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Editor - Vicki Evans

This year sees our 40th Anniversary Meeting returning to Canberra to focus on the Evolution of Neuroscience Nursing—*Yesterday, Today & Tomorrow*. Whilst reviewing the past, where spinal surgery patients were nursed in bed for a week post-op on pillow-packs and sheepskins, we can form the future through nursing research and best practice, that now emphasises assessment, early mobilisation and infection control strategies.

Whilst there have been many changes in the Neuroscience nursing and medical field, the core values that were set at the start remain unchanged—to facilitate and foster the advancement of neuroscience nursing thereby enabling optimal patient-centred care. This was/is achieved by promoting neuroscience nursing and educating neuroscience nurses.

There are many articles of interest in this edition from a variety of settings and countries. The first article on *Humour as a Nursing Intervention* is from The Netherlands and emphasises that humour can be used to establish a good nurse:patient relationship. The Iranian article discusses whether EEG findings can predict stroke; CLIPPERS—a recently diagnosed inflammatory CNS encephalomyelitis (prominently involving the brainstem, especially the pons); *Difficult Decisions..* article from the UK discusses whether to tube feed neurologically impaired nursing home residents; the article on *Neuroscience Methods, Nursing and Patients* highlights an important fact that neuroscience nurses are closest to the patient and together with clinical research, can seek new and improved treatments for better outcomes. Then there is a systematic review of *Protein Requirements in TBI* and the final manuscript showcases the importance of quality nursing care in an unusual case study describing *Guillain Barre Syndrome in a 15month old*.

These manuscripts emphasise that neuroscience is always evolving and that you, as the neuroscience nurse, are at the forefront to advocate and make changes for the betterment of care.

Cheers, *Vicki*



Jenni Blundell

This year we celebrate forty years since a small group of nurses sat around a motel room in Canberra. The gathering had been called by Antonia Koenen, from Royal Prince Alfred Hospital, Sydney who had been accepted as one of the first Australian members of the World Federation of Neurosurgical Nurses (WFNN) at a meeting in Tokyo in 1973. Subsequently she had been elected to the Executive of the WFNN as the Australian Delegate and Editor of Publications. As a delegate for the WFNN, Antonia had the task of establishing a sustainable neurosurgical nurses association within four years. It was to this purpose that the group gathered in that motel room on a cold, wet evening on the 4<sup>th</sup> May 1974.

The group was comprised of nurses from all Australian States and Territories, except for South Australia and Queensland, all with one commonality—that they worked in neurosurgical wards or units and had been invited to attend the Annual Scientific meeting of the Neurosurgical Society of Australasia being held at that time. Although not all states, or New Zealand were able to be represented, the group voted unanimously to proceed with establishing an Australasian Neurosurgical Nursing Association (ANNA), forming a working committee and appointing state delegates. The aim of the Association was to promote an "exchange of knowledge and of nursing techniques in our field and promote interest in this area of nursing."

Within a year the membership grew, including nurses from New Zealand and South Australia, a Constitution formulated, and an insignia designed. Within three years ANNA was accepted as a member of the WFNN, and since then a number of members have followed in the footsteps of Miss Koenen by holding office on the World Federation Executive. In 1987, the membership voted to change the name of the association to better reflect the changing breadth of neuroscience nursing practice.

There was the establishment of standards of practice in neuroscience nursing. The original 'Brainstem' newsletter led to the publication of the first edition of the 'Australasian Journal of Neuroscience' in 1988. The two day ANNA Annual Scientific Meetings with presentations and posters from neuroscience nurses took some time to develop from the half-day session and AGM, held alongside attendance at the NSA meetings by members. These things that members take for granted today as part of a professional organisation, come about due to the sacrifice of time, effort and often their own financial resources of the members, especially those early executive members.

Over the last forty years, neuroscience nursing practice has changed due to an exponential growth in research based scientific knowledge and technological advances. While there are still people who suffer neuro-trauma, changes in surgical approaches, improved patient outcomes and healthcare trajectories, and increased knowledge about many acute and chronic neurological conditions have increased the scope of neuroscience nursing practice.

These advances have not just changed neuroscience nursing practice but also the patient experience from one that often involved multiple inpatient invasive tests to determine a diagnosis, lengthy hospital stays, and medical and nursing practices based on best opinion or limited understanding due to lack of research evidence. Today, nursing research has helped to improve the outcome for those people and their families to whom neuroscience nurses provide care. Examples include the development of patient centred clinical guidelines; nurse initiated patient and family education and /or support groups; community based care for people with chronic neurological conditions; and evidence that good nursing care can improve stroke outcomes.

Throughout these forty years of change it is the many nurses who have held membership in the association, contributed to the sharing of information and/or contributed to neuroscience nursing knowledge who have ensured that ANNA has remained relevant. ANNA may not be the biggest or flashiest group but it has met the brief of those of us who met that night in Canberra all those years ago.

~ Jennifer Blundell

## Book Review

### Neuroscience: A Historical Introduction

Mitchell Glickstein

ISBN; 9780262026802

This book reveals neuroscience knowledge from its sources in history, through neuro-anatomists descriptions, founders of disease and historical perspectives. It brings together historical fact with a modern day approach that simplifies through word and picture.

