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AJON, much more than Anatomy & Physiology

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In This Issue:

Editorial

Linda Nichols
Editor

Making Connections

One would think that the world of academia would have me well versed and prepared for constant change, however one area where I struggle, is the ever growing field of scientific journals. The noun publication is derived from the Latin word *publicare*, meaning "make public." Nursing journals remain the primary method in which we share information and research findings. However, we are now in a world where our reading and interests are peaked by tweets and notifications and this has also transformed the way we share information. The down side of this is that paper based journals are now struggling to reach the wider public. For those returning from Croatia and the WFNN congress I am sure you can all appreciate the importance of sharing and collaboration, so it's probably a fitting time to reflect on this further and strive towards making the AJON more accessible to the public.

With Florence Nightingale a revolution in nursing had begun and the first nursing journal *The Nightingale* was published in March 1886. The *American Journal of Nursing* soon followed in 1900, however it was not until 1952 that *Nursing Research*, the first dedicated research journal was published. There was a steady increase in the number and variety of nursing journals over the years, with the advent of evidenced-based nursing prompting further growth. The internet and online publishing perhaps prompted the largest changes and growth in the number of publications available.

I was recently reading a 1940 extract from the *Medical Journal of Australia* titled 'The problem with medical literature'. The extract focused on the belief that there were far too many medical journals, describing the existence of 130 medical periodicals in Great Britain as appalling and terrifying as well as presenting increased stress for both librarians and researchers. I wonder what the author would think of today's climate with the thousands upon thousands of scientific journals available.

Guest Editorial

Dawid Cecula
CEO Exeley Ics New York

Wire Together….

In the world, over 95% of scholarly journals that offer open access to their electronic content are financed by the research institutes, societies, universities and other organizations, to which the journals belong. Such institutions crave solutions that grow the journals' quality and status, but do not grow their budgets.

They often consider outsourcing some or all publishing functions to professional publishing companies. Also, many editors want to focus on the editorial work and would prefer to not get involved in journal production, distribution and marketing.

The most common outsourced services include:

Journal profile at the professional platform
allocation of DOI and metadata distribution to CrossRef .

Arranging for indexing by abstracting and indexing services.

Arranging for coverage by full-text repositories and content distribution to such repositories.

Arranging for coverage by open access directories.

Indexing by Google and other search engines
preparing application to Clarivate Analytics/ Scopus/Medline.

Consulting on how to grow citations and increase Impact Factor/Citescore.

Consulting on how to grow reference linking
reporting: usage statistics & altmetrics.

As a rule, journals owned by societies can afford only very basic electronic publishing solutions. They simply put their articles as PDF files on their own servers. Readers feel like they are at a car show. They can watch the cars, but cannot enjoy driving them. They can read the articles, but cannot enjoy all the functionalities offered by modern publishing technology solutions.

Although online publishing has eased the burden of managing paper copies, the challenge for researchers has grown exponentially with the task of locating the most recent relevant literature from the best available source a constant challenge.

Neuroscience nurses must be astute and observant as effective, rapid change management continues to be at the forefront of improved outcomes. However, real change can only occur when we share those findings. The Nursing Board of Australia (2016) Standards of Practice dictate that Registered Nurses must engage in professional development, this is not only personal, but also of others through evidenced based educational activities including manuscripts. As we consider making new connections and publishing our work, it is a fitting time for the AJON to be making new connections as it heads to an online platform.

This is an exciting move, the Exeley team have been working hard in the background to develop this online platform and market the AJON. With this partnership we hope that the AJON will continue to grow and be more public. The AJON has always prided itself on the quality of papers and the comprehensive peer review process and as we move towards an electronic format, I know that this quality will continue.

Linda

Exeley is filling the gap by offering top publishing technology to quality society journals all over the world.

The Australasian Journal of Neuroscience (AJON) recently joined Exeley and is now available through a sophisticated hosting platform. Readers can, for instance, read full texts on mobile devices, share content on social media with one click, use live links in references to easily visit cited papers and register for alerts to receive an automatic message if the journal has published a new issue or an article on the topic of their interest. Editors and Authors can track articles' popularity by visiting a metrics dashboard that provides data on visits, mentions and citations thanks to the altmetrics board.

The most important thing is that AJON became a part of a network of scholarly content – will be integrated with databases and services by distributing the content and metadata via Exeley repository and will be linked to the other publications. When one journal becomes a member of this family they benefit from the integration – like neurons that persistently take part in "firing" each other.

Time to make new connections and welcome on board!

