handovers Agreement of dedicated "High visibility beds" – with placement of "flagged" high risk patients into such beds Development of a ward based "Falls Resource Manual" Provide feedback on appropriate "flagging" of high risk patients
Limited range of educational materials for falls prevention strategies available for patients and carers	Source, copy and distribute falls educational material and engage consumer representative regarding appropriateness of material. Source multi-lingual patient/carer falls education material. Inform nurses of range of educational material and encourage distribution to patients/carers as an education strategy. Development of a ward based "Falls Resource Manual"
No appropriate falls risk prevention strategy / management plan documentation structure to record targeted interventions in patient health care records	Make essential practice changes to documentation via implementation of a Falls Risk Assessment & Management Plan (FRAMP) form - falls care plan that highlights individual patient risk factors and records actions implemented To provide nurses with education regarding use of newly implemented FRAMP Development of a ward based "Falls Resource Manual" Provide feedback on appropriate completion of FRAMP
Reluctance of nurses to participate in the project due to increased workload and competing pressures	Engage the multidisciplinary team Use a practice development approach, to explore staff opinions and use their advice and ideas regarding implementation of best practice/patient centered care. Provide positive feedback and encouragement to improve fall prevention practices Make communication channels available Participate in interactive discussion & open communication Provide feedback on ward based falls rates per month

Table 1: (Above) – *GRIP (Identified barriers to best practice and strategies to overcome them)*

Barriers	Strategies
Continued lack of awareness and understanding across some health care staff of the evidence base for in-patients falls prevention strategies	Overview of falls risk factors, impact of falls, falls incidents and phase 3-cycle 1 results, and FRAMP utilisation to be presented formally to staff Build and promote awareness via continued education sessions provided to—Allied Health; Night duty & weekend staff; Non-clinical staff (targeted at ward orderlies, ward clerks, technicians etc); Consumer representatives; Medical Grand Rounds (targeted at medical students and all medical staff) Analysis of falls incident reports at ward/organisational level – present & communicate analysis to widespread committee groups and discuss findings & ongoing strategies. Participate in interactive discussion , open communication & action planning

Table 2: *(Above) GRIP (Identified barriers to best practice and strategies to overcome them).*

tion regarding falls prevention.

Follow-up Audits—Cycle 1

The percentage of compliance for the audit criteria found in the follow-up Cycle 1 audit, together with the results from the baseline audit is displayed in Figure 2. Looking at the results of the follow up audit, compared with those in the baseline audit, there has been an overall improvement in compliance of implementing best practice falls prevention recommendations.

The results suggest that whilst there has been an emphasis on staff education, this translated only partly into implementation in practice for some criteria. The criterion for performing a falls risk assessment upon admission (Criterion 1) remained reasonably static; however the remaining seven criteria showed improvements. The criteria measuring if healthcare professionals had received education regarding falls assessment and prevention strategies (Criterion 6), if there was documented evidence that patient and family education was carried out for patients at risk of falls (Criterion 7), and if there was evidence that patients had targeted interventions implemented according to risk factors (Criterion 8) showed the largest increase in compliance over baseline with improved changes of up to 97%.

Table 2 shows the barriers to best practice falls prevention strategies that emerged from the project team discussions after the phase 3 - cycle 1 results were analysed.

Supplying further education to the clinicians as well as education to non-nursing staff groups was selected by the project team as the next step and a more hospital wide approach was implemented in bringing best practice falls prevention strategies in the facility more in line with best practice.

Building further upon falls prevention awareness, an overview of falls risk factors, impact of falls, and falls incidents (case studies), and results of the Phase 3-Cycle 1 results were presented via a series of inservices and to a hospital wide nursing audience via the facility Nursing & Midwifery Grand Rounds. In addition, specific education was delivered to Clinical Nurse Educators (CNEs) and nominated ward Falls Champions in the form of a master class. The project team also continued engagement with clinicians and enabled essential practice changes to the documentation of targeted falls prevention strategies via implementation of the Falls Risk Assessment Management Plan (FRAMP) Form (Clinical Excellence Commission, 2013).