It begins with an overview of the nervous system from the structure and function of nerves, electrical and chemical transmissions, the senses, memory, language neurological diseases, higher centres, emotions and consciousness. Advances in technology and scientific discoveries are discussed. All are punctuated with descriptive diagrams and pictures which are extraordinarily clear and detailed.

Historical references are made that equate neurologists and neuroscientists with their nervous system discoveries eg: Carl Wernicke and Wernicke's Aphasia. This is particularly interesting for anyone interested in the history of neuroscience discovery.

The last chapter, "Are We There Yet?" sums up the book—we know a great deal about the functioning of the brain and spinal cord, yet much remains to be discovered. This book is a historical account of how we know things about neuroscience. The author sums it up by saying "Neuroscience is a product of individual humans no less than the painting of a picture or the writing of a moving aria. It differs from art in that, like other sciences, it must ultimately be based on evidence." (p. 349).

This is a must read for anyone interested in neuroscience.

Available from local bookshops or direct from Footprint Books at [www.footprint.com.au](http://www.footprint.com.au)

~ Reviewed by the Editor

## Humour as a Nursing Intervention.

Aschwin van Loon

### Abstract

Therapeutic humour is defined to be any intervention that promotes health and wellness by stimulating a playful discovery, expression or appreciation of the absurdity or incongruity of life's situations. This intervention may enhance health or be used as a complementary treatment of illness to facilitate healing or coping, whether physical, emotional, cognitive, social or spiritual.

Humour can be used in all kinds of situations, to relativize, make tense situations less tense or it can be used to make life more pleasant. If a nurse uses humour as an intervention in complementary care, certain patients may complain less. This paper will look at Humour as a nursing intervention. A review of the literature was done and after the review a questionnaire was undertaken.

**Key Words:** *humour, nursing intervention, cheerfulness, expressions.*

### Introduction

Humour can be used in lots of different situations, such as: to relativize, make a tense situation less tense or just to make life more fun and easier. If the nurse is happy and uses humour as a means of communication with patients, it may be noted that patients complain less about pain and/or pain sensation, that time is going slow, issues with nursing care. Victor Borge once said that humour is the shortest distance between two people. He even wrote a book with the same title (Borge, 2001).

Humour is a very large concept. For this reason it is necessary to reduce this to a specific subject. In the movie "Patch Adams" (Universal Studios, 1999), Robin Williams plays someone who lets himself be admitted to a psychiatric hospital. He sees a lot of misery around him and nothing of joy and decides to do something about it. He decides to study medicine to become a doctor. After his first year of medicine study he wants to be more involved with patients and embarks on ward rounds with the medical staff. On one of his rounds he comes in contact with children in a cancer ward. Everything is white, cold and looks sterile. All children lay neat and still in bed. He finds a klysma ball (rectal bulb)

and cuts the tip of and puts it on his nose. All the children start laughing and are having fun. The topic of humour is vast, therefore the subject will be reduced to something more manageable - humour with the patient.

In what instances do nurses use humour? Most nurses use humour to enable people to laugh, to ease a tense situation for example, with a patient going to surgery. Therefore, it can be said that nurses sometimes use humour as a nursing intervention.

### What is laughing?

Laughing according the Oxford dictionary is to make the spontaneous sounds and movements of the face and body that are the instinctive expressions of lively amusement and sometimes also of derision. It is to give an expression of a realisation of happiness. Laughing is also a physical reaction to emotions.

Laughing can also be described as a smile that is used by the entire body (Wooten, 1997). First the corners of the mouth will start to curl slowly, then the muscles around the eyes will participate and the eyes will twinkle. Next a noise, first a bit of growling, followed with spontaneous chuckle. This chuckle will start getting louder and will end in a roar of laughter. The chest and stomach muscles will be activated. Sometimes when the noise is getting louder the body will bend in a rocking motion, sometimes the person will smack their knees, stamp their feet or give the per-

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son next to them a poke in the ribs. When the laughter reaches its peak, tears will start rolling. All this will continue until the person laughing is so weak that they will have to sit down.

**Laughter:** There are several ways to describe laughter.

### Laughter Theories

There are several ways to describe laughter. The four most common are described below:

#### 1. The Superiority Theory

One of the oldest theories about laughter comes from Plato and is called the superiority theory (Brandon, 2005). Plato was convinced that laughing had much to do with the bad side of human kind - where the person laughs at the misfortunes of others, as outlined in Table 1. Laughing comes forth out of the ignorance about oneself (Brandon, 2005). The person laughing thinks himself richer, handsomer, more upright and wiser than he is in reality. The laughter is directed at the inadequacy of the other person. Brandon (2005) suggests that Aristotle agreed with Plato that the idea of laughing in essence is a form of mockery. However, in contrast to Plato he does not totally condemn a humoristic attitude. Certain measures of humour can make life less dull or boring. He went on to say that human-kind has to be careful that one doesn't laugh too much. Too much laughter can make one superficial when dealing with the more important matters of life (Morreall, 1983).

