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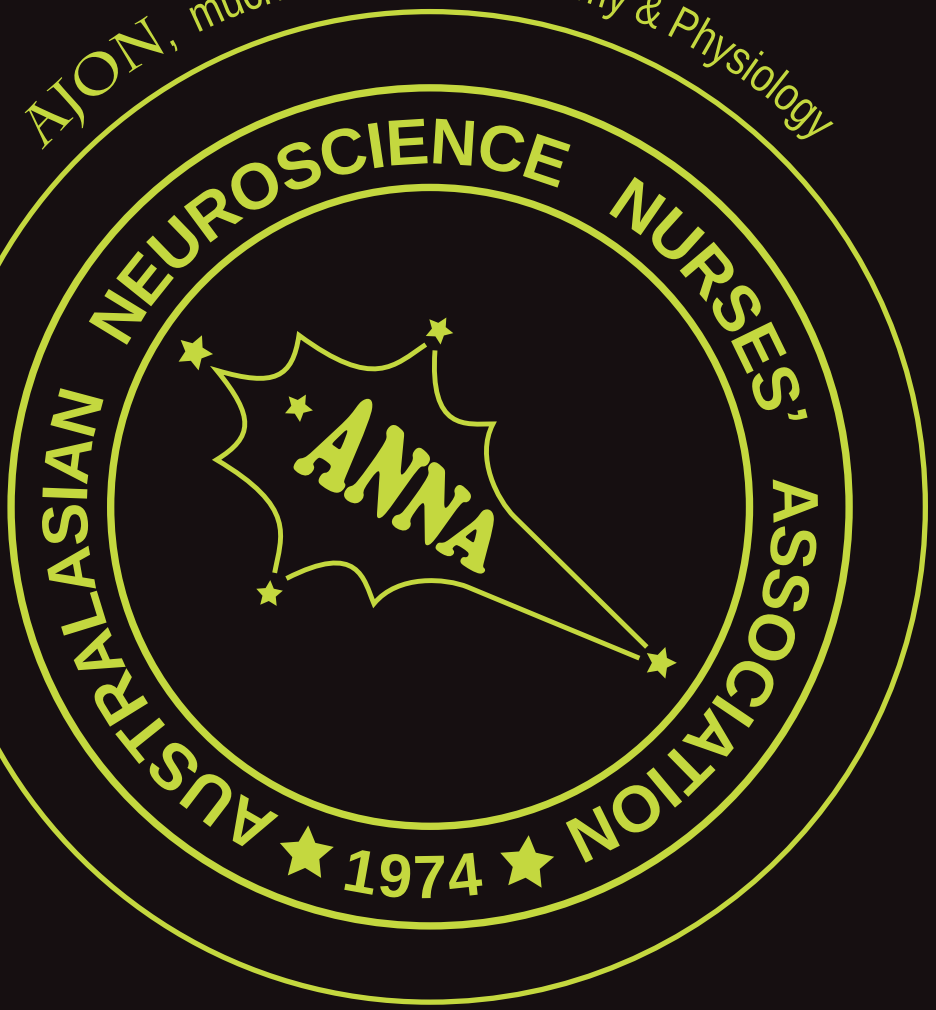

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In This Issue:

Editorial

Editor - Vicki Evans

Professionalism - what's it all about?

Professionalism generally refers to the standards and expectations set for employees in the workplace. These include expectations regarding appearance, behaviour and performance standards. Ethics and professionalism are highly sought characteristics in a workplace setting.

Admirable work characteristics include being honest, reliable, positive, keeping emotions under control, being responsible for your own actions, listening carefully to directions and avoiding negativity. Commitment is demonstrated by meeting deadlines, completing work efficiently and effectively and by being a team player. By feeling good about yourself, you will have more to contribute in a professional setting.

As Neuroscience Nurses, we are governed by the Standards as set out by the Nurses and Midwives Board, ANNA, WFNN and the Code of Conduct by our employers. We are bound by a Duty of Care which encourages professionalism and a consistent increasing knowledge base.

Networking and a desire for learning should be part and parcel of our daily working life. As a professional neuroscience nurse, we should lead by example and encourage others to network through membership of their professional association, to attend conferences, to present a paper/poster and/or write for the journal. Give it a go - you'll be pleasantly surprised!

Our Guest Editor needs no introduction - she is a leader in neuroscience circles. We all have a copy of her book (albeit different editions!) I am pleased to offer you the following comments from Dr Joanne Hickey.

Cheers,

Vicki

Bibliography: Current Factors Contributing to Professionalism in Nursing:-
Wynd, CA Journal of Professional Nursing 2003. Elsevier
Brooks, KL Journal of Professional Nursing 1992. Elsevier

Guest Editorial

Dr Joanne V Hickey

Being All That We Can Be for Patients

It is my honor and pleasure to share some thoughts about nursing with neuroscience nurses in the Australasian readership. A recent report from the Institute of Medicine (IOM) focused on nursing in the United States. However, it speaks to all nurses irrespective of where they live or practice in the world. What is the IOM? It is an independent, interdisciplinary, nonprofit, and non-governmental organization that provides unbiased and authoritative advice to decision makers and the public about the most pressing health and health care questions. The Future of Nursing: Leading Change and Advancing Health (2010) is historic in that it is the first time that the prestigious IOM has focused solely on the profession of nursing.

The four major recommendations in the report include: (1) nurses should practice to the full extent of their education and training; (2) nurses should achieve higher levels of education and training through an improved education system that promotes seamless academic progression; (3) nurses should be full partners, with physicians and other health care professionals, in redesigning health care; and (4) effective workforce planning and policy making require better data collection and information infrastructure.

Professional nurses have a sacred trust with the patients that they serve. Their educational preparation and experience should be fully utilized to serve their patients. A commitment to ongoing education through degree programs and other educational experiences needs to be seamless in order for nurses to provide the best comprehensive and evidence -based care. With the complexity of patient needs and care, collaborative interdisciplinary care models are needed, but nurses must be full partners with other health professionals in designing care and optimizing patient outcomes. Finally, workforce planning and health policy are needed based on reliable databases to provide access to nursing care for all people. These are universal truths for all profe

-ssional nurses throughout the world. How does this translate to neuroscience nursing? Consider the following. Neuroscience nurses:

- have a sacred trust with society and their patients.
- care for particularly vulnerable patients with acute and chronic neurological health problems that often affect the personhood and spirit of the patient, as well as basic life functions and activities of daily living.
- provide evidence-based care and compassionate care based on a wealth of knowledge, compassion, and advocacy for their patients and families.
- are the critical link for coordinating comprehensive care that is patient centered.
- make a significant difference in patient outcomes.

The message of the *Future of Nursing* speaks to all nurses underscoring how critical nurses are for all societies. It also challenges the nursing profession to be all that we can be, not for any self-serving reasons, but for the patients that we serve.

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Letters to the Editor

From **Ms Sharryn Byers. President, Australasian Neuroscience Nurses Association.**

Three separate and yet connected thoughts kept coming to mind over the weekend. A copy of a new text book arrived in the mail last week entitled 'Neuroscience Nursing, Evidence-based practice (review appears in this edition of the Journal), a request for information from a member regarding pre-operative hair shaving practices and differences in practice between surgeons post-operative wound management.

Would opening the pages of the book bring with it the answer? How do we know what dressings will stay on the head after cranial surgery? Can we insist the human race be bald so our jobs are easier (unlikely I'm sure you would agree)? Does any dressing product reduce the risk of wound site infection? Do we need to dress cranial wounds at all?

Turning hopefully to the index I searched for hair shaving and found an entry! Joyously I turned to the page specified to find several lines and a reference. Would it answer my questions? Logging onto the internet my search yielded the paper referred to in the textbook but unfortunately while it discussed shaving the head or not the questions regarding dressing type would remain unanswered. There are many questions in neuroscience nursing that remain essentially unanswered with practice determined by publications, personal experience, preferences of specific individuals and what supplies are available in the hospital itself. Not that I believe these methods result in the wrong answer but that unless we can replicate the substance of a practice we won't know if it was good management or chance that gave us the result.

How do we generate the answers to questions regarding practice? In any individual unit is there a standard approach to practice, is the result measurable, are there enough patients to give power to the answer, are there neuroscience nurses with the same question, and do we have the skills, knowledge and resources to develop a strategy to answer the questions? The answer is not always.
After landing on the moon Armstrong is quoted as saying 'one small step for (a) man, one giant leap for mankind'. What is less well known are the words spoken on the return journey by Aldrin "this has been far more than three men on a mission to the Moon; more, still, than the efforts of a government and industry team; more, even, than the efforts of one nation. We feel that this stands as a symbol of the insatiable curiosity of all mankind to explore the unknown ..." www.wikipedia.org/wiki/Apollo_11 accessed 7/02/2011.

One of the strong skill sets we have as neuroscience nurses is team work. It is time we gather our energy, enthusiasm and skill to look beyond the boundaries of our immediate workplace and look to the wider neuroscience team to start the journey of solving practice issues. Perhaps your 'one small step' is a published paper in this journal? Who knows where the journey may end!

~ Sharryn

Book Reviews

By **Ms Sharryn Byers.** CNC: Neurosurgery, Nepean Hospital. President, ANNA & **Ms Linda Carter**, RN Royal Hobart Hospital, Tasmania.

Neuroscience Nursing Evidence-Based Practice. Edited by Sue Woodward and Ann-Marie Mestecky. *Wiley-Blackwell 2011.*

The Publishers state that this book "...is a comprehensive, practical text that reflects both the richness and the diversity of contemporary neuroscience nursing. It aims to inform the practice of neuroscience nursing through the report of current research, best available evidence, policy and education".

We agree that it is a well written evidenced-based text, which covers a broad scope of neuroscience nursing practice. Despite being UK dominant, it gives great depth to neuroscience nursing world-wide and is a very useful text for both novice and expert, with excellent references to facilitate further independent research.

It covers a wide range of topics integrating anatomy, physiology, pharmacology, microbiology and pathophysiology, whilst also keeping within perspective of the social setting, with epidemiological and etiological data included where relevant.

The text is clearly divided into chapters with subheadings allowing for rapid location of information, this is complimented by the colour images and tables/charts, that provide relevant visual cues and prompts. The focus of the text on assessment, interpretation and management encourages a holistic approach to care. It is concise and succinct and holds a wealth of knowledge.

If you are interested in updating or starting your library then this text is an excellent addition.

The book normally retails at $74.95. However, John Wiley & Sons have given ANNA members a **20% discount.** To access the discount please go to **ww.wiley.com** and follow the instructions as outlined below -

- Find the book in Search (could use ISBN 9781405163569 or author Woodward)
- Add to Cart
- Go through the sales process and then enter the **Promo Code NSN11** when prompted.

Offer is valid through to June 30th 2011.

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 11 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

Rethinking Haemorrhagic Stroke and Bedrest: A Ten Year Project.

Elizabeth M. O'Brien

Abstract

Many nurses carry out projects and small audits, quality initiatives and research on a subject that they are passionate about that often do not reach the final stages of publication. Many things can get in the way of final completion and publication. This is an example of work that commenced in 1998 but is still relevant in today's practice.

Aims: The aim of this project was to review the evidence relating to bedrest after intracerebral haemorrhage and to gain a consensus based on a quality initiative and reassessing current practices that would benefit patient care.