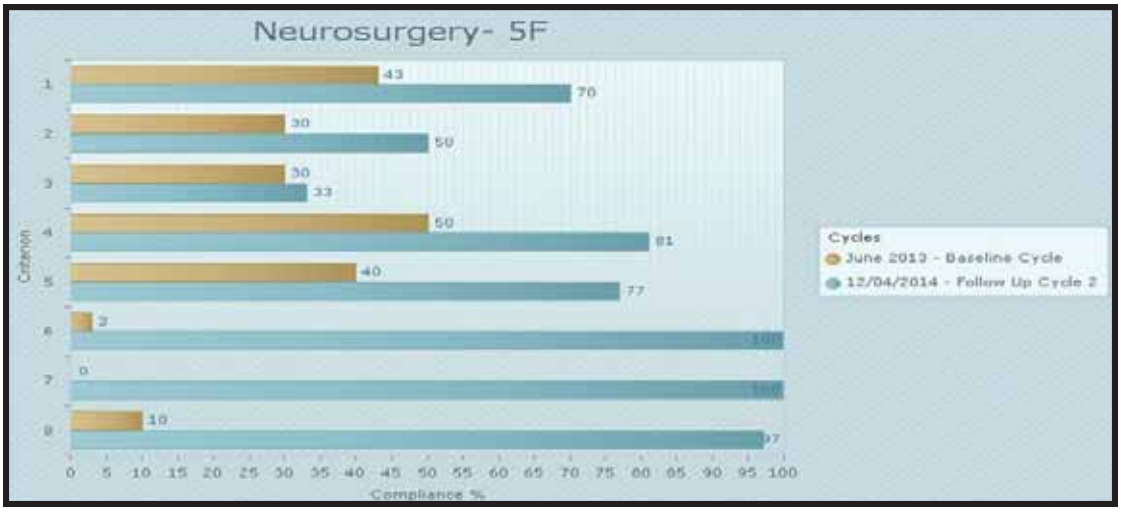

Figure 3: *(Above) Baseline and Follow-up Cycle 2 audit results for the neurosurgical ward*

Furthermore night duty and weekend nursing staff, as well as medical, allied health, non-clinical staff groups, and consumer representatives were also provided with targeted education.

An analysis and discussion of falls incidences were broadened beyond the neurosurgical ward to an organisational wide perspective. Falls incidences and prevention and management strategies were regular agenda items tabled at all committee meetings throughout the hospital.

Cycle 2

The percentage of compliance for the audit criteria found in the follow-up Cycle 2 audit together with the results from the baseline audit is displayed in Figure 3. Looking at the results of the follow up Cycle 2 audit, compared with those from the baseline audit, overall improvement in compliance of implementing best practice falls prevention recommendations has been maintained for most criteria.

The Cycle 2 follow up results suggest that improvements in compliance demonstrated in the Cycle 1 follow up audit were maintained, or further improved upon, for most criteria when the re-audit occurred 3 months later. The criterion measuring if fall risk assessment was done accurately using a falls assessment tool (Criterion 5), and the criterion measuring if healthcare professionals had received education regarding falls assessment and prevention strategies (Criterion 6) remained static. However the criteria for reassessment of falls risk occurring when the patient has been transferred (Criterion 2) and when there is a change in condition or following a fall (Criterion 3) slightly decreased. The remaining four criteria including performing the fall risk assessment upon admission (Criterion 1), deeming patients who have experienced a fall considered at high risk for future falls (Criterion 4), if there was documented evidence that patient and family education was carried out for patients at risk of falls (Criterion 7), and if there was evidence that patients had targeted interventions implemented according to risk factors (Criterion 8) showed further increases in compliance in the cycle 2 follow up audit with improved changes further increasing up to 23%.

Discussion

This best practice implementation project achieved improvements in compliance in all eight best practice recommendations in the neurosurgical ward over the nine month period. These practices, however had a more widespread impact on falls prevention and management across the organisation because strategies were adopted and supported across the hospital and had the underpinning support of the Executive and Governance structures. Medical Governance was obtained and was imperative to the success of this project, particularly in regards to medication reviews, diagnosis and management of delirium and treatment of orthostatic hypotension. This support is reflected in the improvement in compliance with the audit criteria and changes in practice.

The 'Falls Prevention and Management' education package incorporating best practice strategies and practical application was an effective strategy. The content of the package was divided into three sections and included principles in line with the best practice recommendations including how and when to do the falls risk assessment on the EMR, implementation and documentation of targeted management strategies via the FRAMP (Clinical Excellence Commission, 2013), engaging in patient education and post fall management. It also included a case study to highlight each section of the education package which could be modified to suit any clinical specialty. The package gave the staff the knowledge needed to prevent falls and to help their patients stay safe and was designed to be administered as a presentation in a one hour block, or could be given section by section in 20 minute timeframes for clinical areas that had difficulty releasing staff for extended education. Executive support was obtained, attendance was deemed mandatory and the education package was delivered across the facility initially over six consecutive sessions capturing 500 nursing staff.