#### 2. The Incongruity Theory

*The Britannica (2002) states: '[humour is] the perceiving of a situation in two self-consistent, but mutually incompatible frames of reference or associative contexts.' Humour is 'a form of communication in which a complex mental stimulus illuminates or amuses, or elicits the reflex of laughter.'*

This definition of humour is described as the theory of incongruity. In his book *Taking Laughter Seriously*, Morreall (1983) explains the difference between the theory of superiority and the theory of incongruity. The first has to do with emotions or feelings and the second with the ability to learn. In the theory of incongruity, the 'amusement' is an intellectual reaction to something unexpected, illogical or improper behaviour. So in essence, the theory of incongruity has to do with the sudden opposed expectation when

- **The infecting laughter**

This exuberant laughter cannot be withstood by many people. Often one doesn't know why one is laughing.

- **The arrogant laughter**

A contemptuous smile.

- **The liberating laughter**

A relieving smile. For instance the result of a test is being announced and the grade is above average.

- **Compulsive laughter**

This laughter is not based on an emotion but of a pathological origin like a cerebral vascular accident or schizophrenia.

- **The mean false laughter**

An unreal laughter often used to hide emotions.

- **The giggles**

An ill-mannered, mocking and restrained laughter. Often the hand covers the mouth.

- **The screaming laughter**

Hard and very exuberant laughter which in some cases can deprive you of air.

- **The cordial laughter**

A sincere and convincing laughter and the eyes will participate. Often one says that you have twinkling eyes.

- **The laugh kick**

Unnatural laughter that is caused by using drugs like marijuana.

- **The burst of laughter**

A loudly, hard and exuberant laughter that involves real joy. The mouth is widely open when laughing.

**Table 1** (Above): *The Superiority Theory as explained by Brandon 2005.*

laughing is invoked. This can be explained with the following:

*A lady tells her next door neighbour her dream she had had the night before. She told that she was in heaven and saw three doors, two tiny ones and a big one in the middle. She opened one of the tiny doors and saw everyone at peace with one another. She closed that door and opened the other tiny door and there she saw angels playing beautiful music. The neighbour asked her: "Did you open the big door?" And she answered that she tried. She pushed and pushed against the door but she couldn't get the door to open. Just as she was about to give up, Peter came along and offered help. Together they pushed with great effort and then suddenly the door opened. She dropped into the room and there she was in front of God. The neighbour exclaimed "Good lord*



what did God say?" Well He smiled and said: "It jams, doesn't it?" (Zijderveld, 1971).

### 3. The Bergson theory

Another theory is described in the book "The Laughter" by French philosopher Henri Bergson (1993). According to Bergson, laughter has a social function. He says that humour is the caricature of the mechanism of the nature of humans (habits, automatic acts – used frequently by comics and clowns) and the continual creation of new forms. In this way laughter can be provoked. The following demonstrates this theory: When someone is running across the street and he suddenly trips and falls down, there is a good chance that many of the watching pedestrians will start laughing. They probably wouldn't have laughed if they knew beforehand that the person would have fall. We laugh because it is unexpected and involuntary.

### 4. The Jefferson theory

The theories discussed so far are all from the point of philosophy/psychology. Every theory tries to give a convincing answer to the question what humour really is. The theory by Gail Jefferson (1979), is contrary to this, this is because it only occupies itself with the question of how laughter interacts with conversations and what functions it might have. According to Jefferson, laughing is an activity whereby one participant invites the other participant(s) to laugh. This invitation can be accepted or declined by the other.

All these theories talk about 3 different things: first they talk about the form of the humour, second they talk about the message that is in the humour and third they talk about the effect that the humour has -

- **The form:** it's usually the contrast or difference between two aspects that on first sight can't interact with each other but both aspects are real.
- **The message:** it always is about people even when the message is about weird things or objects. The difference can be in many ways: what people think they are, what people think is real, what people think is (their) reality, what is the moral and the idealist reality, what kind of aspect one doesn't see or the real reality.
- **The effect:** recovery of ones own balance by accepting the unaccepted.

### What is humour?

Humour is a topic which has been discussed

often but little written. Humour is a complex phenomenon and it is an essential part of mankind. Through the ages, anthropologists have never been able to find a culture or civilization where there was no humour. A sense of humour is both a point of view about life and the behaviour that this point of view radiates.

There are many definitions. According to the Oxford Dictionary, humour is the quality of being amusing or comic, especially as expressed in literature or speech, a mood or state of mind. According to the Dutch dictionary humour is an eye and a sense of cheerfulness contradictions or cheerfulness contradictory expressions.

- According to Sigmund Freud (1905), humour is a way to relieve oneself of too much energy and tension, a rear and precious gift.
- According to Pasquali (1990) humour is communication, a means to strengthen mutual relationships.
- According to Léon van Woerden, a nurse educator who spoke at the "Caring & Humour Symposium" (2002) in The Netherlands, humour is the shortest social distance between two people.
- According to McCloskey & Bulechek (1998), the definition of humour is to help the patient recognise and appreciate funny, amusing or ridiculous situations so that relationships can be built, tensions released and one can easier cope with difficult situations.

Dr. Barry Sultanoff, former president of the American Holistic Medical Association says "Laughing can be a time of intimacy and communication, a time where we totally can be ourselves and one can come close to humanity and vulnerability. By laughing together one can recognize each other's unity and one can experience this unity. This can be the best way to show our ability to give healing energy" (Ochampaugh, 2009).