Methods: A literature review and discussion with consultant neurologists to review current trends was undertaken. A trial protocol of 12 hours bed-rest and strict blood pressure management was developed. The protocol was utilised in the acute stroke unit for non-surgical haemorrhagic stroke patients.

Results: After 12 months, 20 patients with haemorrhagic stroke had been admitted to the acute stroke unit and had been suitable for the protocol trial. Of the patients treated with this protocol there were no adverse variances recorded after early mobilisation.

Discussion: As a consequence of the evaluation it was decided that the majority of patients with haemorrhagic stroke would benefit from a decreased time of enforced bed-rest. The standardisation of this protocol has assisted in the consistency and efficiency of care, focusing on best practice for haemorrhagic stroke.

In recent years the success of the AVERT (A Very Early Rehabilitation Trial for stroke) study and its focus on very early mobilisation has been an impetus to put forward this information so that further quality improvements and research can be planned.

Key Words: *Haemorrhagic Stroke, Intracerebral haemorrhage, bedrest*

Introduction

The issue of bedrest after haemorrhagic stroke has been explored in a literature review to assess the need for changing practice in an acute stroke unit at a tertiary referral hospital. Historically, bedrest has been the first line treatment for haemorrhagic stroke. Bedrest has varied from 48 hours to 72 hours and in some cases up to 5 days. Anecdotally, the rationale for this has been to allay the risk of further bleed in the acute stroke patient. There are many arguments for changing this practice in order to further benefit the acute stroke patient and improve their outcome. The standardisation of protocols will assist in the consistency and efficiency of care, focusing on best practice.

The purpose of this article is to describe the development and evaluation of a protocol for early mobilisation of patients with haemorrhagic stroke. The protocol was developed after review of the literature and in discussion with the consultant neurologists.

Questions or comments about this article should be directed to Elizabeth O'Brien, Stroke Clinical Nurse Consultant, Royal North Shore Hospital, Sydney at EObrien@nsccahs.health.nsw.gov.au

Literature Review

Allen (1999) describes the use of bedrest to treat many ailments and post operative recovery, in the 1940's studies showed no advantages for complete bedrest after surgery showing dangers such as deep vein thrombosis, bedsores, osteoporosis and pneumonia. Bedrest after myocardial infarction was enforced on the basis that it would decrease the workload on the heart thereby decreasing the risk of further myocardial infarction. In 1938 patients had bedrest for 2 months but there were more reported incidences of pulmonary

infarction, uraemia and pneumonia than cardiac complications. Hence bedrest was shortened from weeks to days (Allen, 1999).

The literature is scant in addressing the issue of bedrest after haemorrhagic stroke, however there is discussion related to duration of the initial bleed. The incidence of re-bleed after intracranial haemorrhage (ICH) does not appear to be reported as often in the literature as does the length of time that bleeding continues. It is noted that recurrence of intracerebral haemorrhage depends on the aetiology of the initial bleed, with those associated with amyloid angiopathy and arteriovenous malformations most likely to recur, whereas, haemorrhages due to hypertension rarely recur if their blood pressure is controlled (Caplan, 1992). Hypertensive ICH is widely believed to be monophasic with only brief active bleeding. Clinical deterioration after admission is usually attributed to brain oedema, hydrocephalus or systemic factors (Chen, Chen, Hsu & Hogan,1989).

Broderick, Connolly, Feldmann, Hanley, Kase, Kreiger, Mayberg, Morgenstern, Ogilvy, Vespa & Zuccarello (2007), investigated the duration of bleeding subsequent to ICH and found that bleeding does continue after the first hour following onset. CT and autopsy data showed that bleeding after initial ICH can continue up to five hours, though rarely longer than that. The duration of bleeding may depend on the underlying mechanism of haemorrhage. The neurological deterioration that is a feature in haemorrhagic stroke in the first 24 hours may be caused by growth of the haematoma, increased intracranial pressure and secondary ischaemic effects of the haematoma upon the surrounding brain parenchyma (Broderick, Brott, Tomsick, Barsan & Spiker, 1990).

The effect of activity on the risk of re-bleed has not been addressed as such but has been alluded to in the literature. Clinical deterioration occurring within 24 hours, and especially within the first 12 hours, may be due to continuous bleeding, whereas re-bleeding accounts for deterioration that is delayed for several days.(Chen et al. 1989) Whether physical effort early after hypertensive intracerebral haemorrhage is detrimental remains to be determined. Powers, (1993) discusses the issue of hypertension in stroke and its prevalence immediately following an intracerebral haemorrhage. Broderick et al (2007) Indicates that a systolic blood pressure greater than 160mmhg has been associated with growth of the haematoma.

In a randomised trial of hypotensive therapy versus bedrest in patients with unclipped ruptured aneurysms, there was no difference in the risk of early re-bleeding or early mortality with either of these treatments (Powers, 1993).The significance of this in relation to stroke from an intracerebral bleed, is the greater potential for re-bleed in untreated aneurysms compared with hypertensive ICH.

The consequences of immobility and bedrest are widely documented. The complications that can ensue after stroke are many, affecting recovery and rehabilitation. Early mobilisation is important and can be as simple as institution of a regular sitting program to prevent orthostatic hypotension and weakness (Alter, Roth, Steins & Young 1995). Stroke care, both in the acute and rehabilitation phases, aims to improve the outcome of the stroke patient. Stroke rehabilitation programs, particularly during the initial days and weeks after stroke, aim to prevent contractures and deconditioning. Muscle strength can deteriorate as rapidly as three percent per day, but rebuilds only half as fast. Deconditioning begins within the first 24 - 48 hours (Yamaguchi Minimatsu & Hasegawa, 1997; Topp, Ditmyer, King, Doherty & Hornyak, 2002).

Resultant complications, e.g. pneumonia, deep venous thrombosis (DVT), pulmonary emboli (PE), decubitis ulcer, cholecystitis and urinary tract infections are a direct consequence of increased catabolism, venous stasis, decreased vital capacity, slowing of the gastro-intestinal tract and urinary stasis (Yamaguchi et al, 1997). However, the earlier the patient is allowed to mobilise, when haemodynamically stable, the earlier functional recovery may be attained (Yamaguchi et al, 1997; Gibbon, 1996; Ianowski, 1996; Leahy, 1991).

Mayer (2003) comments that effective therapies for ICH are limited, treatment is primarily supportive and outcomes remain poor. It is also noted that hypertension and advancing age are the most common risk factors for ICH. Once again, the duration of bleeding was a focus, historically reporting that ICH bleeding is completed in minutes of onset. However more recent studies have indicated that early haematoma growth occurs within 3 hours of onset in up to 40% of cases and correlates with early neurological deterioration (Mayer, 2003).

Bedrest Protocol Development and Evaluation

As the literature is scarce in relation to haemorrhagic stroke and bedrest immediately afterwards, it was decided to investigate non-surgical

haemorrhagic stroke patients and their progress with bedrest limited to 12 hours only. The length of bedrest would depend on the patient's haemodynamic status and presentation. Assessment of patients with haemorrhagic stroke would be the responsibility of the senior Registered Nurse, Clinical Nurse Consultant (CNC) or Resident Medical Officer (RMO)

Prospective documentation review would be performed looking at condition, adverse occurrences and outcome. The variances are documented on the back of the protocol sheet and in the patient case notes. The results of this review would be evaluated to assess any increases in complications, as a result of decreased bedrest following haemorrhagic stroke. Based on the findings a policy review would be made.

The protocol is given in detail in Box 1 (right). The evaluation of the protocol was carried out according to the following recommendations:

- All haemorrhagic stroke patients admitted to the Stroke Unit would be on bedrest for only 12 hours. This will depend on stable haemodynamic status. Blood pressure control is essential.
- Assessment of these stroke patients will be the responsibility of the senior neurologist/ RMO, CNC or senior registered nurse.
- Documentation would focus on time of mobility, adverse occurrences and outcomes.
- At 6 months the records of haemorrhagic stroke patients will be reviewed to assess any increases in complications as a result of decreased bedrest following haemorrhagic stroke.
- A decision based on these findings may lead to a policy review

Haemorrhagic Stroke/Bedrest: PROTOCOL

This protocol may vary depending on the functional status of individual patients. The variances will be documented for each patient. Please document when patient was allowed to mobilise, blood pressure and any adverse complications as a result, eg: hypertensive episode, change in level of consciousness etc.

Haemorrhagic Stroke & Bedrest Protocol

Day 0 - Onset of symptoms; Diagnosis of haemorrhagic stroke confirmed on CT scan.

Strict bedrest for the first 12 hours post haemorrhage.
Maintain blood pressure limits as set by consultant neurologist/ registrar.

Day 1 - Review of patient by team, neurologist/ registrar, CNC/senior RN.
If blood pressure stable over period of bedrest and patient stable, bedrest may be ceased.

Sponge in bed.
Able to sit out in chair beside bed for up to 2 hours.
Physio assessment of chest and limbs.

Day 2 - Continued review of patient, blood pressure within limits.

Assisted shower on commode
Able to sit out of bed up to 4 hours.
Mobilise around bed.

Day 3 - Continued review of patient, blood pressure within limits.

Walk to shower with assistance if able.
Assist shower.
Sit out of bed as tolerated.
Commence physiotherapy program as per physiotherapist.

Day 4 - Continued review of patient, blood pressure within limits.

Supervised walk to shower (if able).
Supervised shower.
Continue rehabilitation program.

Day 5 - Continued review of patient, blood pressure within limits.

Independent mobilising to shower (if able)
Independent shower (if able)
Continue rehabilitation program.

Above: **Box 1 - Haemorrhagic Stroke & Bedrest Protocol**

Results

In a twelve-month period, August 1998 – August 1999, the Acute Clinical Stroke Unit cared for 20 patients with haemorrhagic strokes who were suitable for the Bedrest Protocol Trial. There were a very small number of haemorrhagic stroke patients who were not suitable because their haemorrhages were too severe and limited the available treatment modalities. Examples of this included when surgical intervention was not an option, the bleed was inaccessible; and prognosis was poor due to combined factors of size, site, clinical presentation and neurological status (Broderick , et al 2007; Manno, Atkinson, Fulgham & Wijdicks, 2005).

Initially a review of the Protocol Trial was to take place after a 6 month time span. At 6 months a total of 9 patients had been admitted with a

haemorrhagic stroke and therefore, the Bedrest Protocol. It was felt that this was not a significant number to use as a basis for a protocol review. A decision was made to continue the trial until a total of 20 patients was reached.

The types of haemorrhagic strokes can be classified to lobar, deep and brainstem. There were a total of 6 lobar haemorrhages, 12 deep haemorrhages and 2 in the brainstem. Of the lobar haemorrhages, 2 were frontal, 2 parietal, 1 temporal and 1 cerebellar. Of the deep haemorrhages, 3 were in the putamen, 3were basal ganglia, 4 were thalamic, 1 internal capsule and 1 external capsule. There was infiltration into the ventricles from 1 of the thalamic haemorrhages and 1 of the brainstem haemorrhages.

Out of the 20 haemorrhagic strokes, 11 had a documented history of hypertension. Throughout their admission the blood pressure of 6 of these patients' was well controlled after their initial hypertension on admission. The remaining 5 were administered statum antihypertensives in line with set parameters and commenced on regular antihypertensives if required.