Due to difficulties capturing all staff, CNE's within the facility were engaged in the education process and were given the education package as a PowerPoint presentation so it could be delivered to after-hours and weekend staff. In addition, they were empowered to take informal and formal opportunities to teach their ward colleagues to amplify and personalise the learning and make it ward specific. As a result, clinical staff across the facility received tailored education and are now more aware of the best practice recommendations for falls risk management and how to incorporate this into their clinical practice. Further education strategies and continued education at a ward and organisational level resulted in an additional 500 staff undergoing falls prevention and management education, hence by

the conclusion of the project 1000 staff had received targeted falls education.

Improvements in practice is evidenced by weekly "snap shot" falls risk assessment compliance scores across the organisation improving from 76% at the beginning of the project to an all-time high of 93.5% compliance within one month of the education package delivery. Furthermore falls rates for the neurosurgical ward have remained static throughout the study period however severity assessment codes of falls have improved across this high risk inpatient area.

The successful implementation of the FRAMP (Clinical Excellence Commission, 2013), impacted targeted falls prevention interventions organisation-wide. This tool incorporates a care plan that highlights individual patient risk factors and systematically records and evaluates the targeted actions that have been implemented. This meant that many more patients received individual assessments and care that is essential to preventing falls in hospital. The FRAMP (Clinical Excellence Commission, 2013), and its utilisation was introduced to clinicians via the 'Falls Prevention and Management' education package as well as one to one education administered at the bedside. Despite initial resistance and comments such as "not more paperwork", it was embraced by all clinical areas once its usefulness had been established and the form utilisation has now become embedded into the nursing admission process and ongoing practice.

Some best practice recommendations improved to a lesser degree in the follow up audits than others. The performance of a falls risk assessment upon transfer of a patient, and change in condition warrants ongoing attention. The primary resource required to improve these areas of practice is clinician time for re-assessment, and hence, this may partly be explained by clinicians feeling that their workload is too high to spend the additional time required to repeat the falls risk assessment when it may have only been recently performed. Falls prevention is one of a number of initiatives competing for staff and managerial time and with large, busy wards with a high turnover of patients and staff, such as the neurosurgical ward, problems with fulfilling some of the best practice criteria were challenging for the bedside clinicians. Furthermore, documented evidence is required for some criteria to be achieved. It is thought that through the awareness strategies implemented via this project that clinicians may have improved their practice in relation to falls management strategies, but do not always document their care hence the practice change is not recognised at any point of the audit. A lack of documentation does not necessarily indicate a lack of care, skill or knowledge and a future strategy will be to raise awareness of falls prevention related documentation and reporting of practice in the patients' EMR.

The reduction and prevention of falls is a quality initiative that is directly related to the National Standards. It is a priority for the facility to keep the focus on falls in its efforts to improve patient safety and continue to meet this standard. Implementation of the evidence through this project will be sustained by continued analysis and distribution of falls data, falls tally boards, environmental checks and 'measles charts' in clinical units. Facility policies and procedures have been revised to reflect changes in practice as a result of this project and multiple dedicated "Falls Champions" who have undergone specific falls champion training via a one day workshop have been appointed in each ward. Ongoing engagement with the Falls Prevention Committee will continue and the audit will be repeated annually.

Conclusion

It is indisputable that patient falls and patient fall related injuries are considered an indicator of inadequate nursing care and are currently one of the most worrying clinical issues amongst clinicians. Preventing falls and harm from falls has been recognised as a standard that must be met by the National Safety and Quality Health Service (NSQHS) Standards. A high priority needs to be given to the prevention of in-hospital falls in order to avoid poor patient outcomes.

The purpose of this project was to increase staff compliance with falls prevention best-practice within an acute neurosurgical setting. This included an audit of in-hospital falls prevention practices, implementation of evidence-based best practice, and assessment of the effects of implemented strategies in a Neurosurgical ward in a large tertiary hospital. The project succeeded in achieving the objectives as all criteria used to audit practice improved after a 'Falls Prevention and Management' education package incorporating best practice recommendations and various other strategies were implemented. Whilst it is suggested that the implementation of evidence based best practices will improve patient care and outcomes, this cannot be assured on the basis of

this project alone. Some criteria measured in this project did not improve to a great degree with increases in compliance minimal, leaving plenty of room for improvement. By the end of the project however, attitudes to falls prevention on the neurosurgical ward and across the facility had been 'transformed' from passive acceptance of falls, to active engagement in falls prevention and minimisation of injury.