Junction Humour (Glissenaar, 2002), describes humour as "a short circuiting in one's brain that leads up to spastic movements", in other words, laughter. What is life without laughter? There are times when it is perceived that humour is only for happy situations. However, humour has a role when times get tough and can help manage and improve the situation. Humour is a way of

communication, a coping mechanism. It gives people the opportunity to build relationships. It helps to put things into the right perspective so that one can better cope with the difficult and tragic moments. (Wooten, 2002).

### What is a nursing intervention?

According to the Nijgh & Versluys (1997), a nursing intervention is every direct form of health care on behalf of a patient by a nurse. The nurse devises, plans and implements the interventions so that the objective is reached together with the patient. Implementation can be:

- **Using one's own intuition as a nurse**— The nurse uses his or her own intuition / feelings to decide what the best intervention is.
- **Using practical skills as a nurse.** Nursing is a practical occupation. It has been passed down from generation to generation.
- **Using your professional knowledge as a nurse.**
  - ⇒ **Generic knowledge:** the general knowledge that every nurse has to have no matter where they work.
  - ⇒ **Specific knowledge:** the knowledge that the nurse has when taking care of a specific group of patients and their specific problems.
- **Using the result of scientific nursing research:** The nursing science occupies itself with the question: "*What nursing intervention does really work?*"

By directing humour at a patient one uses one's own feelings and/or intuition to see if the patient is sensitive and receptive to humour. The question that has to be asked is if humoristic behaviour is part of the nursing attitude. It is not primarily part of one's nursing attitude this because one more often than not, has to be serious in dealing with the more serious matters of life. Humoristic behaviour can be a secondary part of professional attitude. There is a time and a place for both humouristic attitudes and serious attitudes. If only using humour, the risk is that no one takes you seriously anymore. Patients and family will start to address their questions to other colleagues.

### Styles and levels of humour

Humour is the lubricant of society. It knows many different shapes: parody, satire, jokes, pranks, funny stories, double-meanings, cartoons and weird situations. Humour can be

#### Upside down.

William Osler: "Ask not what kind of a disease someone has but what kind of a disease the person has."

#### Absurd humour.

Giving someone a note which says "To take away."

#### Double-meaning.

The waiter says to a customer: "Do you like to eat wild?". And the response of the customer: "No, I like to eat in peace."

#### Grim humour.

Not suited for someone who is going to be hung.

#### Sick humour.

"Can you remove this cosmetic flaw?"  
"Sorry, we don't decapitate."

#### A pun.

"Doctor, I'm having hallucinations."  
"No, you are just imagining them."

#### Inside jokes.

Doctor is being urgently called away during his consulting hours: "Don't get better, I'll be back soon."

#### Present humour.

Only by laughing one can become immune to Mad Cow Disease.

#### Mockery.

"It's been a long time since I've seen you", says the blind man against an old friend. <sup>(13)</sup>

#### A surprising turn.

"I bit my brother on his leg and then my tooth fell out of my mouth."

**Table 2:** (Above) Styles and levels of Humour, Truyen & Portael (1996).

distinguished in several styles and levels as outlined above in Table 2.

Humour is difficult to define. It is subjective. What one thinks is humourous, may not apply to another.

### The Essence of Humour

Humour comes with certain physical consequences. These can be distinguished by physical, social, psychological, emotional and cognitive behaviours.

#### Physical

Humour applies physical laughter. A well known saying is that laughing is healthy. This is true because laughing involves the whole body: muscles, respiration, cardiovascular system, hormonal system and the immune system (Truyen & Portael, 1996). Muscles are being put to use during a fit of laughter.

This is evident and can impact on the jaw leading to sore jaw muscles and also the bladder muscle. Respiration speeds up and gets deeper, especially exhalation. This induces better lung ventilation and a higher oxygen blood level. Due to the deeper respiration the diaphragm pushes down on the bowels. These in turn are massaged and gives better peristalsis.

When laughing the heart frequency increases as does the blood pressure, which can even temporarily double! Eventually the blood pressure will drop to below the starting pressure. This increased blood pressure stimulates the brain, especially the hypothalamus, affecting hormonal levels and releasing endorphins, which improves over-all well-being. This can last up to two hours after the induced burst of laughter (Truyen & Porteaal, 1996). It is pain reducing, increases libido and has the same effect as anti-depressive medications. During laughter, immunoglobulin A level rises improving immunity to diseases (Lefcourt, 1990).

### **Social**

Humour can make relationships easier. Humour can have a discharging effect on certain situations. It is often infectious and can give a feeling of unity. A group's bond can be enhanced by using humour and can also have an influence on certain members within a group. Humour can also reduce fearful emotions (Baks, 2002).

### **Psychological**

Humour and laughter determines how things are seen and reaction to things that are experienced. Herbert Lefcourt, (1986), a well-known psychologist of the University of Waterloo Canada, has researched how the feeling of humour and the use of humour can change emotional reaction to stress. In his research, people were asked to look back to the previous six months. They had to look how much and how often stressful situations were involved in their life. At the same time negative humour disturbances were evaluated. Lefcourt scored and evaluated the test on use of humour, sense of humour, assessment of humour and the possibility to use humour and laughter in the lifestyle of the test persons. Results of this research have shown that the sense and the assessment of humour can possibly be a buffer zone against humour disturbances which occur after a negative life experience (Lefcourt, 1986 & 1990).