Evaluation of the patient's readiness to mobilise after the initial 12 hours bedrest was undertaken. If their condition was assessed as suitable for mobilisation the nursing staff followed the regimen as indicated. Certain factors were taken into consideration. Blood pressure was assessed and treated if required. Level of consciousness was assessed, level of alertness and sitting balance was determined. If these criteria were met, and it was deemed safe for the patient to mobilise the protocol could be followed.

Of the 20 patients with haemorrhagic stroke treated with only 12 hours bedrest, no variances were recorded after early mobilisation. The variances looked for were signs of increased intracranial pressure, decreased level of consciousness, declining motor function or cognitive deficits, which could have been a result of bleeding.

Summary and Recommendations

As a consequence of the evaluation, it was decided that the majority of patients with haemorrhagic stroke would benefit from a decreased time of enforced bedrest, i.e. the incidence of DVT's, PE's and muscle wasting will be decreased. Careful monitoring and treatment of hypertension would decrease the risks of rebleed or continued bleeding and therefore assist in early mobilisation and rehabilitation of the acute stroke patient.

Recommendations from the evaluation were:

- To formalise the policy for bedrest following haemorrhagic stroke as a result of this Protocol Trial.
- All patients with haemorrhagic stroke will be on total bedrest for 12 hours post diagnosis of the haemorrhage.
- Blood pressure parameters will be set by the neurologist and maintained using antihypertensives as prescribed.
- Before mobilisation the patient will be reviewed and their blood pressure, level of consciousness and sitting balance will be assessed.
- Daily mobilisation will be followed as per protocol.

In April 2000, the Neurology Department of Royal North Shore Hospital approved the protocol change for haemorrhagic stroke and bedrest. It was hoped that from this we can decrease secondary complications and continue to improve our patients' functional outcome.

In 2003 the protocol was re-evaluated to monitor its success. The average yearly ICH rate admitted to this Stroke Unit was 20 - 30 cases. In January to July of 2003, 23 patients with intracerebral haemorrhage had been through the Unit. There was a documented history of hypertension in 15 cases, 6 had persistent hypertension during admission, 4 had a documented increase in ICH/ rebleed (1x10 days; 1x8 days; 1x7days; 1x1day), 2 underwent surgery and insertion of an external ventricular drain (EVD), low serum sodium (Na+) was documented in 2 cases, and 2 had DVTs.

From these results the incidences of complications post ICH do not appear to be directly related to the timing of bedrest. All hypertension was treated appropriately with medical therapy. Overall the patients showed no significant variances in relation to the duration of bedrest.

Conclusion

The Stroke Unit has continued this Protocol adherence for haemorrhagic stroke with an aim for a wider distribution to all ICH in other Area Stroke Units. Patients are continually monitored for adverse events and there have been no documented issues with the Haemorrhagic Stroke and Bedrest Protocol.

Since the commencement of this project, the AVERT (A Very Early Rehabilitation Trial for Stroke) study has continued to promote the benefits for early mobilisation after stroke finding that it

is safe and feasible (Bernhardt, Dewey, Thrift, Collier & Donnan, 2008). Furthermore, Cumming, Collier, Thrift & Bernhardt, 2008, noted a reduction in depressive symptoms at 7 days after very early mobilisation. The combination of physical and psychological benefits with early mobilisation and decreasing bedrest for stroke patients can only be a good thing.

The information gained from this project focusing on bedrest after haemorrhagic stroke and the ongoing AVERT study warrant more research on this topic to increase the evidence base for this group of neurological patients.

References

Allen, C., Glasziou, P. and Del Mar, C. “Bedrest: a potentially harmful treatment needing more careful evaluation.” *The Lancet* 1999: 354 1229-1233

Alter, M., Roth, E., Steins, S. and Young, M. "Stroke rehab; the critical early period", *Patient Care* 1995; October: 14-22.

Bernhardt, J., Dewey, H., Thrift, A., Collier, J. and Donnan, G. (2008) A Very Early Rehabilitation Trial for Stroke (AVERT) Phase II safety and feasibility. Stroke (39) 390-396

Broderick, J., Brott, T., Tomsick, T., Barsan, W. and Spilker, J. "Ultra - early evaluation of intracerebral haemorrhage". *Journal of Neurosurgery* 1990; 72: 195-199.

Broderick, J., Connolly, S., Feldmann, E., Hanley, D., Kase, C., Kreiger, D., Mayberg, M., Morgenstern, M., Ogilvy, C., Vespa, P. and Zuccarello, M. (2007) Guidelines for the Management of Spontaneous Intracerebral Haemorrhage in Adults. (38) 2001-2023.

Caplan, L., "Intracerebral haemorrhage", *The Lancet* 1992; 339: 656 - 658.

Chen, S,T., Chen, S,D., Hsu, C. and Hogan, E. "Progression of hypertensive intracerebral haemorrhage". *Neurology* 1989; 39:1509 - 1513.

Cumming, T., Collier, J. Thrift, A., and Bernhardt, J. (2008) The Effect of Early Mobilisation after Stroke on Psychological Well-being. Journal of Rehabilitation Medicine. 40, 609-614.

Gibbon, B. "Stroke care and rehabilitation", *Nursing Standard* 1996; 11(12): 51-56.

Ianowski, M. "A roadmap for stroke recovery", *RN* 1996; 59(3): 26-31.

Leahy, N. "Complications in the acute phase of stroke, nursing’s pivotal role". *Nursing Clinics of North America* 1991; 26, (4): 971-983.

Mayer, S. “Ultra- Early Hemostatic Therapy for Intracerebral Hemorrhage” *Stroke.* 2003: 34 (1) 224 - 229.

Manno, E., Atkinson, J., Fulgham, J. and Wijdicks, E. (2005) Emerging Medical and Surgical management Strategies in the evaluation and treatment of Intracerebral haemorrhage. *Mayo Clinic Proceedings* 80 (3) 420-433.

Powers, W. "Acute hypertension after stroke: the scientific basis for treatment decisions". *Neurology* 1993; 43: 461 - 467.

Topp, R., Ditmyer, M., King, K, Doherty, K. and Hornyak, J. “The Effect of Bedrest and Potential of Prehabilitation on Patients in the Intensive Care Unit.” *Advanced Practice in Acute and Critical Care* 2002. 13 (2) 263 - 278

Yamaguchi, T,. Minematsu, K. and Hasegawa, Y. "General care in acute stroke", *Cerebrovascular Diseases* 1997; (suppl 7):12 - 17.

Tramadol and Seizures: An Overview

Linda J. Nichols

Abstract

This review aims to expose the myths and misinterpretations of data concerning the risks of Tramadol Hydrochloride (Tramadol) and seizures within the neurosurgical environment. Within the neurosurgical environment there are differing views on the safety of Tramadol and the risk of seizures. Since Tramadol's release in Australia its unique biochemistry, pharmacodynamics and pharmacokinetics have resulted in it being one of the most widely prescribed analgesics and it continues to play an important role for neurosurgical patients. The non-controlled status and varied available routes of Tramadol administration also contribute to it being one of the most valued and widely prescribed analgesics. However the potential for seizures should not be underestimated, even when prescribed within the therapeutic range. Tramadol should be used cautiously and the benefits of treatment should outweigh the potential of seizures in patients who are at risk or taking other medications that lower the threshold for seizures. The phenomenon of seizures associated with Tramadol is likely to increase with increasing rates of analgesic and antidepressant use in the general population. Here lies two areas of importance for neuroscience nurses; managing patients admitted following seizures associated with Tramadol, and Tramadol administration to at risk patient's. It is crucial that nurses have a comprehensive knowledge of the seizure risk of Tramadol.

Key Words: *Tramadol, seizures, convulsions, idiopathic, serotonin, neurosurgical*.

Introduction

Within the neurosurgical environment there are differing views on the safety of Tramadol and the risk of seizures. It is crucial that neuroscience nurses have a comprehensive knowledge of the seizure risk of Tramadol. This review aims to expose the myths and misinterpretations of data, highlighting the increasing acceptance of Tramadol as a beneficial analgesic option within the neurosurgical environment. Since Tramadol's release in Australia in 1998, its unique biochemistry, pharmacodynamics and pharmacokinetics has resulted in it being one of the most widely prescribed analgesics in Australia. Tramadol is a centrally acting synthetic agent, with a dual mode of action. The first mode of action involves the inhibition of reuptake of serotonin (5-HT) and/or noradrenaline. The second mode of action involves a weak affinity for opiod (mu) receptors (Kaye & Theaker 2001).

Since Tramadol's release it has been recommended that patients with pre-existing epilepsy and those taking Tricyclic antidepressants, or other medications that reduce the seizure threshold such as the selective serotonin reuptake inhibitors (SSRIs), avoid Tramadol or use with caution. It is also recommended that the use of Tramadol is limited in patients with head injuries, due to increased levels of catecholamines that lower the seizure threshold (Tramadol Product Information 2002). Despite these recommendations Tramadol is frequently prescribed for neurosurgical patients including those undergoing craniotomy surgery. Seizures associated with Tramadol can be related to several distinct areas: idiopathic, related to serotonin syndrome; known risks; other contraindicated medications and over dosage.

Questions or comments about this article should be directed to Linda Nichols, RN, Neurosurgical Unit, Hobart Tasmania at linda.carter@dhhs.gov.tas.au

Literature Review

Despite the identified risks associated with Tramadol, a blanket contraindication is not constituted in all neurosurgical situations and Tramadol warrants consideration as an analgesic option for selective patients. This review will focus on adverse events associated with Tramadol, particularly seizure activity, and also briefly review the efficacy of Tramadol use within the neurosurgical set-

ting. A literature search strategy was undertaken utilising CINAHL, MEDLINE and PubMed in July 2010 using the keywords Tramadol, seizures, convulsions, idiopathic, serotonin and neurosurgical. Articles were limited to English papers published from 1995 to present. Adverse event reporting data was also reviewed from the Food and Drug Administration (FDA, USA), Adverse Drug Reaction Advisory Committee (ADRAC, Australia) and the Committee on Safety of Medicines (CSM, United Kingdom). A total of 48 studies were reviewed with the following 30 studies included. Animal experiments and studies that included results from seminal studies were excluded, as they were of little evidential support. The most significant period of seminal research occurred prior or just following the release of Tramadol in the USA and Australia. The studies included for this review provide an international perspective and range from large cohort randomised studies to single case studies. A specific focus on the use of Tramadol in the neurosurgical setting was fundamental as Tramadol continues to be frequently prescribed across a number of neurosurgical settings.

A number of studies support the efficacy of Tramadol in providing pain relief. McQuay & Edwards (2003) present a meta-analysis of 1400 adult patients, with data collected from seven randomised, double blind and placebo controlled trials which supported the use of Tramadol in combination with Paracetamol for the control of post-operative pain. In particular the study noted that the combination of Tramadol and Paracetamol may benefit patients who often cannot tolerate non-steroidal anti-inflammatory drugs. Fleischmann, Caldwell, Roth, Tesser, Olsen & Kamin (2001) reported results from a short-term placebo controlled clinical trial of 129 patients which suggested that Tramadol is effective therapy for osteoarthritic joint pain. Vergnion, Degesves & Magotteaux (2001) conducted a randomised, parallel-group, double-blinded study of 101 patients and compared the efficacy of intravenous Tramadol verses Morphine for the management of pain in trauma patients in the pre-hospital situation. The study utilised a four point verbal rating of pain intensity in conjunction with sedation and psychological data. Tramadol was found to be as rapid and effective as Morphine with minimal differences in side effects, mainly nausea and vomiting.