Dawid

The complications of jejunostomy tubes for patients receiving Duodopa: New challenges for neuroscience nurses

Rachael Elizabeth Mackinnon

Abstract:

The use of Duodopa ® Levodopa-Carbidopa intestinal gel offers patients with advanced Parkinson's disease (PD) an effective alternative therapy for the treatment of severe motor fluctuations and dyskinesia. This therapy requires the use of percutaneous endoscopic gastrostomy/ jejunostomy tube (PEG/J) to deliver gel directly into the jejunum which poses new challenges for neuroscience nurses for the care and management of patients with PD. Due to the reported number of complications associated with PEG/J our facility opted to use a direct jejunostomy tube for the first of two PD patients which resulted in an adverse outcome for our 80 year old patient. This experience highlighted that the neuroscience nurses need to increase knowledge and understanding of PEG/J and jejunostomy care as more future patients will be treated with Duodopa, and that future studies regarding the safety and value of the direct jejunostomy tubes are warranted.

Key Words: Parkinson's disease, percutaneous endoscopic gastrostomy/jejunostomy (PEG/J), Direct Endoscopic Jejunostomy (DEJ), Jejunostomy, Duodopa, complications

Background:

An 80 year old man with advanced Parkinson's disease (PD) was admitted to the neuroscience unit with a worsening decline in mobility. Medical management was the commencement and titration of the levodopa-carbidopa intestinal gel (LCIG) Duodopa ® via a naso-jejunal tube, which had been inserted under fluoroscopy in Interventional Radiology. Over a ten day trial period the patient responded well to the administration of the LCIG with much less periods of difficulty with movement (known as OFF times) although he did continue to experience periods of dyskinesia, some paranoid behaviours and notable episodes of 'punding' (repetitive non-purposeful movements). Following these promising results from the LCIG infusion, the patient consented to proceed to the insertion of a direct jejunostomy for permanent trans-jejunal intestinal infusion by a upper gastrointestinal surgeon. On day 2 post insertion of the jejunostomy tube the patient complained of nausea and vomiting and overnight he was transferred to the intensive care unit (ICU) with a suspected bowel obstruction and an aspiration pneumonia.

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Do the benefits of Duodopa eclipse the complications?

Duodopa ® has been increasingly accepted as an effective treatment for troublesome motor fluctuations and dyskinesia for patients with advanced Parkinson's Disease (Lang et al., 2016; Nyholm et al., 2008; Olanow et al., 2014).Oral administration of levodopa leads to variable plasma levels from erratic gastric emptying (Nyholm et al., 2008).The intraduodenal delivery of levodopa connected to a portable pump provides a relatively steady plasma level of levodopa (Chang et al. 2016).

The usual delivery of LCIG is via a percutaneous endoscopic gastrostomy/ jejunostomy tube (PEG/J). This system uses a percutaneous gastrostomy tube with fine bore jejunal extension, so that the gel can be directly infused into the jejunum where absorption of the medication will be optimized (Tsui, 2014). For the patients the neurologists opted for the insertion of a direct jejunostomy tube, with the placement of the tube directly into the small intestine for the administration of the gel. This decision was based on the reported complications associated with the PEG/J delivery system (Kimber & Shoeman (2014). It also highlighted the notion that persistence in focusing on the clinical benefits of Duodopa has the potential to eclipse the challenges and complications of introducing a new jejunostomy tube for patients with an already debilitating disease (Bianco et al., 2012).

Complications related to the use of the PEG/J tube:

Within the studies on the benefits of Duodopa are reports of tube/stoma complications. Nyholm et al (2008) reports the most common complication for the PEG/J tube was dislocation of the tube from the small intestine to the stomach. Another two studies noted that the main safety issue of the LCIG related to the infusion system with technical problems associated with kinking and blocking of the tube (Senek & Nyholm, 2014; Zibetti et al., 2014). Zibetti et al (2014) reported one duodenal perforation out of 59 patients, while Kimber & Shoeman (2014) report two gastric perforations out of 17 patients which required laparotomy to repair. Recurrent minor problems were tube malfunction and dislocation secondary to punding (Chang et al., 2016). A constantly dislodged tube requires repeated re-siting of the jejunal tube (Foltynie et al., 2013), which exposes patients to a return to the endoscopy suite and the increased risks of hospitalization and general anaesthesia (Kimber & Schoeman, 2014).

Complications are also related to infection around the stoma, (van Laar, Nyholm, & Nyman, 2016) with reports of excessive granulation tissue, incision site erythema, abdominal pain, peritonitis and pneumoperitoneum (Fernandez et al., 2015; Zibetti et al., 2014). Zibetti et al (2014) noted infection tended to occur within one month of the PEG/J procedure and were successfully treated with antibiotic therapy, however device complications were the contributing reason for discontinuation of the infusion for a proportion of patients.

A study of 85 patients undergoing Duodopa infusion was conducted regarding nutritional status and weight loss in patients and determined that those without tube complications had significant weight gain over a 6 month period (Galletti et al., 2011).