### **Emotional**

Psychological humour has a great revitalising aspect. Humour can protect against extreme stressful situations. In other words, humour poses the strength to discover the comical side of a tragic situation. Humour can create an atmosphere of openness and freedom. It can also reduce anxiety because fear brings the body in a state of restless vigilance (Truyen & Porteaal, 1996).

### **Cognitive**

Humour develops around the age of three years. How exactly this happens, is unclear. There has been little written on this subject. During a 2002 symposium, the anthropologist Prof. van Hooff explained that humour exists with extremely young children (from three month old). This develops during the interaction between the parents and the child. If parents show the child humour, then the young child will mimic this behaviour. When the child grows older he will give his own interpretation to the concept of humour. Children learn by mimicking their parents/guardians. Babies and children use humour and laughter to reduce anxiety, anger or pain. Humour is subjected to age, education, interest & preference, geographical position and culture. In 2012 a study was done by Mireault, Sparrow, Poutre, Perdue and Macke, 'The laughing babies study'. It came to the same conclusion as Prof. Van Hooff in 2002.

### **Humour and the taboo**

When thinking of a hospital, most people have a certain image of serenity. Many people think for instance that when they are admitted to hospital, they have to lay down on a bed in their pyjamas. Many people also think that there is no place for humour in a hospital. It is not a joyous place (Glissenaar 2002). Jokes have a time and a place. Laughing should not occur when someone is ill or is in pain. There is almost a kind of taboo when it comes to humour and taking care of someone. Bogers (1997), pointed out that there was some resistance while organising a symposium and the use of humour. Prior to the symposium, a large group of nurses thought that it was unprofessional behaviour to use humour in hospital with patients. However, after the symposium a majority of this group were thinking differently. This was probably because there was a lot of publicity surrounding the potential benefits of laughter and making light of situations that are confronting. In 2002 Bogers organised another symposium on 'Humour & Taking care of someone'. At this symposium he concluded that humour

has already been more accepted than in 1997 but still had some way to go.

Nowadays many people think that there should be no taboo on humour in the hospital. Every person has the need to laugh and make fun, as outlined in Figure 1. At home, people laugh at humorous situations. So why is this frowned upon in hospital? Humour can be used in every situation – at birth and at death. Pick the time. One always has to take a look at the situation and if the person in question would benefit from humour.

#### The use of humour

Humour happens, often without conscious thought. There are times when humour needs to be a conscious thought. If you are

You may occasionally see us laughing, or even take note of some jest.  
 Know that we are giving your loved one our care at its very best.  
 There are times when the tension is highest.  
 There are times when our systems are stressed.  
 We've discovered humour as a factor in keeping our sanity blessed.  
 So, if you're a patient in waiting, or a relative, or a friend of one seeing,  
 Don't hold our smiling against us; it's the way we keep from screaming.

Figure 1: (Above): Poem by Wooten, 1998.

going to apply humour directly you have to consider a few points including:

- Is the patient receptive to humour?
- What kind of humour does the patient like?
- Determine how the patient usually reacts to humour (for example by smiling or by a burst of laughter).
- At what moment of the day is the patient the most receptive to humour?
- Don't make any jokes about sensitive subjects.
- Don't use humour around patients with a cognitive disability.
- Always pay attention to the reaction of the patient and if necessary stop the intervention.
- Always be on alert for the negative effect of humour for instance if someone doesn't like your kind of humour.
- Don't run on to the negative side of humour.
- Don't offend people by using your humour.

Humour can even be used in terminal care but this obviously requires a lot of sense and tact.

#### Humour, the practical side

The use of humour isn't for everyone. Humour is a quality one either has or has not – it is a quality that is hard to learn.

An example of humour in practice in The Netherlands, are the CliniClowns. The philosophy of the CliniClowns is relaxation, giving pleasure, make a positive contribution to overcome the difficult times during hospital admittance and make the stay in a hospital more bearable.

Everybody knows what a clown looks like - a red nose and a silly outfit, thereby creating a world with different standards and values. His reactions to this world are completely unjust, unnatural and unexpected. He is scared for certain things with no just cause. He cries when we think that he will start laughing and he is completely careless when it comes to danger. He is spontaneous and because the clown looks different to our world he often makes a fool of himself. The value of being neat and polite are completely the opposite in his world. The clown is part of a fantasy world. He's a magical figure with a freaky and eccentric appearance.

The clown makes sure that children during their hospital admittance forget their sorrows and let them be their normal healthy self. They laugh about the dumb, foolish helpless clown. Children step out of their role and help the clown thereby often feeling powerful and proud because they have helped someone. Adult patients also step out of their role if there is humour applied and can behave themselves as a normal healthy person (Truyen & Porteaal, 1996).

The use of humour during daily work gives people pleasure and energy. It reduces stress related to work and one can better cope with these situations. With humour people can put certain events in a better perspective. It is a coping mechanism. Humour can create a certain bond between people. In nursing such a bond is very pleasant if one can build it with a patient. This bond does not have to originate from the nurse but can originate from the patient.