Identification of time limitations within a nurse's daily role and often poor documentation practices were two of the main barriers underpinning the gaps between best practice recommendations and actual practice. Identification of these barriers facilitated understanding why for some of the audit criteria performance improved only minimally. This has highlighted the importance of future education initiatives targeted at clinicians including a focus on this aspect of care.

Although it is acknowledged that a focus on falls tends to increase the number of reported falls on a ward, potential long term benefits such as a reduction in the neurosurgical ward falls rates will be measured over a longer study period. Future audits are planned to ensure changes are sustained and improved with the aim that the neurosurgical ward and wider hospital not only prevents falls and harm from falls but can give a patient centred approach and instil confidence in our patients and their carers that we are doing all we can to prevent such events.

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Freezing of Gait: What to do when the Parkinson's Medications do not work

Melissa Drury

Abstract
Parkinson's Disease (PD) is the second most common neurological disorder affecting around 80,000 Australians. One of the most troublesome and incapacitating motor symptoms affecting people with PD is freezing of gait (FoG). FoG has been recognised to be a major cause of falls, hospitalisation, decreased quality of life and loss of independence. FoG is described as a transient start and turning hesitation, where the patient's feet appear to be glued to the ground. The relationship between FoG and PD medication remains complicated, as FoG sometimes does not respond to PD medication or in rare cases; the medication can actually cause FoG. It is important to implement other interventions such as auditory/visual cueing, physiotherapy as well walking aids with auditory/visual cueing devices. The role of the Parkinson's Disease Nurse Specialist (PDNS) also plays an important role in educating the patient with Parkinson's and their family as well as identifying the stage/s of FoG. Further research and interventions are required to provide a better quality of life for people with this debilitating motor disease.

Keywords: *Freezing of Gait, Parkinson disease*

Introduction

Parkinson's Disease (PD), is the second most common neurological condition affecting Australians, second only to Alzheimer's. Currently there are approximately 80,000 Australians who have been diagnosed with this slow, chronic and non-curable disease (Parkinson's Australia, 2011). PD is characterised by both motor and non-motor symptoms resulting from the loss of dopaminergic neurons in the basal ganglia (Vandenbossche, Deroost, Soetens, Coomans, Spildooren, Vercruysse, Nieuwboer & Kerckhofs, 2013). One of the most troublesome and incapacitating motor symptoms affecting people with PD is freezing of gait (FoG). FoG has been recognised to be a major cause of falls, hospitalisation, decreased quality of life and loss of independence (Panisset, 2004).

FoG is defined as "brief, episodic absence or marked reduction of forward progression of the feet despite the intention to walk" (Nutt, Bloem, Giladi, Hallett & Horak, 2011). Patients with PD describe the sensation of FoG as if their feet are glued to the floor. The prevalence of FoG varies between reports: Contreras & Grandas (2012) found FoG in 7% in early stage disease which increased to 60% in more advanced patients with Parkinson's; Heremans, Nieuwboer & Vercruysse (2013) reported 26% in the earlier stages and 70% in the later stages of PD.

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FoG most commonly occurs on initiation of walking, walking in a straight line, passing through narrow passages, turning and on reaching the destination (Nutt et al, 2011). FoG can last from a few milliseconds to over 30 seconds. Certain circumstances such as narrow doorways, dual tasking, mental distractions and crowed places have been identified to exacerbate FoG (Nutt et al, 2011). However, Nutt et al (2011) also reported that emotional excitement, auditory cueing at the proper pace, targets for stepping and climbing stairs can ameliorate FoG.

Levodopa therapy has been identified as the gold standard of treatment for people with Parkinson's (PWP); however the relationship between FoG and PD medication remains complicated. FoG typically occurs when the PWP is in the pre-medicated, "OFF" state. FoG is abolished when the person reaches their optimal medicated, "ON" state. However, PD medications do not always alleviate FoG and the freezing continues into the "ON" state. There is also a rarer phenotype in which the PD medications induce the "ON" state FoG, even when FoG is absent in the "OFF" state (Espay, Fasano, van Nuenen, Payne, Snijders & Bloem, 2012). These rare

FoG conditions can create a troublesome and complicated symptom that is refractory to medical treatment.

Pathophysiology

The pathophysiology of FoG is largely unknown and seems to differ from that of the cardinal features of Parkinsonism (tremor, bradykinesia and rigidity). This is not surprising as FoG occurs in other conditions such as Normal Pressure Hydrocephalus (NPH), Dementia with Lewy Body (DLB), Progressive Supranuclear Palsy (PSP) and Vascular Parkinsonism (VP) (Nutt et al., 2011). After reviewing the literature from 1966 to 2011, Nutt et al (2011) proposed five directions for developing a hypothesis on the pathophysiology of FoG. More recent publications (reviewed by Heremans et al (2013) support these five theories:

1. *Abnormal Gait Pattern Generation*

The person with FoG also exhibits a progressive shortening of step-length prior to the freezing episode. This form of spatial-temporal incoordination would suggest a disrupted pattern generation.