Major complications of PEG/J - Buried Bumper Syndrome:

Buried bumper syndrome (BBS) occurs when there is an overgrowth of the gastric mucosa over the inner bumper of the gastrostomy tube. Predisposing factors for BBS are tight fitting gastrostomy tubes, weight gain and no mobilization of the tube for the first month (Santos García et al., 2016). BBS was reported to have a higher incidence of occurrence in Freka PEG tubes (which is the preferred PEG/J tube for Duodopa), compared with a Corflo PG tube in one study (Dowman et al., 2015). Although another similar sized study claims a low incidence of BBS from Freka tubes published in October this year (Clarke & Lewis, 2016). Notably both studies examined the incidence of BBS in PEG/J tube that had been required for the purposes of enteral feeding and not for Duodopa administration.

Bezoars and Phytobezoars:

Bezoars are composed of undigested food material that has been orally ingested (Altintoprak et al., 2012) and are classified based on the type of material they contain. Phytobezoars are described as occurring in patients who consume high amounts of fibrous and long fibre foods such as asparagus or spinach that may be difficult to digest (Altintoprak et al. 2012). In a case of a 21 year old male who had received LCIG for 6 months a blockage of his tubing was discovered to be jejunal tube being knotted in the stomach around a bezoar (Negreanu et al., 2010).

A 70 year old man presented with abrupt motor deterioration from tube obstruction from a bezoar. He was treated with a liquid diet and the use of Coca-Cola ® over four days until the bezoar was successfully dissolved (Stathis, Tzias, Argyris, Barla, & Maltezou, 2014).

In another case it was reported that a phytobezoar entrapped the tip of a 71 year old male patient's jejunal tube and resulted in a jejunal wall perforation and fistulisation of 3 intestinal loops. Unfortunately the patient was reported to have died post-operatively following repair of the fistula (Vuolo et al., 2012). Given the non-motor symptoms of Parkinson's disease that include poor gut motility and constipation (Fasano, Visanji, Liu, Lang, & Pfeiffer, 2015) it would seem that the risk of bezoars would be higher when coupled with reduced gastrointestinal motility caused by the PEG/J.

A long term PEG/J study determined that the procedural outcomes and adverse rates in patients treated using the PEG-J drug delivery system were acceptable, and that benefits of the therapy outweighed these complications (Epstein et al., 2016). Kimber & Shoeman (2104) felt that the high number of PEG/J complications was justification to introduce the use of the DEJ tube.

Small bowel obstruction secondary to Jejunostomy tube:

An abdominal CT scan reported that our 80 year old patient had a jejunal obstruction due

to kinking at the level of the jejunostomyballoon; a distended stomach and fluid filled oesophagus, with consolidation in the lung bases secondary to aspiration. The balloon of the jejunal tube was deflated and a Salem sump nasogastric tube was inserted. Blood cultures returned an Enterobacter bacteraemia which was treated with IV antibiotics. He was given a beta-blocker for a new onset of atrial fibrillation secondary to his aspiration pneumonia and returned to the ward after 3 days in ICU. Once on the ward a new jejunostomy tube was inserted under fluoroscopy and sutured into place, following dislodgement of the first Jejunostomy without the ballon inflated for securement of the tube. He was restarted on the LCIG again with good motor results, less punding and no further hallucinations or paranoia. Two days after insertion of the second Jejunostomy tube, oozing around the tube necessitated review by the Stoma Clinical Nurse Consultant and the placement of an ileostomy bag. He was eventually discharged to a rehabilitation hospital and eighteen months later the patient reports fluctuations in the amount of ooze/ leaks around the stoma site, which is temporarily alleviated with reductions in faecal loading through the use of regular aperients.

PEG, PEG/J and Jejunostomy tubes:

The nurses on the neurological ward are familiar with percutaneous endoscopic jejunostomy (PEG) tubes which are used routinely for enteral nutrition for patients at high risk of aspiration typically following stroke or traumatic brain injury. PEG/J feeding tubes are rarely used on our ward, but were reported to be developed for jejunal feeding to reduce gastroesophageal reflux occurring in PEG feeding. These tubes presented new challenges with PEG/J malfunction due to clogging and proximal migration of the extension tube back into the stomach (Panagiotakis, DiSario, Hilden, Ogara, & Fang, 2008), which was also noted in the studies for Duodopa. The same authors studied the benefit of a direct percutaneous endoscopic jejunostomy tube (DPEJ) to PEG/J and determined in a retrospective study of 75 patients a decrease in the overall incidence of aspiration pneumonia.

A search of the hospital's protocol on PEG and jejunostomy tube returned guidelines for the role in enteral nutrition only with limited information for the care of a direct Jejunostomy for the sole purpose of medication administration. A search on CINAHL to compare rates of complications between DEJ to PEG/J retrieved only one retrospective study of 560 patients where the tubes were used for the purposes of enteral feeding indicated in patients with GIT / Head and Neck cancers, Stroke and other neurologic conditions which were not specifically identified (Ao, Sebastianski, Selvarajah, & Gramlich, 2015). Ao et al. (2015) concluded that there was a higher risk of tube related complications, particularly the requirement of tube replacement in the patients with the DEJ tubes (48.4%) than that of the PEG group (21.5%). To date the only other study which directly compares the two devices is a small study of 17 patients for Duodopa ® infusion where the authors advocated DEJ as a feasible alternative to the PEG/J tubes. This study reported a lower incidence in tube malfunction when comparing 8 patients undergoing PEG/J to 9 patients who received DEJ devices (Kimber & Schoeman, 2014).