Using humour on the ward requires certain pre-conditions. Such essential pre-conditions

can be:

- The whole nursing staff must apply humour together.
- Every member of the team has to decide for himself if he's going to use humour.
- Humour must not predominate.
- There has to be an open atmosphere on the ward.
- Collect and put together all humoristic situations on the ward so that you can tell them to the patients.

### Humour, the research.

This research was done using a questionnaire and was held on three different wards. The wards were Neurology, General Surgery and Internal Diseases of a general hospital in The Netherlands.

The 75 questionnaires were distributed and 24 were returned. Of these 24, two were discarded as they were not correctly filled in. The results are arranged per question and several pie charts were made. In these charts the results are given in actual numbers as in percentages. The shortening 'WN' stands for 'Don't know', 'Ja' stands for 'Yes' and 'Nee' stands for 'No'. Question 2 and 8 are open questions and all other questions are closed (Yes / No / Don't know) questions combined with opinion questions.

*Question 1: As a nurse it is unprofessional to laugh during my work about my work.*

Many nurses replied that they don't mind laughing during their work. Also many nurses said that laughing can put things into perspective and it can create a comfortable atmosphere where patients can relax better and the bond between nurse and patient can prevail.

*Answer:* Yes: 9% **No: 82%** Don't know: 9%

*Question 2: What is your definition of humour?*

This was an open question. Many different answers were given. The following answers are the most common answers:

- If I can laugh about it. This can be an experience of oneself, self-mockery or jokes from a patient.
- A joke with a revitalising action.
- A means of communication to create a bond between the patient and the nurse.
- Sociability, to be happy.

- To anticipate humour, but never at the expense of the patient.

*Question 3: Has, according to you, the implementation of humour an effect on the therapeutic environment?*

Many of the nurses agreed with this question because it often creates a distracting and relaxing atmosphere. Some nurses even wrote that it can reduce the pain and stress with patients. Some wrote that humour has a revitalising aspect to itself and it can create a bond between the patient and the nurse. Others wrote a critical note here that said implying humour consciously was not going to lead to success due to the fact that it then would be forced humour.

*Answer:* **Yes: 82%** No: 9% Don't know: 9%

*Question 4: I can laugh about myself and about some of the mistakes I made during my work.*

The majority agreed.

*Answer:* **Yes: 64%** No: 27% Don't know: 9%

*Question 5: Do you think it is possible to implement humour as a nursing intervention with a whole team on a ward?*

Many nurses think it is not possible because humour is so personal. It often is spontaneous and is always dependable of what kind of bond you have with the patient.

*Answer:* Yes: 27% **No: 64%** Don't know: 9%

*6: Humour and laughter are important and should therefore be admitted to the nursing plan.*

Many nurses think these are moments of emotion and feelings. It should all be spontaneous and should not be forced and should therefore not be admitted to the nursing plan. However, they think that they should always try to create a favourable environment.

*Answer:* Yes: 9% **No: 77%** Don't know: 14%

*Question 7: I worry that patients and family of the patients don't take my professional behaviour seriously if I act foolish and / or funny at my job.*

Here there are various opinions. One half of the nurses think it is improper, while the other half think one can behave foolishly and/or funny at work. This half also says that even whilst using humour as an intervention, you should also always show your professional behaviour as well, so that the patient and the family see that you can also be serious and professional.

*Answer:* Yes: 41% **No: 50%** Don't know: 9%

*Question 8: What is, according to you, humour as a nursing intervention?*

Many different answers were given. Below are the most common answers:

- Nothing, it should happen spontaneously.
- It is no nursing intervention.
- To anticipate on the humour of the patient.
- At certain times to make a joke or to do something funny and to step out of the role of nurse and to be for a short period of time a human being.

*Question 9: If I can laugh about my own problems this helps me to put this problem in the correct perspective and cope better with the daily stress of my job.*

The majority agreed with this statement.

**Answer: Yes: 68% No: 18% Don't know: 14%**

*Question 10: I share jokes and funny stories with my patients and their families.*

The majority agreed with this statement.

**Answer: Yes: 55% No: 27% Don't know: 18%**

*Question 11: Do you think that humour has an effect on the relationship between patient and nurse?*

Almost every nurse thinks that humour has a positive effect on the relationship between patient and nurse. The nurse learns more about the patient's behaviour. The patient is happy when he sees the nurse again because he knows he had and will be having a good time with that nurse as a nurse as well as an individual.

**Answer: Yes; 90% No: 5% Don't know: 5%**

*Question 12: When I'm stressed my sense of humour makes it possible to cope.*

According to many nurses humour helps as a coping mechanism in response to stress. Nurses who answered this question with 'No' could not give an argument why they thought this way.

**Answer: Yes: 63% No: 32% Don't know: 5%**

*Question 13: My executive manager encourages me to use humour at work.*

The majority thought this not to be true.

**Answer: Yes: 26% No:44% Don't know: 30%**

*Question 14: Together with my patients I try to laugh about their experiences.*

Many nurses try to laugh with their patients so that the patient can forget his sorrows and/or his disease for just a moment of time so his life is just for a brief moment pleasurable and uncomplicated. Others think that it is dependable on the situation and the experience of the patient.