Tramadol is attributed as being useful for both long and short-term pain; it is readily prescribed, and is classed as a Poison Schedule 4 drug and not a Scheduled 8 drug. Bamigbade & Langford (1998) purport that Tramadol is significantly inferior to Morphine however it is frequently prescribed due to the lack of clinically significant adverse events such as respiratory depression and its long-term reduction in efficacy. However the authors highlight that Tramadol should be utilised with caution in patients with head injuries, as seizure thresholds are lowered with the increased levels of catecholamines associated with traumatic brain injury. Hence Tramadol is often disregarded as a possible pain relief option for neurosurgical patients due to its association with seizure activity. However there have been a number of studies that have trialled and supported Tramadol in the neurosurgical setting.

Jeffery, Charlton, Mellor, Moss & Vucevic (1999) compared Codeine Phosphate and Tramadol in a prospective, 75 patient, double blind study of postoperative analgesia after elective intracranial surgery. The study concluded that codeine provided better postoperative analgesia than Tramadol after craniotomy and suggested that Tramadol should be avoided following craniotomy surgery due to side effects of increased sedation, nausea and vomiting. Whilst the use of Tramadol for post craniotomy surgery has been limited by concerns about potential adverse events there is evidence to support the use of Tramadol following spinal surgery. Schnitzer, Gray, Paster &Kamin (2000) evaluated the efficacy and safety of Tramadol for the treatment of chronic low back pain utilising a three-phase trial including a four-week placebo controlled randomised trail of 380 patients, including 127 that were treated with Tramadol. The study concluded that Tramadol was well tolerated and identified it as an effective treatment for chronic back pain. More recently Emir, Serin, Erbay, Sungurtekin & Tomatir (2010) concluded that Tramadol can be utilised safely and effectively for the postoperative period after spinal vertebral surgery. The study, although only assessing 75 patients, utilised solid and varied data collected from nausea, pain and sedation scales in conjunction with vital signs. Klimek, Ubben, Ammann, Borner, Klein & Verbrugge (2006) conducted an observational study consisting of a structured questionnaire that compared perioperative pain quality and intensity in patients undergoing elective neurosurgical procedures. The study assessed 649 patients over a one-year period to compare different types of neurosurgical procedures and related pain, supporting the use of Tramadol for post craniotomy surgery.

This conclusion is in contrast to Roberts' (2005) evaluation of post-craniotomy analgesia survey that identified that Codeine Phosphate continues to be the mainstay of post-craniotomy analgesia. It is evident that the use and research of Tramadol within a neurosurgical setting is restricted by people's beliefs and interestingly the

issue of seizures, or risk of seizures associated with Tramadol use, have not been of primary concern during these studies. The relationship between Tramadol and seizure activity is well researched and published however, taken as a whole, the risk of Tramadol and idiopathic seizures is limited. Further large randomised trials within neurosurgical settings are required to gather empirical data. The association of Tramadol with seizure activity remains contentious and a topic of significant importance for neurosurgical nurses due to the susceptibility of the patient population, and the common prescription of Tramadol.

Although Tramadol has been available in Germany since the 1970's its release in the United Kingdom, USA and Australia was not until the 1980's and 1990's. In both pre and post-clinical studies Tramadol has been associated with a low risk for idiopathic seizure activity. Following the release of Tramadol in the United Kingdom, Budd & Langford (1999) editorial utilise data reported to the committee on the Safety of Medicines 1995. They identify that in the first two years of Tramadol availability in the UK there were 27 reports of possible Tramadol induced seizures, a rate of 1 in 7000. The authors conclude that there is no idiopathic risk of seizures associated with Tramadol as the majority of patients reviewed had a history of epilepsy and several others had received large intravenous (IV) doses of Tramadol. Supporting this is evidenced based reviews such as LeRoux & Coetzee (2000) who purport that the side effects associated with Tramadol appear to be dose as well as route-dependent. Tamam, Aslan & Tamam (2007) utilise a case report of recurrent seizures and agitation following Tramadol administration identifying the route of administration as a fundamental factor to seizure potential. Mehrpour (2005) reviewed two cases of seizures following doses of Tramadol of intravenous administration. All three studies support that seizure potency is increased by intramuscular or intravenous administration.

Seizure activity is commonly attributed to Tramadol, however reported seizures can rarely be confirmed as idiopathic. Jick, Derby, Vasilakis & Fife (1998) conducted a post-marketing surveillance nested case control study to identify the risk of idiopathic incident seizures. The study data was derived from the General Practice Research Database based in the United Kingdom for 1994-1996. The authors compared the risks of idiopathic incident seizures over a 90-day period. A total of 10916 cases were analysed and only 17 cases of idiopathic seizures were identified. Of the 17 cases 11 were identified as definite and 6 were identified as possible. All of the subjects had taken other medications within 90 days of the Tramadol dose, including eight having being exposed to opiates, and three to other analgesics. The authors concluded that there is no risk of idiopathic seizures associated with the use of Tramadol alone. However is this sufficient evidence for this conclusion when the authors are unable to confirm the cause of some of the seizures reported?

Fricke, Karim, Jordan & Rosenthal (2002) support the safety and efficacy of Tramadol. Their single-centre, double-blind, parallel-group, placebo, active-controlled study of 200 adults compared the efficacy and safety of Tramadol/Paracetamol tablets, Hydrocodone Bitartrate/Paracetamol tablets and placebo in the treatment of postoperative dental pain. The authors concluded that a combination of Tramadol/Paracetamol was able to provided comparable analgesia with better tolerability than the opiate medications. The 2005 literature review published by Close (2005) highlighted that despite reports of idiopathic seizures to the FDA and ADRAC, there appeared no increased risk of idiopathic seizures associated with Tramadol. This review had focused on emergency medicine and concluded that the risk of occurrence was less than 1%. Gardner, Blough, Drinkard, Shatin, Anderson, Graham & Alderfer (2000) utilised a retrospective cohort and case-control study to investigate the occurrence of Tramadol-associated seizures. Of the 9218 patients studied, fewer than 1% (80) suffered any possible seizure following an initial Tramadol prescription. The authors supported that the risk of idiopathic seizures was low. Gasse, Derby, Vasilakis-Scaramozza & Jick (2000) utilised a nested case control design with two years of data from the General Practice Research Data base from 1996-1998. Comparison of the 11383 subjects during 90 day follow up of exposed and unexposed periods when Tramadol had been taken found that only 10 definite cases of idiopathic seizures were identified, and 11 possible cases. The authors support that the risk of seizures for patients taking Tramadol was not increased compared with other analgesics.

There are a number of recognised risk factors that increase the risk of Tramadol associated seizures. It is recommended that Tramadol be prescribed with caution with medications that lower seizure threshold; including selective serotonin reuptake inhibitors, tricyclic antidepressants, antipsychotic drugs, and opiods (Tramal product information 2002). A history of seizure activity, alcohol intake or withdrawal, head injury, CNS malignancies and metabolic disorders are associated with an increased risk for seizure activity and are also recognised as associated risks for sei-

zure activity and contraindicate Tramadol prescription. Prior to the release of Tramadol in Australia, American studies such as Kahn, Alderfer & Graham (1997) had highlighted the risks associated with administering Tramadol with antidepressants, and other medications that lower seizure threshold. Tramadol and the co-administration of antidepressants are highlighted in the literature as an increased seizure risk. A report issued in 2003 by the Adverse Drug Reactions Advisory Committee (ADRAC) reported a total of 354 interactions, of this 26 being seizures. By 2005 this had increased to 921 reports with a total of 66 associated with convulsions. Boyd (2005) highlighted that of the 66 cases there were suspected drug interactions in 39 cases, and in 13 cases there were other medications taken that had the potential to lower seizure thresholds. Boyd (2005) also highlighted that seizures are more likely to occur with the associated use of medications that lower seizure threshold, particularly selective serotonin reuptake inhibitors. In this case the co-administration of selective serotonin reuptake inhibitors with Tramadol can result not only seizures but also potential serotonin syndrome.

Serotonin syndrome can be a potentially life-threatening adverse drug reaction. Diagnosis generally requires symptoms including agitation, confusion, hypomania, shivering, tremor, fever, reduced co-ordination, diaphoresis, diarrhoea, hypre-reflexia or myoclonus. Takeshita & Litzinger (2009) utilised a case study to illustrate possible serotonin syndrome caused by overdose of Tramadol. The patient was not on any other medications, and other possible causes of the presentation were dismissed. This case was the first potential report of serotonin syndrome associated with Tramadol only. Serotonin syndrome is commonly associated with the co-administration of medications, and/or over dosage of medication or medications. Houlihan (2004) reported a case of serotonin syndrome resulting from the addition of Tramadol to a medication regimen of Venlafaxine and Mirtazapine. The author implied that increased concentrations of serotonin resulted in peripheral and central 5-HT receptor overstimulation, resulting in serotonin syndrome. Tashakori & Afshari's (2010) prospective observational case series described the electrophysiological, clinical findings, hemodynamic findings and the frequency of potential serotonin syndrome in Tramadol overdose. Of the 158 cases, 24 (15%) experienced seizures, with 8 patients treated for potential serotonin syndrome. The authors contend that management of Tramadol over dosage should focus on both μ-opioid agonism and potential mild serotonin syndrome.

There have been a number of international studies that have studied the risk of seizures following Tramadol overdose. Spiller, Gorman, Villalobos, Benson, Ruskosky, Stancavage & Anderson (1997) evaluated reports made to seven poisons centres between October 1995 and August 1996 identifying 87 cases of Tramadol overdose. Of these 87 cases only seven (8%) experienced seizures. However larger and more current literature suggests that the risk of seizures associated with Tramadol over dosage is much higher. Talaie, Panahandeh, Asadi & Abdollahi (2008) utilise a cross sectional study of 215 Tramadol user/abusers that were recruited from a Poisoning Centre in Tehran in a period of 5 months during 2007. Of the 169 patients included in the study 62 (36.7%) suffered seizures within 24 hours after Tramadol use. However this large number of identified seizures can be attributed to the fact that a number of the subjects had also taken other medications including Benzodiazepines. Further the mean Tramadol intake dose was 2322 mg, this being significantly higher than the majority of other studies. A continuation of the study published in 2009 by Talaie, Panahandeh, Fayazonouri, Asadi & Abdollahi (2009) professed that the incidence of seizure with Tramadol is not dose dependent. Shadnia, Soltaninejad, Heydari, Sasanian & Abdollahi (2008) reviewed poisoning cases that admitted to a Hospital Poison Centre over a period of one month in 2007. Medical staff reviewed 114 cases of intentional Tramadol intoxications and data was collected from a questionnaire completed by the treating doctors. In this study 40 (35%) patients were noted to have experienced seizures. The authors concluded that inter-individual difference in the metabolism of Tramadol could be a cause for the broad range of toxic dose reported in the literature, highlighting that the majority of seizures occurred following the use of high doses of Tramadol, but in one case it occurred from 300 mg of Tramadol. However Marquart, Alsop & Albertson (2005) retrospective review of 190 cases of Tramadol exposure reported to a multi-site poison control system over a two and a half year period identified that seizures occurred in only 13.7% of reports. Of the cases studied the authors report that acute ingestions represented 90.0% of the overdoses with chronic ingestions representing 7.9% and acute on chronic 2.1%.