Conclusion:

The administration of the LCIG has provided patients with advanced Parkinson's disease with great benefits in motor fluctuations and dyskinesia. The delivery of the intestinal gel requires an invasive PEG/J tube which brings a new set of challenges for these patients and the nurses caring for them. There is a lack of compelling evidence to support the introduction of the direct Jejunostomy tube having greater benefits, as opposed to the PEG/J. Further future studies are warranted not only to compare the safety and the rates of complications between the two devices, but also to increase knowledge and develop sound protocols for patients/families and nursing staff when using the direct Jejunostomy device to reduce complications and adverse outcomes.

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Treatment of subarachnoid haemorrhage complicated by hyponatraemia

Jordyn A Butler

Abstract:

Background statement: Developing hyponatraemia after a subarachnoid haemorrhage is common, however it is known to worsen patient outcomes. This paper aims to review the practice of managing hyponatraemia in acute subarachnoid haemorrhage patients with administration of 3% hypertonic saline solution.

Aim: To enquire into the practice and policy of one of Melbourne's large Metropolitan hospital's current management of hyponatraemia in subarachnoid haemorrhage patients, and determine if the policy is both current and evidenced based.

Methods: A search of the terms "subarachnoid haemorrhage", "hyponatraemia" and "hypertonic saline" was used in databases including Pubmed, Medline and CINAHL. Literature was included if it discussed the use of hypertonic saline for hyponatraemia, the effect of hyponatraemia on subarachnoid haemorrhage patients and the potential causes of acute hyponatraemia. The articles and literature reviews were assessed for inclusion by the author.

Results: Patients with a subarachnoid haemorrhage and hyponatraemia should not be fluid restricted, as this is contraindicated. Patients should be administered 3% hypertonic saline to avoid hypovolaemia and slowly increase serum sodium to prevent onset or exacerbation of cerebral oedema.

Limitation: Lack of evidence based data and studies in regard to the dosing of hypertonic saline resulted in the lack of consensus with prescribing rates and volumes to be infused for severe hyponatraemia.

Key words: *Subarachnoid haemorrhage, hyponatraemia, hypertonic saline*

Introduction:

Aneurysmal subarachnoid haemorrhage can be complicated by acute hyponatraemia in neurosurgical patients. De Oliveira Manoel et al., (2016, p. 1) define aneurysmal subarachnoid haemorrhage (SAH) as *'a complex neurovascular syndrome with profound systemic effects and is associated with high disability and mortality'*. An aneurysmal SAH is the result of cerebral aneurysm rupture or trauma, thus resulting in bleeding in the subarachnoid space. Rupture of cerebral aneurysms commonly occurs at bifurcations and branches within the Circle of Willis (Hickey 2014).

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Patients with a SAH commonly develop hyponatraemia within two weeks of cerebral rupture (Vrsajkov, Javanovic, Stanisavljevic, Uvelin, & Vrsajkov, 2012). Hyponatraemia is the most common electrolyte abnormality to develop in patients with a SAH. It is defined by Hickey (2014, p. 203) as *'serum sodium less than 135mEq/L'.* High-grade SAH patients with anterior circulation aneurysms have a 50% incident rate of developing acute hyponatraemia (De Oliveira Manoel et al., 2016), yet the pathophysiology linking SAH and hyponatraemia is not fully understood (De Oliveira Manoel et al., 2016; Manzanares, Aramendi, Langlois & Biestro, 2014; Mapa et al., 2016; See, Wu, Lai, Gross, & Du, 2016).

This enquiry into practice focuses on the treatment and management of hyponatraemia in SAH patients using hypertonic saline, with a structured discussion and analysis of current evidence based practice. The following will be explored; importance of a

high standard of care, a review of local policy relevant to SAH and hyponatraemia management and the impact on future nursing practices including a need for increased education. For this paper, the hospital has been de-identified, the reviewed policy is from a large metropolitan hospital with a 32 - bed ward with twelve dedicated neurosurgical beds including a 4 - bed neurosurgery high dependency unit (HDU).