**Answer: Yes: 68% No: 23% Don't know: 9%**

## Conclusion

The use of humour with a patient is a nursing intervention. It can be concluded that in theory and in practice, humour is an excellent nursing intervention and should be used as complementary care. In practice it is difficult to use humour as an intervention with every patient, because humour is an individual concept or emotion. One's humour, by definition, does not have to be another's humour.

If nurses are asked about humour as a nursing intervention, many will say that it is not attainable as a team but that the individual can use it for the total care of the patient. Humour, in its social context, improves the relationship between nurse and patient. Sometimes in using humour, the patient can better say what their feelings are so that the nurse can more fully understand.

Humour is not encouraged in some workplaces. Many aspects are responsible for this but one of the greatest aspects is that the executive manager of a ward is not receptive to humour. Another aspect can be that nurses think it inappropriate to use humour openly, laugh or behave silly when a patient or his family are present. They think it is unprofessional. Conversely, it can be seen that nurses are individuals and humour can be incorporated into patient care.

People usually define humour by jokes, pranks, happiness, laughing, funny stories, acting silly or foolish. However, there is more to it. Humour can be encouraged by dressing differently than other nurses. For example, wearing a red nose, like clowns or colourful socks or watches. This attire will brighten the room, maybe with a smile or funny verbal response. This is how you are spreading humour.

Using humour as a nursing intervention with a patient, some requirements need to be met such as:

- Check if a patient is receptive to humour and when they are the most receptive to humour.

- Check what kind of humour the patient likes.
- Determine how the patient usually reacts to humour (for example by smiling or laughter).
- Do not make any jokes about sensitive subjects.
- Encourage fun and playfulness.
- Always pay attention to the reaction of the patient and if you necessary stop the intervention.
- Do not use any humour if a patient has a cognitive disability.
- Humour should be used as a means to, and should not be used as the goal.

### Recommendations

Implementing humour as a nursing intervention on a ward, requires some changes to the basic concepts on the ward. The concepts are:

- The whole nursing staff must know that humour may be applied on the ward.
- Every member of the team must decide for themselves whether or not they are going to use humour.
- Humour must not predominate.
- Humour should be used as a means to, and should not be used as the goal.
- Do not use negative humour.
- There has to be an open atmosphere on the ward.
- Do not use racial or dark humour.
- Approach the patient from a holistic point of view.
- Explain the necessity of humour to nurses and patients.
- React positively to the attempt to spread humour.
- Humour should not be used to profile yourself as a nurse. The patient must always be the central person.

### Other humorous things:

- Collect and put together all humorous situations on the ward so that you can tell them to the patients.
- Select humorous materials for the patient, such as puzzles, games, cartoons, caricatures, videos or music
- Reduce environmental boundaries that are an obstacle for spontaneous humour.

- Draw a person's attention to the humour of certain situations or moments.

Considering these requirements, humour can be used as a nursing intervention keeping in mind however, that there needs to be a serious side as well.

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# The Short Term Outcome of Ischemic Stroke Patients: The role of Electroencephalographic Findings.

Alireza Saberi , Alia Saberi , SeyedAli Roudbary, Sara Savaheli

## Abstract

**Background:** Predicting the patients' functional outcome and mortality is difficult in the acute phase of stroke. Our aim was to determine the relationship between EEG findings and the short term outcome of ischemic stroke.

**Methods:** This cross sectional study was performed on the patients with ischemic stroke in the territory of middle cerebral artery (MCA) confirmed by CT scan in a neurology emergency in Guilan, Iran. The EEG is accomplished during 48-72 hours after stroke, and the patients were grouped in three according to its abnormality: normal EEG, moderate abnormal EEG and severe abnormal EEG. The patients' disability, functional independency, functional status and muscle force were assessed by Modified Rankin scale, FIM, Bethel index, and Medical Research Council (MRC) scale respectively and also their mortality rate in the first 48 hours, 1 and 3 weeks after stroke. The data were analyzed by Chi square, independent T, ANOVA, Repeated Measure ANOVA, post HOC Tukey, Kruskal-Wallis and Sphericity tests in SPSS20.

**Results:** A total of 47 patients (25 women, 22 men) with mean age of  $73.09 \pm 9.0$  years participated in this study. The 3 groups contained 15, 15 and 17 patients respectively, with any statistical difference concerning the age ( $p=0.44$ ) and sex ( $p=0.08$ ).

No statistically significant difference was found between groups in means and the change of means of Rankin score, FIM, Barthel Index, MRC, and the mortality during study period. ( $P>0.05$ )

**Conclusion:** EEG findings don't provide any prognostic value for the short term functional outcome and the mortality of ischemic stroke.

**Key Words:** *Electroencephalography, ischemic stroke, functional independency, functional state, muscle force, short term outcome.*

## Introduction

Cerebrovascular events are the most common neurologic disorders that are the 3<sup>rd</sup> cause of mortality and the most common cause of disability worldwide (Mack, Dusick, Martin & Gongalez, 2012). Despite the decrease in mortality rate related to cerebrovascular events in developed countries, it is still increasing in developing countries and even if the patients survive, their disability rate is high.

Ischemic stroke is the most prevalent stroke type and contributes to more than 70% of all strokes (Biller, Love & Schneck, 2012). Ischemic syndromes in some vascular territory are not only related to the territory of vessels which are occluded, but also to previous cerebral damage, collateral circulation and normal variation of brain circulation (Burst, 2005)

Cerebral infarct occurs when the cerebral blood flow disrupts which causes electrical suppression during 12-15 seconds and inhibition of cortical neurons synaptic excitability after 2-4 minutes and inhibition at the electrical excitability after 4-6 minutes.