Within the range of accepted therapeutic doses there are a small number of papers identifying idiopathic seizures associated with Tramadol alone. Many current studies utilise a small number of selective case studies and professional opinions in journals and letters to editors that suggest a higher risk for seizure activity to support their opinion. Labate, Newton, Vernon &

Berkoviv (2005) evaluated 197 potential seizure patients at a First Seizure Clinic Austin Health. The patients were identified over a one-year period. Of the 97 patients who had confirmed seizures, eight were associated with Tramadol. Two had received high doses, two were taking long term psychotropics, of the four others seizures had occurred between 2 and 365 days after taking Tramadol. None of the patients had a history of seizures, and there was no recurrence after ceasing Tramadol. The authors do not make any calculations of the risk, however purport that the frequency of Tramadol related seizures might be under reported. Petramfar & Haghighi (2010) conducted a cross-sectional study of 106 patients with Tramadol-induced seizures referred to Nemazee Hospital, Iran, from 2006 to 2008. They suggest that Tramadol may provoke seizures. The study implied that 80% of the patients had seizure after therapeutic doses. However the study was not a population-based study, and was limited in that a large percentage of the cases reviewed were associated with known Tramadol abuse. The majority of these studies that profess the risks of idiopathic seizures fail to recognise other trials such as that by Fricke et al. (2002). A balanced review of the literature does recognise a risk of seizures associated with Tramadol, but it does not support the association of idiopathic seizures to Tramadol.

Conclusion

Tramadol plays an important role in analgesia including for neurosurgical patients. However the potential for seizures should not be underestimated, even when prescribed within the therapeutic range. The literature is conflicting with mixed evidence, and it is important to acknowledge that a large percentage of the literature can be associated with employees of pharmaceutical companies. The potential for seizures is often overlooked and there is the misconception that Tramadol is not an opiod and therefore it has gained a better safety profile than opiods. The non-controlled status of Tramadol also contributes to it being one of the most widely prescribed analgesics. Tramadol should be used cautiously and the benefits should outweigh the potential for seizures in patients who are at risk of seizures or taking other medications that lower the threshold for seizures. The phenomenon of seizures associated with Tramadol is likely to increase with increasing rates of analgesic and antidepressant use in the general population. The literature generally supports that Tramadol rarely causes idiopathic seizures when prescribed alone. A number of case-controlled and large cohort studies provide some perspective on the risk of seizures in patients receiving Tramadol. However, most identified cases have been associated with cofactors including; concomitant use of medications, over dosage, a history of seizure activity or substance abuse. Here lies two areas of importance for neuroscience nurses. Firstly managing patients admitted following seizures associated with Tramadol. Secondly the administration of Tramadol to at risk patients where a comprehensive understanding of the seizure potential involved with the use of such a drug and the caution of its use within the Neurosurgical population setting should be well understood. Further, nurses who are aware of considering Tramadol as a possible cause of seizure may avoid inappropriate prescription of anti-epileptic medications.

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References

Adverse Drug Reactions Advisory Committee. (2003). Tramadol – four years' experience. *Australian Adverse Drug React Bulletin*, 22(2), 2.

Bamigbade, T.A., & Langford, R.M. (1998). The clinical use of tramadol hydrochloride. *Pain Reviews*, 5, 155-182.

Boyd, I.W. (2005). Tramadol and seizures. *Medical Journal of Australia,* 182(11), 42-43.

Budd, K., & Langford, R. (1999). Tramadol revisited. *British Journal of Anaesthesia*, 82(4), 493-495.

Close, B.R. (2005). Tramadol: does it have a role in emergency medicine. *Emergency Medicine Australasia*,17(1), 73-83.

Emir, E., Serin, S., Erbay, R.H., Sungurtekin, H., & Tomatir, E. (2010). Tramadol versus low dose tramadol-paracetamol for patient controlled analgesia during spinal vertebral surgery, *Kaohsiung Journal of Medical Sciences*, 26(6), 308-315.

Fleischmann, R.M., Caldwell, J.R., Roth, S.H., Tesser, J.R., Olson, W., & Kamin, M. (2001). Tramadol for the treatment of joint pain associated with osteoarthritis: a randomized, double-blind, placebo-controlled trial. *Current Therapeutic Research*, 62(2), 113-128.

Fricke, J.R., Karim, R., Jordan, D., & Rosenthal, N. (2002). A double-blind, single-dose comparison of the analgesic efficacy of tramadol/

acetaminophen combination tablets, hydrocodone/acetaminophen combination tablets, and placebo after oral surgery. *Clinical Therapeutics*, 24(6), 953-968.

Gardner, J.S., Blough, D., Drinkard, C.R., Shatin, D., Anderson, G., Graham, D., & Alderfer, R. (2000). Tramadol and seizures: A surveillance study in a managed care population. *Pharmacotherapy*, 20(12), 1423-1431.

Gasse, C., Derby, L., Vasilakis-Scaramozza, C., & Jick, H. (2000). Incidence of first-time idiopathic seizures in users of Tramadol. *Pharmacotherapy*, 20(6), 629-634.

Jeffrey H.M., Charlton P., Mellor D.J., Moss E., Vucevic, M. (1999). Analgesia after intracranial surgery: a double-blind, prospective comparison of codeine and Tramadol. *British Journal of Anaesthesia*, 83(2), 245-249.

Jick, H., Derby, L.E., Vasilakis, C., & Fife, D. (1998). The risk of seizures associated with Tramadol. *Pharmacotherapy*, 18(3), 607-611.

Houlihan, D. (2004). Serotonin syndrome resulting from co-administration of tramadol, venlafaxine, and mirtazapine. *Annals of Pharmacotherapy*, 38, 411-413.

Kahn, L.H., Alderfer, R.J., & Graham, D.J. (1997) Seizures Reported With Tramadol. *Journal of the American Medical Association*, 278(20), 1661.

Kaye, K., & Theaker, N. (2001). Tramadol a Position Statement of the NSW Therapeutic Assessment Group Inc. NSW Health Department, http://www.ciap.health.nsw.gov.au/nswtag/publications/posstats/Tramadol%202001a.pdf, viewed July 2010.

Klimek, M., Ubben, J.F., Ammann, J., Borner, U., Klein, J., & Verbrugge, S.J. (2006). Pain in neurosurgically treated patients: a prospective observational study. *Journal of Neurosurgery*, 104, 350-359.

Labate, A., Newton, M.R., Vernon, G.M., & Berkoviv, S.F. (2005) Tramadol and new-onset seizures. *Medical Journal of Australia*,182(1), 42-43.

le Roux, P.J., & Coetzee, J.F. (2000). Tramadol today,' *Current Opinion in Anaesthesiology*, 13 (4), 457-61.

McQuay, H., & Edwards, J. (2003). Meta-analysis of single dose oral tramadol plus acetaminophen in acute postoperative pain. *European Journal of Anaesthesiology*, 28, 19-22.

Marquardt, K.A., Alsop, J.A., & Albertson, T.E. (2005). Tramadol exposures reported to statewide poison control system. *The Annuals of Pharmacotherapy*, 39,1039–1044.

Mehrpour, M. (2005). Intravenous tramadol-induced seizure: two case reports. *Iranian Journal of Pharmacology & Therapeutics*, 4(2), 146-147.

Petramfar, P., & Haghighi, A.B. (2010). Tramadol induced seizure: report of 106 patients,' *Iranian Red Crescent Medical Journal*, 12(1), 49-51.

Roberts, G.C. (2005). Post-craniotomy analgesia: current practices in British neurosurgical centres – a survey of post-craniotomy analgesic practices. *European Journal of Anaesthesiology*, 22 (5), 328-332.

Schnitzer, T.J., Gray, W.L., Paster, R.Z., & Kamin, M. (2000). Efficacy of tramadol in treatment of chronic low back pain. *Journal of Rheumatology*, 27, 772-778.

Shadnia, S., Soltaninejad, K., Heydari, K., Sasanian, G., & Abdollahi, M. (2008). Tramadol intoxication: a review of 114 cases. *Human & Experimental Toxicology,* 27, 201–205.

Spiller, H., Gorman, S.E., Villalobos, D., Benson, B.E., Ruskosky, D.R., Stancavage, M.M., & Anderson, D.L. (1997). Prospective Multicenter Evaluation of Tramadol Exposure. *Clinical Toxicology*, 35(4), 361-364.

Takeshita, J., & Litzinger, M.H. (2009). Serotonin syndrome associated with Tramadol. *Primary Care Companion Journal of Clinical Psychiatry*, 11(5), 273.

Talaie, H., Panahandeh, R., Asadi, Z., & Abdollahi, M. (2008). Seizure associated with tramadol use/abuse. *Clinical Toxicology*, 46(7), 628.

Talaie, H., Panahandeh, R., Fayaznouri, M.R., Asadi, Z., & Abdollahi, M. (2009). Dose-independent occurrence of seizure with Tramadol. *Journal of Medical Toxicology,* 5(2), 63 -67.

Tamam, Y., Aslan, S., & Tamam, B. (2007). Parenteral tramadol-induced seizure in therapeutic doses: a case report. *The Anatolian Journal Of Clinical Investigations*, 1(4), 273-276.

Tashakori, A., & Afshari, R. (2010). Tramadol overdose as a cause of serotonin syndrome: a case series. *Clinical Toxicology*, 48(4), 337-341.

Tramadol product information. (2002). CSL Limited. Melbourne.

Vergnion, M., Degesves, S., & Magotteaux, V. (2001). Tramadol, an alternative to morphine for treating posttraumatic pain in the pre-hospital situation. *Anaesthesia and Analgesia*, 99(6), 1543-1546.

"Neuron" 2010 by USA artist, Roxy Paine, installed at the front of the Museum of Contemporary Art, Sydney. Picture SMH 2010.

Evaluation of a Prune Juice Ginger-ale Cocktail for the Management of Constipation following Spinal Surgery.

Maria Chiera-Lyle, Yvette Lashley, Petal Samuel

Abstract

Aim: To explore the efficacy of prune juice and ginger ale as an alternative to the suppository.
Background: For many patients post operative constipation is common with the potential for complications including bowel impaction or bowel obstruction. Most people admitted to a spinal cord unit eventually require bowel routines or a cocktail of prescriptive medication. A review of the electronic patient record (EPR) suggested that in caring for these patients it had been noted that many will decline from having a suppository when it was offered to them. Many patients have asked for alternatives to the suppository.
Method: Over a 3 month period, 16 spinal patients were recruited by convenient sampling. Patients were offered a cocktail consisting of 125ml prune juice mixed with 125ml cup of ginger ale as an alternative to the suppository.
Results: It was determined that the cocktail produced a favourable outcome in eight out of sixteen patients because they were reported as having a BM within one hour to several hours later with just one to two cocktails. While the other eight patients who did not respond to the cocktail had a previous history of taking bowel meds at home.
Conclusion: The utilization of this cocktail is a safe and cost effective approach for the management of mild constipation that can be both managed in the hospital and at home.

Key words: *prune juice, ginger ale, constipation*

Introduction

For many patients post operative constipation is common with the potential for complications including bowel impaction or bowel obstruction. Most people admitted to a spinal cord unit eventually require bowel routines or a cocktail of prescriptive medication. Our spinal unit in a major teaching hospital addressed this issue with a bowel regime that is routinely ordered for most post operative patients after neuro/ ortho spinal surgery.

The bowel routine consisted of sennekot, colace, milk of magnesia (MOM) and a conditional order for bisacodyl suppository. A bowel routine is very important for this patient population as it will help to minimise constipation, a common side effect of opioid use.