Pathophysiology of SAH and hyponatraemia:

According to See et al. (2016) 30% of SAH patients develop hyponatraemia 1-week post rupture. Hannon et al., (2014) report 56% of patients admitted with SAH will develop hyponatraemia during their hospital admission. Due to the significant percentage of patients that develop hyponatraemia post rupture, these patients are at high risk for deterioration. It is crucial to observe for complications and symptoms associated with both SAH and hyponatraemia, with care provided being in accordance to current evidence based practice and local hospital policies. Hyponatraemia can occur in patients with a SAH due to syndrome of inappropriate anti-diuretic hormone (SIADH), cerebral salt wasting (CSW), glucocorticoid insufficiency and excessive use of diuretics (Hannon et al., 2014; Saramma, Menon, Srivastava & Sarma, 2013; See et al., 2016; Walcott, Kahle & Simard, 2012). According to Vrsajkov et al., (2012) & Mapa et al., (2016) the location of the aneurysm could potentially influence the patient's risk of developing hyponatraemia as aneurysms that rupture within the anterior circulation can affect the hypothalamic-pituitary region of the brain, consequently resulting in SIADH or CSW. SAH can lead to either an increase in secretion of antidiuretic hormone (ADH) causing SIADH or CSW due to the enhanced release of atrial natriuretic peptide, brain natriuretic peptide and noradrenaline. SIADH and CSW are fundamentally different conditions that can be difficult for clinicians to differentiate in regards to treatment (De Oliveria Manoel et al., 2016). It is crucial to initiate treatment targeted to the correct aetiology to ensure serum sodium levels are corrected appropriately; with a treatment plan reflective of the clinical situation and consideration of potential adverse effects (Ball & Iqbal, 2015; Hannon et al., 2014). Hyponatraemia can be life threatening if incorrectly treated and managed.

Intracellular and extracellular osmolarity must be equal. If there is a low serum sodium level, cells will begin to swell as fluid moves from the extracellular compartment to the interstitial fluid, resulting in intracellular oedema due to changes in osmolarity. When hyponatraemia develops rapidly the brain can be slow to adapt to the hypotonic environment (Mapa et al., 2016; Spasovski et al., 2014; Verbalis et al., 2013). When low serum sodium levels are over corrected too rapidly it can cause blood brain barrier breakdown and injury to myelin in the central nervous system, precipitating osmotic demyelination syndrome (Ball & Iqbal, 2015; Sterns, Hix & Silver, 2010; Verbalis et al,. 2013). Acute symptomatic hyponatraemia secondary to a SAH can have severe complications, with symptoms including cerebral oedema, seizures and cerebrovascular spasm (Saramma et al., 2013). Patient outcomes can vary significantly from a full recovery to severe disability or death post SAH, depending on severity of the bleed and associated complications (De Oliveira Manoel et al., 2016).

Nurses must accurately assess patients for changes in Glasgow Coma Scale (GCS) score and neurological condition. Whilst concurrently observing for signs and symptoms related to acute hyponatraemia including headache, nausea and vomiting, the nurse must be aware that the patient's condition can rapidly deteriorate leading to confusion, seizures, respiratory arrest and severe cerebral oedema resulting in death (Rafat et al., 2014; Verbalis et al., 2013).

TABLE 1: Serum sodium levels
This table lists serum sodium ranges of hyponatraemia and associated symptoms (Stern, 2015).

Hyponatraemia	Serum sodium range	Symptoms
Mild hyponatraemia	130-135 mmol/L	Nausea, vomiting, short-term memory loss & dizziness.
Moderate hyponatraemia	121-129 mmol/L	Confusion, muscle weakness, generalised malaise & headaches.
Severe hyponatraemia	<120 mmol/L	Lethargy, agitation, increased ICP, respiratory depression, cerebral oedema & disorientation.

Treatment of hyponatraemia:

Treatment and management of acute hyponatraemia in SAH patients should comply with hospital local policies, in conjunction with global evidence-based practice. The local policy at this hospital for 'IV infusion of hypertonic saline for hyponatraemia management'

has recently been reviewed and updated based on current evidence-based journal articles. Although medical professionals are prescribing the treatment, it is nurses administering the medication therefore it is paramount nurses administering hypertonic saline for severe hyponatraemia have sufficient knowledge and understanding of the high-risk infusion. According to See et al., (2016) the proportion of patients that developed hyponatraemia post clipping or coiling of an aneurysm, was almost equal. Hannon et al., (2014) also reiterated there was no difference in the incidence of hyponatraemia based on patients that had an aneurysm clipping or endovascular coiling. It is understood hyponatraemia may develop in response to hypothalamic injury as a result of SAH, consequently leading to aforementioned complications (Dority & Oldham, 2016; Vrsajkov et al., 2012). Due to increased renal reabsorption of free water in SIADH, fluid restriction is considered the gold standard of treatment. However, treatment of SIADH with fluid restriction in the setting of SAH is contraindicated and potentially detrimental to patient outcomes due to the increased risk of hypovolaemia-associated cerebral infarct and worsening vasospasm (De Oliveira Manoel et al., 2016; Hickey, 2014; Manzanares et al., 2014; Saramma et al., 2013; Walcott et al., 2012). Management of hyponatraemia in patients with a SAH includes preventing hypovolaemia and administration of isotonic fluid to prevent onset or exacerbation of cerebral oedema (De Oliveira Manoel et al., 2016; Raya & Diringer, 2014).