2. *A problem with Central drive and Automaticity of Movement*

In FoG, there is an impairment of the automated tasks such as posture and gait. With this impairment the normally automated tasks demand increased attention. This explains why freezing is exacerbated when attending to dual tasks and how a decrease in FoG is derived from external cues (which provide a drive to the patients' stepping patterns).

3. *Abnormal Coupling of Posture with Gait*

The upper body propels forward in anticipation to start walking but a miscommunication causes difficulty in the lower limbs initiating forward progression, such that the lean and the limb movements are not well coordinated.

4. *A Perceptual Malfunction*

Perceptual malfunctions causing difficulty in interpreting length, width and distance of the environment may lead to FoG. This is seen in worsening of FoG through doorways, other narrow constraints and by environmental obstacles.

5. *Frontal Executive Dysfunction*

There is increasing evidence that patients with FoG show greater dysfunction in executive control compared with patients without FoG. The severity of the patient's executive dysfunction correlates with freezing severity.

Demographic factors such as family history, ethnic background, age, disease onset and gender have not been associated with the likelihood of developing FoG (Panisset, 2004). However, it has been identified that longer disease duration, more advanced PD, greater disability from PD symptoms, prolonged treatment with PD medications and depression are associated with increased risk of developing FoG (Panisset, 2004).

Intervention

Since not all forms of FoG are responsive to PD medications, it is important to implement other therapeutic options to help with mobility. Physiotherapy and exercise assist in walking, falls prevention and strengthening and are identified as an important aspect of treatment of FoG (Nutt et al, 2011).

Auditory and visual cueing has been identified to be an important intervention for FoG (Delval, Moreau, Bleuse, Tard, Ryckewaert, Devos & Defebvre, 2013). Cueing acts to substitute the failed automation of walking to a conscious learnt activity. Visual cues that alleviate FoG include lines on the floor and other visual targets which stimulate the PWP to walk – including LASER projections onto the floor from a specially designed walking aid (stick or walker). Auditory cues include walking to a metronome beat or music, counting numbers, singing a rhythm out loud or simply reminding the patient to adopt a 'marching' (high-stepping) gait.

The PDNS plays an important role in the management of a PWP with FoG. A dose cycle (a trial of the clinical response to either levodopa or apomorphine) is a helpful diagnostic and investigative tool to correlate the persons FoG in relation to their medication cycle. It involves a comprehensive motor assessment of the patient's FoG in at least three medication states: the "OFF" state, "ON" state and the "Supra-ON" state where the person is given a double dose of their regular PD medication to distinguish which state/s FoG occurs (Espay et al, 2012). The PDNS also has an important role in educating the PWP and their carers on the nature of FoG, strategies to manage it and equipment available to combat this troublesome symptom.

With the advent of cheaper and more advanced electronic technology, there have been multiple products recently developed to assist in people with FoG. The U-Step Walker® is a four-wheeled walking frame which has both a device that makes rhythmical mu-

sic for auditory cueing as well as a laser line that shines on the ground and serves as a visual cue for the person to walk over. Other laser beam devices include the Agilitas® - a circular electronic device with a motion sensor that is worn on the person's belt and automatically activates a laser beam on the floor when the person is in a freezing episode.

In the future, mobile phone applications might be used to trace a PWP's freezing episodes during the day and provide on demand visual cues, for example through Google Glass® eyewear. The voice activated pair of glasses is linked to the Internet and can remind the patient when to take their PD medications. However, most PWP tend to be relatively elderly and may not have the financial ability or the technological wherewithal to afford or function complex electronic devices.

Conclusion

FoG remains a mysterious phenomenon that may ultimately be one of the most debilitating motor symptoms of PD. When PD medications do not alleviate FoG, it is important to identify and implement other therapeutic interventions such as education in using auditory/visual cues, implementation of walking devices and/or physiotherapy to ensure that PWP suffering from FoG can sustain a good quality of life and reduce the risk of unnecessary FoG-associated falls and hospital admissions. The PDNS plays an important role in identifying the nature of a PWP's FoG through dose cycle assessment and can provide important education to both the PWP and their carers. Further research is required into the pathophysiology of FoG, however, in the interim, such interventions can assist PWP in providing a better quality of life.

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