A review of the electronic patient record (EPR) suggested that in caring for these patients it had been noted that many decline a suppository when it was offered to them. Our patients' cultural background is varied and one could only infer that perhaps the insertion of a suppository is a private and intimate act; one that may cause embarrassment for the patient. Many patients asked for alternatives to the suppository.

As an alternative, practitioners on study unit offered patients a cocktail of prune juice and ginger ale to help with relieving the effects of constipation. In keeping with the mission of the University Health Network to enhance and maintain a Patient Centered Care philosophy it was important to offer patients a choice of bowel routine. By offering choices we were acknowledging and respecting the patients' right to make informed decisions about their care. By engaging in this process, collaboratively we were also enabling the patient to exercise their autonomy in choosing what was best for their health. In order to prevent patients from developing complications from unresolved constipation, the evaluation of this cocktail was conducted to determine its effectiveness, efficacy and safety.

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Clinical Observation/Literature Review

An alternative to relieve constipation was introduced to one of the researchers 22 years ago by her clinical teacher. Since that time the researcher utilized this cocktail as an alternative to relieving constipation in her clinical practice with

positive outcomes in managing patients with constipation. According to the literature, prune juice has been documented to have a mild laxative effect in adults (Piirainen, Peuhkuri, Backstrom, Korpela, and Salminen 2007) and that a 250ml serving of prune juice can also be an adjunct to a healthy diet (Stacewicz-Sapuntzakis, Bowen, Hussain, Damayanti-Wood,Farnsworth,2001). Ginger Ale contains ginger which has been documented as being useful to relieve ailments such as nausea and vomiting (Badreldin, Blunden, Musbah, Abderrahim, 2008). Ginger ale is a common remedy that has been marketed for consumers to use for the alleviation of some gastro intestinal symptoms.

In clinical observation, the utilization of prune juice and ginger ale has been successful as an alternative laxative. Inferences can be made that the cocktail facilitates bowel elimination by these actions; the ginger ale makes the prune juice more palatable to drink. Ginger, an active ingredient in ginger ale, contains oleoresins, which act as a laxative (Ginger (Zingiber Officinale Roscoe), 2011). When ginger ale is combined with prune juice it may have a catalyst-type effect and become more potent as a laxative.

Purpose

The purpose of this quality initiative was to explore the efficacy of prune juice and ginger ale as an alternative laxative for post operative spinal surgical patients within the study unit.

Expected Outcomes

It was hypothesised that most patients would have a regular bowel movement daily in response to this treatment.

Methods and Materials

Staff members aware of this project were the Nurse Manager, Advance Practice Nurses, Four Spinal Surgeons, and the Educator. A tracking tool was developed by the research team in order to determine baseline data about bowel function and cocktail efficacy in comparison to the standard bowel routine. For this project the Review Ethics Board (REB) was not consulted because this was intended as a QI initiative and not a research study.

A consent form was developed and given to the study participants prior to beginning this project. The consent was given to the participants once the purpose of the project was explained to them and questions were answered. The consent was administered by a data collector. Confidentiality of the participants was protected by identifying them with a tracking number. The data collected was shredded post study.

The design and sampling method for this quality initiative was by convenience sampling with a range of twelve to sixteen participants over a three month period. The rationale for the length of time was that it allowed for the researchers the opportunity to enrol participants who would be screened during the pre-operative period during the pre admission class. An independent data collector not affiliated with the project was recruited. The data collector had a nursing background of greater than 10 years of nursing experience.

Inclusion criteria were all elective spinal patients undergoing procedures including laminectomy, spinal decompression, fusion or spinal tumour resection between 18-85 years. Exclusion criteria included those patients with stroke, head injury, brain tumours, subdural haemorrhages, chronic pain and anyone who has had a history of gastrointestinal obstruction and disorders such as Crohn's disease or colitis.

The data collector was scheduled to come in once a week to select patients that were best suited for this QI initiative based on the inclusion criteria. The data collector discussed the goals/ purpose of this QI initiative with the patients who met the inclusion criteria and answered any questions. Patients were also told that if the prune juice and ginger ale mixture was not effective, other medicinal alternatives would be used such as the administration of the suppository or perhaps receive other laxatives. Consent was obtained from the patient if he/she agreed to participate. Patients were given an honorarium ($5.00 gift card from a coffee shop) as a thank you for their participation.

Once the consent was obtained, patients were told that they would receive the prune juice and ginger ale cocktail in the morning as a replacement for the suppository (usually scheduled at 0900am). Additionally they were told that they could receive more than one cocktail if they felt that the one cocktail was not sufficient. The ratio for this cocktail was 125ml prune juice: 125ml of ginger ale.

This project was evaluated through the utilization of a tracking tool as well staff focus groups to determine the feasibility of the study. During the follow up telephone call, the questions that patients were asked were related to the taste of the cocktail and the timing (i.e. did you like the taste? And how long did it take to work before having a regular BM?).

Limitations

A confounding variable that was not isolated was the current bowel routine. The ordered bowel routine made it difficult to determine if the prune juice ginger ale was the effect or the colace and senekot .Other variables such as nursing attitudes towards bowel routines, timing of administration of cocktail, measurement of baseline bowel routine and patients' perceptions of the cocktail (i.e. dislike the prune juice), limitations with ambulation, history of chronic constipation and over the counter use of laxatives were also variables that also may have also affected the results.

Results

Eighteen subjects were recruited over a period of three months. There were only eighteen participants as two had declined participation in this QI initiative. Some were unable to participate due to a language barrier. The final sum was sixteen participants as one patient did not wish to continue and for the other patient, no data was entered. It was determined that the cocktail produced a favourable outcome in eight out of sixteen patients because they were reported as having a BM within one hour to several hours later with just one to two cocktails. However, bias could have been introduced into this initiative because of the utilization of the colace and senekot which was never removed from the EPR record. The remaining eight patients who did not respond to the prune juice ginger ale cocktail had to receive other bowel medications such as the suppository.

Discussion

The first data collector that was recruited had to withdraw from the project due to staff shortages. Therefore, another data collector was recruited in late February to continue with the project. The fact that there were staffing issues resulted in the project falling short on the priority list for the staff nurses. Hence there was an inconsistency in recruiting patients for the study. Some patients that were suitable were missed and others not as appropriate. Pre admission classes were cancelled and therefore it became difficult to recruit patients during the pre-operative period. As a result a small sample size was recruited from the unit by the data collector.

Documentation of the project in the nursing care plan was also inconsistent in that it was not clear as to which patients were in the study. A visual prop such as a poster regarding the project was also posted in the report/handover room in order to provide a visual reminder. The Advanced Nurse Practitioners and nurse manager also supported staff with this QI initiative with daily reminders.

All patients included in the study were given the prune juice ginger ale cocktail. The cocktail had a fifty percent positive response. Those patients who used the cocktail had a bowel movement within a few hours. There are many possible reasons for this and it may not be related to the cocktail. These reasons may be due to decreased opioid use, early ambulation, dietary intake and or the resumption of normal bowel patterns. Fourteen of the patients who received the cocktails said the taste was "good" or 'liked the taste" and would consider using it at home. This may also be due to the fact that the ginger ale makes the prune juice more palatable to drink while the prune juice is still the dominant factor as a laxative. At the same time when ginger ale with a stronger ginger taste was used, it yielded more favourable results. The fact that the prune juice ginger ale cocktail was rated as having a good taste may be an indication to use this cocktail in other settings such as paediatrics where adherence to a bowel routine may pose challenges for the younger groups. However, all the patients recruited in the study also continued to receive the senekot and the colace. Therefore, these two medications were confounding variables and could have affected the validity and strength of the study.

Questions that were not asked but may have been beneficial to this study was; once at home, whether or not the patients received a prescription for a bowel routine and if they were using any other bowel routine other than what was prescribed in hospital (i.e. did patients have their own special bowel routine?).

The remaining fifty percent of the patients (eight subjects) did not have a BM with the cocktail and had to resort to using the prescribed bowel routine which included the suppository. However, it is interesting to note that those eight patients had a history of taking bowel meds at home including metamucil or some other form of laxative. It may be that many of these patients tried the existing bowel routine currently prescribed. These data emphasise the importance of health care profssionals obtaining a detailed bowel history including bowel habits at home and implementing a bowel routine unique and specific to the patients own needs fostering a patient centered care approach.

Implications for Practice

The utilisation of this cocktail has taught us that it is a safe and cost effective approach for the man-

Participant #	Bowel routine at home	Date of last BM	Date cocktail given	Number of cocktails given	Length of time from administering cocktail to BM	Number of BM's after cocktail given	Data collector

Above: Study Data Table 1

agement of mild constipation that can be both managed in the hospital and possibly at home. Spine nurse practitioners have reported that some patients whom they have seen at follow up appointments have continued to use the cocktail at home with good results.

Implications for Future Research

The project yielded a fifty percent success rate; the researchers have identified areas for future research that may enhance the outcome of a future study regarding this cocktail as an alternative for a bowel routine. The study should be repeated with a larger sample size and a true experimental and control group with better understanding of the variables. Elimination of the confounding variable would help to strengthen the validity of the project. To also maintain integrity and inter reliability, a consistent data collector would be recruited.

To assist staff, structured educational sessions such as lunch and learns would be helpful for any future research activities. Through these sessions, staff would have the opportunity to ask questions and seek clarification if the information was unclear.

Conclusions

In conducting this quality project the researchers were able to provide a snapshot of an alternative to the existing bowel routine presently in use on a neurosurgical spine unit. Although there were many variables that may have threatened the validity of the outcome, valuable lessons were learned that provided insight for further research opportunities in regards to the development of a better bowel routine while preserving client choices.

Acknowledgements

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References

Baldeldin, H.A., Blunden, G., Musbah, T.O., Abderrahim, N. (2008).Some phytochemical, pharmacological and toxicological properties of ginger: A review of recent research. *Food and Chemical Toxicology 46,* 409-420

Ginger (zingiber officinale roscoe) 2011. Natural Standard. The authority on integrative medicine. Retrieved from http:// natural standard.com/ databases/herbsupplements/ginger

Piiainen, L., Peuhkuri, K., Backstrom, K., Korpela, R., Salminen, S. (2007).Prune juice has a mild laxative effect in adults with certain gastrointestinal symptoms. *Nutrition Research 27* 511-513

Stacewicz- Sapuntzakis, M., Bowen, P.E., Hussain, E.A., Damayanti-Wood, B.I., & Farnsworth, N.R. (2001). Chemical composition and potential health effects of prunes: A functional food? *Critical Reviews in Food Science and Nutrition*, *41*(4), 251-286

Down-under Case Reviews of Paraneoplastic Limbic Encephalitis Associated with Ovarian Teratoma

Diane Lear, Violeta Sutherland.

Abstract

Paraneoplastic limbic encephalitis (PLE) is an unusual disorder that is characterised by the association of clinical limbic system abnormalities with neoplasia, usually malignancy.
The disease has rarely been reported in children and manifests mainly in the teenage years. Patients with limbic encephalitis usually present with rapidly progressive short term memory deficits, psychiatric symptoms and seizures. Diagnosis is based on the clinical manifestations, the exclusion of viral and systemic autoimmune disorders, cerebrospinal fluid inflammatory findings, EEG or MRI abnormalities in the temporal lobes and antineuronal bodies (Lawn, N. D., Westmoreland, B. F., Kiely, M. J., Lennon, V. A. & Vernino, S. 2003). However diagnosis is often delayed, especially when the tumour has not been recognised.