Hypertonic Saline:

The local hospital policy recommends for acute symptomatic hyponatraemia, an IV 3% hypertonic saline bolus of 100-250ml over 10 -20 minutes to correct low serum sodium levels, aiming for a sodium increase of 5mmol/L. The bolus can be repeated twice at 10-minute intervals if serum sodium remains unchanged (Adrogue & Madias, 2012; Verbalis et al., 2013; Grant et al., 2015; Spasovski et al., 2014). Starke & Dumont (2014) discuss the effects of hypertonic saline, due to its ability to move fluid from the interstitial and intracellular spaces via osmotic gradient into the intravascular system, thus reducing associated symptoms. The administration of hypertonic saline in SAH patients has been shown to increase arterial blood pressure, cerebral perfusion pressure and flow velocity whilst simultaneously reducing intracranial pressure and cerebral oedema (Starke & Dumont, 2014; Thongrong et al., 2014; Walcott et al., 2012). It is crucial to re-check serum sodium levels and urine osmolarity simultaneously post IV bolus and then repeat 2-4 hours post administration. To ensure accurate interpretation of values urine osmolarity and bloods should be taken at the same time (Spasovski et al., 2014). According to Adrogue & Madias (2012) 2-4 hourly neurological observations including GCS and vital signs should be performed, as well as serum sodium and urine electrolytes post-hypertonic saline administration, to ensure rapid overcorrection has not occurred. As per the reviewed hospital policy, patients administered with hypertonic saline require continuous cardiac monitoring and pulse oximetry in the HDU. In addition, an indwelling urinary catheter is inserted for accurate fluid balance due to the potential for large diuresis and this ensures the ability to obtain frequent urine osmolarity samples. Due to the high risks associated with hypertonic saline administration for SAH patients with severe symptomatic hyponatraemia, patients should not be left unattended whilst receiving the IV infusion. It can be observed that hospital policies are medical based, and can often lack guidance towards nursing practice and responsibilities. This signifies the need for policy change in conjunction with further nursing education for managing acute symptomatic hyponatraemia patients.

Hypertonic saline indications, infusion rates and target sodium concentrations have been described by Spasovski et al., (2014) as unclear, which can be challenging for nurses when prescribed and administered to patients. Currently there are no consensus guidelines for optimal concentration, infusion rates and dose. This is debated both in Australia and internationally regarding the administration and dosage of hypertonic saline for acute hyponatraemia, and remains an ongoing area of research due to inconsistencies in clinical recommendations. However evidence -based clinical practice guidelines are utilised to provide recommendations for clinically appropriate treatment and pathology testing (Nagler et al., 2014; Starke & Dumont, 2014; Thongrong et al., 2014). A review of current evidence-based practice at this hospital indicated that this local policy is in compliance with research findings, where a senior neurosurgical registrar decides rate and dosage for SAH patients.

Despite hypertonic saline improving symptoms associated with hyponatraemia and raising sodium levels, it can result in side effects including hypernatraemia, hypokalaemia and acute renal failure (Manzanares et al., 2014). It is important nurses administering hypertonic saline are aware of these side effects especially to observe for hypernatraemia, as rapid changes in serum sodium can have detrimental and permanent

neurological effects on the patient. Hence, serum sodium should not rise more than 10mmol/L within 24 hours and 18mmol/L within 48 hours to prevent osmotic demyelination syndrome (Ball & Iqbal, 2015; Grant et al., 2015; Rafat et al., 2014; Sood, Sterns, Hix, Silver, & Chen, 2013). Osmotic demyelination syndrome typically occurs 2-7 days post treatment and is clinically characterised by irreversible neurological damage (Sood et al., 2013). Manzanares et al., (2014, p. 236) reports, *'…in SAH, triple H therapy (hypertension, hypovolaemia and haemodilution) as an anti-vasospasm strategy promotes natriuresis and the risk of hyponatraemia'.* Thus reiterating the contraindication of fluid restricting SAH patients, as hypovolaemia and a negative fluid balance will worsen patient outcomes. Therefore utilising 3% hypertonic saline is the preferred treatment (Dority & Oldham, 2016).

Impact on future nursing practice:

It is imperative for nurses and clinicians to reflect upon current practices, challenge nursing interventions and management. Reflecting on current hyponatraemia management ensures the care provided to patients is based on current published research, whilst continuing to have a diagnostic approach in regards to accurate interpretation of serum sodium values. It was observed by McKeever et al., (2016, p. 85) that, '*delivering evidence-based nursing care contributes to improved patient outcomes, a superior quality of care, and potential cost efficiencies'.* Nurses can feel empowered and engaged when given the opportunity to contribute to improvements in nursing care and future practices within their speciality area, therefore improving the quality of care provided to patients. Although the local neurosurgery policy reflects current evidence based practices, there is a need for further nursing education. This is due to the infusion being high risk and may not be commonly administered in HDU settings, rather in the Intensive Care Unit. Increasing education will ensure nurses caring for SAH patients whom are at risk of developing hyponatraemia have a comprehensive knowledge of the condition, appropriate treatment as well as identifying signs and symptoms of deterioration. In conjunction, awareness of the risk factors of osmotic demyelination syndrome when administering hypertonic saline, such as malnutrition, liver disease and hypokalaemia (Rafat et al., 2014). Hyponatraemia can often lead to increased length of hospital stay, increased costs and associated complications, consequently highlighting the need for close monitoring of sodium levels and implementing appropriate treatment to reduce morbidity and mortality (Ball & Iqbal, 2015; Hannon et al., 2014; See et al., 2016). Increased knowledge of risk factors associated with SAH and hyponatraemia include advanced age, smoking, re-bleeding and cerebral vasospasm. This knowledge may prove to be vital in monitoring for sodium changes post – rupture in patients with pre-existing risk factors (Saramma et al., 2013).

Conclusion:

In conclusion, enquiry into practice is vital in continuing to develop and improve upon professional nursing practice whilst maintaining a high standard of evidence-based care. The incidence of SAH patients developing hyponatraemia is 50%, illustrating the importance of close monitoring of serum sodium concentrations and associated symptoms. Neurosurgical patients that become hyponatraemic during inpatient admission can result in increased length of hospital stay, increased morbidity and mortality, signifying the importance of monitoring serum sodium levels promptly, as well as for prevention of clinical consequences that can occur with untreated acute symptomatic hyponatraemia, such as cerebral oedema and increased intracranial pressure.

It can be concluded the reviewed local hospital policy for the monitoring, management and treatment of acute symptomatic hyponatraemia with administration of 3% hypertonic saline is in accord with current evidence based practice and global standard of care. However currently there remains a lack of consensus regarding the dosage and infusion rate of hypertonic saline, thus referring to clinical practice guidelines for recommendations in regards to treatment and management with SAH patients. Therefore to improve upon future local practice, increased nursing education is needed to be able to identify and determine signs and symptoms associated with hyponatraemia, monitoring for changes in serum sodium levels and in a timely manner escalating this to medical teams for appropriate intervention that is reflective of the clinical situation. Reflection into clinical practices and local policies proves invaluable to patient outcomes.

Acknowledgements:

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WFNN 2017 Congress Report

Vicki Evans
WFNN Vice President and Scientific Chair

Breaking news – www.wfnn.org

After four years of planning, the WFNN Congress in Opatija, Croatia has come to a successful completion.

As Scientific Chair of the Congress, the quality and range of abstracts was comprehensive and we tried to showcase a varied international cross-section, as this congress saw speakers from all corners of the globe. The program itself comprised 4 plenary sessions, 4 morning satellite sessions, 86 concurrent sessions and 72 posters.

There were over 450 delegates, including 46 Australians. The Australian contingent included 14 speakers and 6 presenting posters, a great representation from ANNA.

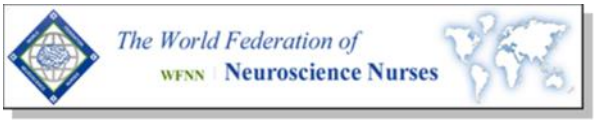
The Scientific Program commenced with an outstanding Pre-Congress workshop conducted by Linda Littlejohns entitled "What is wrong with my patient? Tying assessment of stroke, trauma and degenerative disease to 3D anatomy" using the computerised Anatomage table. Linda's association with WFNN has been lengthy and is always a great learning experience.

WFNN and the Neurocritical Care Society came together to offer the ENLS (Emergency Neuro Life Support) course for delegates prior to the start of the Congress. This was a successful offering and will be considered prior to each Congress.

The Social program was built into the Scientific Program, with half day outings to - Rovinj & Agrolaguna Vineyards; Brijuni National Park; Island of Krk & Vrbnik Wine Region; and Pula & the Olive Oil Museum.

Some countries have more resources than others and whilst the languages may differ, the process behind neuroscience nursing does not. Similar issues are faced the world over. Conferences like this one offers a great opportunity for networking and making new friends. However, a Congress such as this cannot run without the dedicated support of a great team of people, most of whom are volunteers. The respect and admiration for these people cannot be measured and professional and personal associations have been enhanced by this experience. Get involved and you'll reap the rewards. The networks you make will last for years and the friends forever.

Lastly, but most significantly, the WFNN Board of Directors, voted almost unanimously that the next Congress will be held in **Darwin, Australia 2021**. So let's make that Congress "the best ever"!



WFNN 2017 Congress Awards and Australian Presenters

Congratulations to the award winners and to the Australian presenters from the WFNN 2017 Congress. Before we know it 2012 will be upon us and I hope that you can take encouragement and inspiration from the following award winners, posters and presentations.

AGNES MARSHALL BEST PAPER

Prevention of and caring for patients with agitated behaviour in a sub-acute rehabilitation unit: From a manager´s perspective
Vivi Nielsen , Denmark.