In a study of eight reported cases by Corsellis, J. A. N., Goldberg, G. J. & Norton, A. R. (1968), a link between limbic encephalitis and systemic cancer was established. However, in the 1980s and 1990s increased awareness of the clinical manifestations of the disorder and advances in neuro imaging led to the identification of patients with similar syndromes but without paraneoplastic antibodies or tumours, or with "atypical" tumours.

Once considered an extremely rare disorder, usually related to malignancy and refractory to treatment, limbic encephalitis is now regarded as a relatively frequent disorder, often unrelated to cancer and with clinical – immunologic variants that respond to treatment. (Dalmau, J. & Tuzun, E. 2007).

We describe two case studies from separate tertiary referral hospitals in Sydney, Australia both involving teenage girls diagnosed with paraneoplastic limbic encephalitis secondary to ovarian teratoma. These case studies present the patient journey highlighting the difficulties faced in diagnosing this challenging disorder, the varying treatment modalities received by both patients and the rehabilitation process to their eventual discharge from hospital.

The case studies highlight the challenges to the patients and their families, medical, nursing and allied health staff who work together to achieve the optimal outcomes for two young neuroscience patients.

Key words: *Encephalitis, Paraneoplastic Limbic Encephalitis, Ovarian Teratoma.*

Literature Discussion

The following databases were reviewed: Cumulative Index of Nursing and Allied Health Literature (CINAHL), Ovoid Medline and Exerta Medica cd-Psychiatry (EMBASE).

The syndrome of limbic encephalitis is characterised by a subacute onset of temporal lobe epilepsy, loss of short term memory, and cognitive and psychiatric symptoms. There is a lot of published literature on paraneoplastic limbic encephalitis many focusing on the impact of the disease on the patient's and carers' quality of life.

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Khadem, G. M., Heble, S., Kumar, R. & White, C. (2009), state that although this condition has been recently described in the published literature most patients improve with immunosuppressive treatment. Kerling, F., Blumcke, I. & Stefan, H. (2009), writes that when limbic encephalitis is diagnosed, extensive tumour searching is mandatory to detect and treat the paraneoplasm of limbic encephalitis. In a case study published by Henry et al., 2009, the authors describe the full recovery within four months of a 35 year old female following complete resection of a teratoma and high doses of intravenous methylprednisolone and immunoglobulin's. In paraneoplastic limbic encephalitis the symptoms as previously described will usually precede the diagnosis of a teratoma. In an article by Stein-Wexler, R., Wootton-Gorges, S. L. & Greco, C. (2005), describe

the case of a 14 year old girl who presented with an unusual behavioural disturbance leading to coma, who, ten weeks later was found to have an immature ovarian teratoma. Although her symptoms improved slightly after excision of the tumour, she died whilst in rehabilitation. However the article does not go on to explain the cause of death. Early diagnosis and treatment which includes surgical removal of the teratoma followed by plasmapheresis and corticosteroids results in prompt neurological response and recovery, as supported by Seki, M., Suzuki, S., Iizuka, T., Shimizu, T., Nihei, Y., Suzuki, N. & Dalmau, J. (2008).

Teratoma

Outwater, E. K., Siegelman, E. S. & Hunt, J. L. (2001), describes teratomas as a class of tumours known as nonseminomatous germ cell tumour and are the result of an abnormal development of germ cells and embryonal cells. Teratomas derived from germ cells occur in the testes in males and ovaries in females. A teratoma is a congenital encapsulated neoplasm containing tissue normally found in organs such as the brain, thyroid, liver and lung. On rare occasions they may contain organs, often reported as hair, teeth, bone, or complex organs. Although teratomas are considered to be congenital, they are often not diagnosed until later in life, the patient often presenting with abdominal or pelvic pain. Classified using the Gonzalez-Crussi grading system a mature teratoma is graded a zero and considered benign, however, the definitive diagnosis is based on the histology.

Patients and Method

The authors identified two patients at separate tertiary referral hospitals admitted concurrently who exhibited acute psychiatric and behavioural symptoms with movement disorder and prolonged unresponsiveness. The similarities between the two cases and the eventual diagnosis of paraneoplastic limbic encephalitis associated with ovarian teratoma lead us to review and report the cases. The presenting features, clinical course and ovarian pathology are consistent with the reported literature and will be discussed.

Case One

A 17 year old female was admitted to her local hospital for investigation of a two week history of headache and fever associated with one day of confusion. The patient, who was born in Australia, was a full time school student with no history of travel outside of Australia. On presentation to hospital she became agitated with severe short term memory impairment. At this time, a differential diagnosis of viral encephalitis was made so she was transferred to a tertiary referral hospital with a specific neuroscience service for further investigation and treatment. She was commenced on intravenous methylprednisolone and acyclovir. A lumbar puncture was attended with the following results: 1 polymorph, 48 mononuclears, and 11 red blood cells, the cerebrospinal fluid (CSF) was negative for herpes simplex virus (HSV) and cryptococcal infections.

On day 1 post admission the patients Glasgow Coma Scale (GCS) decreased to 4 (E1, V1, M2), with airway compromise, requiring intubation and subsequently she was transferred to the intensive care unit (ICU). She displayed no obvious seizure activity, however, the electroencephalogram (EEG) showed diffuse delta wave activity, a high amplitude wave that is usually associated with slow wave or deep sleep. The magnetic resonance imaging (MRI) demonstrated high signal, indicating an increase in neural activity. The hypersensitivity in the limbic region is frequently seen in two disorders: paraneoplastic limbic encephalitides and encephalitis associated with voltage gated potassium channel antibodies (Sansing, L. H., Tuzun, E., Ko, M. W., Baccon, J. Lynch, D. R. & Dalmau, J. 2007). She was seen by the consultant neurologist who subsequently documented “In view of the patient’s age, sex, acute presentation and rapid deterioration with respiratory failure, the clinical features were consistent with the diagnosis of paraneoplastic limbic encephalitis”. An urgent abdominal and pelvic CT scan was ordered to look for a possible ovarian teratoma.

Three days after her admission she underwent an oophorectomy and cystectomy, the pathology confirmed a mature teratoma with neural elements. Despite the removal of the teratoma, the patient continued to deteriorate with a decrease in responsiveness, loss of reflexes and the commencement of rhythmic facial movements with grimacing and tongue protrusion. At times the mouth movements were synchronous with complex movements and dystonic posturing of the upper and lower limbs. The movements increased in severity when the patient was stimulated even with simple nursing care and were refractory to sedative drugs including an infusion of dexmetmidine. She subsequently required an extended period of sedation on propofol and ventilation. With consent from her mother, another lumbar puncture was attended and the CSF was sent to a North American laboratory for analysis by Dr. Joseph Dalmau, the only institution testing for N-methyl-D-aspartate (NMDA) receptor antibodies. NMDAR is a specific type ionotropic glutamate receptor which is the predominate molecular device for controlling synaptic plasticity

and memory function (Li, F. & Tsien, J. Z. 2009). Dr. Dalmau is a neurologist who has a particular interest in paraneoplastic encephalitis and has published numerous papers on the condition. The CSF analysis detected anti NMDA receptor antibodies.

Three weeks following admission, there was no sign of improvement in her condition and a repeat abdominal CT scan showed a small lesion on her remaining ovary, which may have been residually secreting antibodies. A family conference was convened in which the patient's poor neurological condition and CT scan results were discussed. An oophorectomy of her remaining ovary and cryopreservation of the ovary was discussed at length. Three days following the family conference, the patient's mother gave permission and the patient underwent a right sided oophorectomy. However, the frozen section indicated normal tissue. A trial of plasmapheresis was also initiated in an attempt to rapidly remove the disease causing auto-antibodies. Plasmapheresis is commonly used to treat a variety of disorders including those of the immune system such as myasthenia gravis and Guillain-Barre syndrome. The patient required a mouth guard to be fitted due to tongue trauma from the constant rhythmical mouth movements to her tongue, which was protruding. To enable the patient to be weaned from the ventilator a tracheostomy was inserted, two months following her initial admission to hospital, the patient was transferred out of the ICU.

Six months following admission the patient's condition remained essentially unchanged. She was inconsistently responding to her mother's voice and tracking with her eyes to various movements and sounds in the room, however, she was still unable to follow commands. As the patient's condition remained in a constant state with no obvious benefits from previous treatments, the family were reluctant to trial any suggested new treatments. One such treatment was a trial of cyclophosphamide, a chemotherapy agent which decreases the immune systems response to various diseases and conditions. During this time, the patient was successfully weaned to continuous positive airway pressure (CPAP) and eventually a humidified circuit via a tracheostomy. A percutaneous endoscopic gastrostomy tube (PEG) was inserted for feeding. She was commenced on hormone replacement therapy following her bilateral oophorectomy and baclofen for the prevention of limb contractures and dystonia.

Following a family conference, a vascath was inserted and plasma exchange commenced, however, there was no visible improvement with this treatment and the patient was commenced on a six month course of cyclophosphamide. The tracheostomy tube was weaned slowly and eventually removed and the patient was transferred to a brain injury specific rehabilitation unit eight months after her initial admission. At the time of her transfer, she was requiring hoist transfers from bed and maximum assistance with back support and head control whilst sitting for periods of 5-10 minutes. She would close her eyes during therapy sessions but would respond to voice, assurance and touch. She remained aphasic, with PEG feeding and required full assistance with all care including showering and personal grooming. The dystonic movements continued despite baclofen and intermittent oral doses of diazepam.

The patient was discharged home in the care of her family fourteen months following her initial admission where she continues ongoing outpatient rehabilitation therapy. Unfortunately it is now over two years since her presentation and admission, she remains aphasic, wheel chair bound, tube fed and requiring full assistance with all activities of daily living with severe behavioural deficits including aggression.

Case Two

A 21 year old female presented to a psychiatric unit at a major teaching hospital with a manic episode, she was experiencing an elevated mood, poor sleep pattern and disinhibited behaviour. She was also displaying episodes of delusions, where she thought she had special powers and could read peoples' minds and predict the future. She was commenced on olanzapine a medication commonly used to treat schizophrenia and bipolar disorders, sodium valproate and diazepam. There was a family history of schizophrenia.

Her mania persisted and over the next two weeks she became more delirious and attempted to jump a three metre brick fence, which resulted in, a fractured left foot. The left foot fracture was surgically repaired and she was transferred to the neuroscience unit postoperatively. Three weeks following admission she was commenced on lithium, to treat her mania. Lithium poses a very important antisuicidal effect not shown in other stabilising medications. She had a tonic clonic seizure for which she was given 10mg of midazolam and an EEG was ordered.

Following her first session of electroconvulsive therapy (ECT) she became mute. Her observations were as follows, blood pressure was 120 / 82mmHg, pulse 99bpm and temperature of 37.6° C. That evening she became agitated and febrile and had another seizure for which she was given 10mg midazolam. Following a review by the psychiatrist her midazolam was changed to haloperi-

dol to treat her psychoses. Also at this stage she underwent a septic workup, but all results returned negative.

A MRI was undertaken, revealing old lesions likely secondary to drug use of limited significance and not indicative of active disease. There was no evidence of encephalitis, cerebritis or vasculitis.

Over the next two days her high body temperatures persisted and she had a lumbar puncture under general anaesthetic. Following this procedure she had a catatonic episode. On day 39 she had a further three seizures over a 15 minute period where her oxygen saturation dropped to 79%, her airway was compromised, she became unresponsive and had a cardiac arrest. She was intubated and sent for an urgent CT scan, loaded with 1gram of phenytoin and also commenced on 300mg sodium valproate twice daily. She was extubated the same day as she was able to maintain her own airway. The lumbar puncture results showed clear CSF with no cells.

Over the next five days her body temperature remained elevated, her blood cultures were normal, and she remained agitated and aggressive. During this time correspondence with colleagues in the USA suggested that "impression – paraneoplastic effect, primary unknown (possible ovarian teratoma)". She was then commenced on intravenous octagam 25 milligrams and 21 milligrams alternate days for one month. Octagam is a plasma derived blood product containing human normal immunoglobulin with a broad spectrum of antibodies against infectious agents.

Over the next three weeks her GCS fluctuated from 7-9. During this period E-coli was identified in her urine for which she was treated with Timentin. Another lumbar puncture was completed and the CSF remained clear. The formal CSF results came back from the USA which showed positive NMDA antibodies in the CSF and serum. This was consistent with ovarian teratoma precipitating a paraneoplastic effect.

On day 74 she and her mother were seen by the fertility fellow to discuss cryopreservation of her ovaries. She remained agitated and requiring small doses of benzodiazepines. She continued her plasma exchange, and her GCS had improved to 13-15.

Four months following admission she underwent a laparoscopic bilateral oophorectomy. Over the next week, the plasmapheresis continued and serum was sent to the USA to check NMDA antibody levels. Over the next couple of weeks her agitation began to improve and the benzodiazepines was ceased. The antibody results were positive for NMDA-R for which she was given 600mg daily of intravenous rituximab over the next four weeks. Rituximab is an anti CD20 antibody that depletes B-cells and potentially the source of paraneoplastic antibody production. B-cells cross the blood brain barrier and intrathecal production of antibodies correlates with clinical severity in paraneoplastic disease. Rituximab is primarily used in the treatment of many lymphomas, leukaemia's, transplant rejection and some autoimmune disorders.

For the next three and half months she fluctuated from being confused and agitated to being pleasant and cooperative. She was transferred to a rehabilitation unit nine months following her initial admission, but only stayed for one day, agreeing to outpatient rehabilitation services, on the stipulation she remain in her mother's care.

She is now back to her previous social activities and has returned to her performing artistic endeavours. Two neuropsychology assessments have shown that she is cognitively performing at a high-average range.

Conclusion

The authors have described two similar cases of young women who presented to separate tertiary referral hospitals in Sydney, Australia. Nursing these young women was extremely challenging as was caring for their family members due to the complex and often uncertain course of their illness. Families often experience a wide range of emotions and adapt in their own way in a time of acute illness. The unfamiliar surrounds and treatment options often interfere with a family's normal coping mechanisms. The Clinical Nurse Consultants at both hospitals assumed the role of contact person for the families. Both families expressed concern in regards to the uncertain prognosis. Support was offered in a variety of ways and short term realistic goals were established to assist in providing equilibrium and both families were kept updated on the patient's condition and progress on a regular basis. Family members were encouraged to express their feelings and keep a journal of their child's daily experiences to help them notice small gains and improvements. Counselling services were also offered. The flexibility of visiting hours and encouragement to participate in the patients care resulted in positive outcomes such as improved orientation and decreased anxiety. This also encouraged communication and collaborative relationships between staff and family members.

As primary caregivers, the nursing staff were also

provided with education in regards to the disease process as well as verbal support to minimise burnout due to the unusual and complex disease and the duration of admission.

Although the patients presented simultaneously they had differing time frames to confirmation of a diagnosis and ultimately vastly different outcomes. In both cases the patient's presented with neuropsychiatric symptoms, prolonged periods of unresponsiveness, accompanied by a distinctive movement disorder. The movements consisted of repetitive ocular, chewing, facial grimacing, limb and trunk movements, including sustained posturing. At times the movements were synchronous and were influenced by sensory stimulation even though they often occurred during a depressed conscious state. Neuropsychiatric symptoms and movement disorders are prominent in the literature reporting ovarian teratoma associated encephalitis (Vitaliani, R., Mason, W., Ances, B., Zwerdling, T., Jiang, Z. & Dalmau, J. 2005 & Klenig, T. J., Thompson, P. D., Matar, W., Duggins, A., Kimber, T. E., Morris, J. G., Kneebone, C. S. & Blumbergs, P. C. 2008). PLE is a complicated and complex condition and diagnosis and treatment decisions rely on the clinical picture combined with MRI and EEG abnormalities in the temporal lobes and frequent presence of inflammatory changes in the cerebrospinal fluid.

Early recognition of PLE and prompt intervention with immune therapies at the onset of presentation should translate into more favourable neurological outcomes, however as highlighted in these case studies this may not always be the case.

Following these challenging cases the authors stress the importance of considering the diagnosis and investigation of paraneoplastic limbic encephalitis in any young female presenting with psychiatric symptoms, personality changes, cognitive and behavioural dysfunction, memory loss and seizures.

References

Corsellis, J. A. N., Goldberg, G. J. & Norton, A. R. (1968). Limbic encephalitis and its association with carcinoma. Brain 91: 481-496.

Dalmau, J. & Tuzun, E. (2007). Limbic encephalitis and variants: classification and treatment. Neurologist 13 (5) 261-271.

Henry, C., Husson, H. & de Broucker, T. (2009). Autoimmune limbic encephalitis with anti NMDA antibodies and ovarian teratoma: a treatable case of paraneoplastic limbic encephalitis. Revue Neurologique 165 (1) 70-75.

Kerling, F., Blumcke, I. & Stefan, H. (2009). Pitfalls in diagnosing limbic encephalitis- a case report. Acta Neurologica Scandinavica 118 (5) 339-342.

Khadem, G. M., Heble, S., Kumar, R. & White, C. (2009) Anti-N-methyl-D-aspartate receptor antibody limbic encephalitis. Internal Medicine Journal. 39 (1) 54-56.

Klenig, T. J., Thompson, P. D., Matar, W., Duggins, A., Kimber, T. E., Morris, J. G., Kneebone, C. S. & Blumbergs, P. C. (2008). The distinctive movement disorder of ovarian teratoma-associated encephalitis. Movement Disorders 23 (9) 1256-1261.

Lawn, N. D., Westmoreland, B. F., Kiely, M. J., Lennon, V. A. & Vernino, S. (2003). Clinical, Magnetic Resonance Imaging, and Electroencephalographic findings in paraneoplastic limbic encephalitis. Mayo Foundation for Medical Education and Research 78, 1363-1368.

Li, F. & Tsien, J. Z. (2009). Memory and the NMDA receptors. The New England Journal of Medicine 361, 302-303.

Outwater, E. K., Siegelman, E. S. & Hunt, J. L. (2001). Ovarian Teratomas: tumor types and imaging characteristics. The Journal of Continuing Medical Education in Radiology 21, 475-490.

Sansing, L. H., Tuzun, E., Ko, M. W., Baccon, J. Lynch, D. R. & Dalmau, J. (2007). A patient with encephalitis associated with NMDA receptor antibodies. Nature Clinical Practice Neurology 3 (5) 291-296.

Seki, M., Suzuki, S., Iizuka, T., Shimizu, T., Nihei. Y., Suzuki, N. & Dalmau, J. (2008). Neurological response to early removal of ovarian teratoma in anti-NMDAR encephalitis. Journal of Neurological and Neurosurgical Psychiatry 79 (3) 324-326.

Stein-Wexler, R. Wootton-Gorges, S. L. & Greco, C. (2005). Paraneoplastic limbic encephalitis in a teenage girl with an immature ovarian teratoma. Paediatric Radiology 35 (7) 694-697.

Vitaliani, R., Mason, W., Ances, B., Zwerdling, T., Jiang, Z. & Dalmau, J. (2005). Paraneoplastic encephalitis, psychiatric symptoms, and hypoventilation in ovarian teratoma. Annals Neurology 58 (4) 594-604.

Calendar of Upcoming Events

2011:

May 4 - 7, European Association of Neuroscience Nurses Congress. Belgium.
www.eann2011.org

Neuroscience & Trauma 2011, Seminars.
$66/day Westmead Hospital, NSW.
Contact: Katherine Schaffarczyk, 9845 7590
Katherine_Schaffarczyk@wsahs.nsw.gov.au
June 30: Subarachnoid Haemorrhage
August 25: Neurology Update (MS, Huntington's Disease, Epilepsy & PD)
October 20: Trauma Nursing

June 3, NNPDSC. Menzies Hotel, Sydney.
www.dcconferences.com.au

July 12 - 14, Toronto, Canada.
SickKids Centre for Brain & Behaviour 2nd Biennial Conference - Brain Injury in Children www.sickkidsbrainconference.ca

August 4 - 5, Smart Strokes. Queensland.
www.strokefoundation.com.au

August 5 - 6, Cerebro-Vascular & Neuro-Oncology. Macquarie Neurosurgery, Sydney**.** www.mqneurosurgery.com.au
Tel: (02) 9812 3510

October 5 - 7, ANNA Conference. Perth.
www.anna.asn.au

October 6 - 8, Nurse Practitioner. Adelaide South Australia
www.acnp.org.au

October 27 - 28, The **8th Annual Krembil Neuroscience Symposium.** The Old Mill Inn & Spa, Toronto, Canada.
www.krembil.com or
Rosalie.Magtoto@uhn.on.ca

November 18 - 20, British Association of Neuroscience Nurses Conference. Sheffield, UK.
www.bann.org.uk

November 23 - 25, Nurse Educators. Hamilton, New Zealand.
www.nursed.ac.nz

2011
ANNA ANNUAL SCIENTIFIC MEETING
"Black & White", Or Is it? – Neuroscience Nursing Practice.

Hyatt Regency. Perth, WA.
5 – 7 October, 2011

~ Call for Abstracts ~

Submit abstracts via the website
Closing Date: **12th July**
www.anna.asn.au

2012: American Association of Neuroscience Nurses Conference

Saturday, April 28 – Tuesday, May 1
Washington State Convention & Trade Center
Grand Hyatt Seattle
Hyatt at Olive 8
Seattle, WA. USA

2013: American Association of Neuroscience Nurses Conference

Saturday, March 9 – Tuesday, March 12
Charlotte Convention Center
Westin Charlotte
Charlotte, NC. USA

2013: World Federation of Neuroscience Nurses Congress

September 13 - 17.

Nagaragawa Convention Center.
Gifu, Japan.
www.wfnn.nu

The World Federation of
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Agnes Marshall Research Grant Award (AMRGA)

Agnes Marshall-Walker is credited with co-founding the American Association of Neuroscience Nurses in 1968 – an organisation thriving today. One year later, Agnes orchestrated an organizational meeting with neuroscience nursing leaders worldwide and WFNN was created. Today, the WFNN boasts more members than ever and continues to promote the highest standards for patients and nurses alike.

The WFNN Board of Directors created the Agnes Marshall-Walker Research Grant Award to support neuroscience nurses in their scientific pursuits of scholarly inquiry into patient care and management issues. The first award was presented at the 5th WFNN Congress held in Anaheim, USA in 1989. Today, this award totals US$4,000 and is awarded at each WFNN Congress.

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