AGNES MARSHALL BEST POSTER

Importance of multi- and/or interdisciplinary approaches to the quality of life of people affected by MS
Gabrijela Simunic, Tijana Milošević, Croatia.

LILY POTVIN AWARD

Childhood Epilepsy: A Comprehensive Overview for Nurses
Daniel Crawford, USA.

SGAMBELLURI AWARD

Destigmatisation Of Epilepsy In Children Through The Epilepsy Camps
Kristina Kuznik, Croatia.

2017 MITSUE ISHIYAMA LEADERSHIP AWARD
Lenka Kopacevic, Croatia.

2017 AUSTRALIAN ABSTRACT TITLES (ORAL PRESENTATIONS)

A Patient's Journey with Mitochondrial Disease
Aneeta Lal, Shajidan Maimaiti , Sydney, Australia.

How to keep pace with the changing clinical management in acute stroke
Kylie Tastula, Sydney, Australia.

EVD: Research or Routine?
Sharryn Byers, Sydney, Australia.

"Let's get physical"-The art of movement though a structured rehabilitation/ Parkinson's exercise 10 week program (PEP)
Sandra Krpez, Sydney, Australia.

A potential near miss: Could new technological innovation in nursing documentation have prevented a revolving door syndrome post pituitary surgery?
Erin O'Rourke, Victoria, Australia.

One in a million: a case of Stiff Person Syndrome
Joan Crystal, Queensland, Australia.

Nursing management of patients with spontaneous intracerebral haemorrhage
Jeanne Barr, Sydney, Australia.

Concussion: What's the State of Play for Children & Adolescents in 2017?
Vicki Evans, Sydney, Australia.

Functional Neurological Disorders
Vincent Cheah, Mikailah Nuske, Queensland, Australia.

Neurological Enablement and Advice Track (NEAT) Model - A Generic Community Neurological Nursing Service
Marilia Pereira, Kym Heine, Kathy McCoy, Western Australia.

Does Intrathecal Morphine make you Breathless?
Alison K. Magee, C.W. Huo, J.Wong, Y.Y.Wang, Melbourne, Australia.

Australia's first patient with Huntington's Disease treated with Deep Brain Stimulation surgery.
Emma J. Everingham, Sydney, Australia.

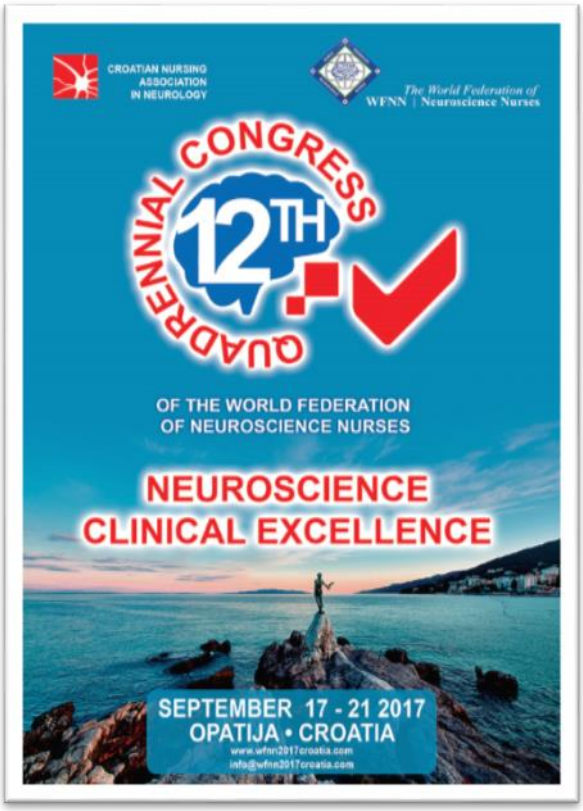
2017 AUSTRALIAN ABSTRACT TITLES (POSTERS)

Inoperable insular epilepsy: A world first – there is hope.
Erin Beard, Queensland, Australia.

Decompressive Craniectomy for Malignant MCA stroke: A Case Study
Sheila Jala, Queenie Leung, Sydney, Australia.

Cessation of routine CSF sampling: Is there an effect on infection rates?
Jane Raftesath, Marie Goodwin, Sydney, Australia.

Support my Spine ASAP! An Australian rural telehealth model of care for patients who have suffered a spinal fracture and need a TLSO
Sarah Zehnder, Ryan Gallagher, Michelle Giles, Jane Morison, Judith Henderson, Sydney, Australia.



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- **AANN Conference**
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  17—20 March
  Marriott Marquis
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2021:

- **WFNN Congress**
  Darwin Australia

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Australasian Neuroscience Nurses' Association

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Successful applicants presenting an oral paper **must** submit their written paper to be published in the *Australasian Journal of Neuroscience* as part of their award requirements. The successful applicants name will be forwarded to the Journal Editor for follow-up.

The Louie Blundell Prize

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

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

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

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

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

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