The rate of cerebral blood flow is 50-55 cc/min for each 100 gr of brain weight. If it decreases below 50 cc/min, the electrical failure begins. When it reaches 18 cc/min, electrical failure stabilizes, and is not responsible for producing action potential. When it reaches below 18 cc/min, membrane failure starts and by decrements to 8cc/min, membrane failure stabilizes and cellular death occurs (Biller et al, 2012).

Cortical neurons produce electrical potentials and so electroencephalographic signals. Spontaneous EEG activity results from electrical flow in the extracellular space of cortical neurons that is in fact the summation of excitatory synaptic potentials. EEG potentials are not only the demonstrator of spontaneous activity of cortical neurons but also are re-

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lated to afferents from sub-cortical structures such as thalamus and brainstem reticular formation. EEG abnormality can be caused by cortical neurons' network dysfunction or changes of sub-cortical afferents function. EEG has a role in establishing the focal brain dysfunction in the absence of frank structural pathology and also in the presence of known pathology (Ronald & Timothy, 2012).

In acute stroke, the prediction of patient survival and functional outcome is difficult (Cillessen, van Hoffelen, Kappelle, Algra, & van Gijn, 1994). But to better inform and help the patient and their relatives and choose the best guideline for emergent intervention, it is essential to predict the patient's prognosis which is not possible by studying the patient's clinical status, according to a special para-clinic modality.

Multiple researchers have investigated the role of the evoked potentials in determination of stroke prognosis and their abnormalities (Zeman & Yiannikas, 1989; Ring, Bar & Aboud, 1999). Albeit, the results of all studies are not very reassuring and do not confirm the effective role of evoked potentials. Moreover, these methods are expensive and they aren't available in all medical centers.

Positron emission tomography (PET) detects the brain metabolism but its radiation is not safe to be used in everyone and this method is also expensive (Stoessl, 2012).

Electroencephalography has been used to record the cerebral electrical activity from 1924 and it is the first diagnostic device in neurology that helped with clinical assessment. Moreover, the result of electroencephalography is abnormal in almost all cerebral disorders (Saber, 2010; Alferove, Alekseeva & Goroshkova, 1994). Its abnormalities appear immediately after ischemic stroke, whenever brain computed tomography (CT) scan shows the lesion expansion after 1-3 days.

DWI-ADC map modality of MRI brain can show the abnormality in acute and hyper acute phase. But it does not exist in all medical centers and it also shows only structural abnormalities and cannot show or predict brain dysfunction. On the other hand, the other factors such as metabolic factors and previous cerebral dysfunction affect on present cerebral function this cannot be assessed by CT and MRI. If a device is able to show the brain function, it can also

consider these factors. Considering that EEG records cerebral electrical activity, and this activity is due to normal and abnormal function of neurons, so that can detect the normality of brain function partially and so the patient's function.

There are many controversies about the role of EEG in predicting the future outcome of ischemic stroke. In some studies, they show that EEG has a prognostic role and it shows the extent of brain dysfunction. Others claimed that the focal slowing of EEG activity is appropriate with clinical neurological defect at the time of EEG recording but for future prognosis, the focal and background EEG activity can't give any reliable information to predict the patient's functional outcome and survival (Bazil, Herman & Pedely, 2003; Kayser-Gatchalain & Neundorfer, 1980).

Some studies report that the contralateral background abnormalities are proportionate to level of consciousness and poor prognosis (Ahmed, 1988). Cillessen et al. (1994) support the predictive value of EEG in relation to the neurological deficit severity. Some of the previous work introduced only quantitative EEG (qEEG); which uses Wavelet and Fourier transforms in analysis of EEG (Finnigan, Walsh, Rose & Chalk, 2007) and serial EEG with determined interval for this purpose (Giaquinto, Cobianchi, Macera & Nolfe, 1994) and some others denied even the role of qEEG (De Weerd, Veldhuizen, Veering, Poortvliet & Jonkman, 1988). Neidermeyer (2005) suggested that there are few relationships between EEG findings, the disturbance of cerebral blood flow and the metabolism velocity in the early phase of stroke. Although, they also mentioned that preservation of the background activity is a good finding for prognosis.

In the present study, the relationship between EEG findings with present and future disability, functional status, muscle strength and mortality among ischemic stroke patients was investigated.

## Materials & methods

This cross-sectional study was conducted in a stroke center in Poursina Hospital, Guilan (north of Iran), in 2012. The subjects were ischemic stroke patients with involvement of middle cerebral artery (MCA) territory. They ranged in age from 50-90 years old. The project achieved the confirmation of Ethics Committee of Guilan University of Medical Sciences and all the data were confidential. Pa-

tients that reported or had documented evidence of previous stroke or seizure, pneumonia, pulmonary emboli, sepsis, metabolic disorders, Parkinsonism, dementia, or patients who received TPA in the present stroke, were excluded from this study, as they could affect the EEG recording.

For all patients, a brain CT scan was done in admission and the control brain Computed Tomography (CT) scan and/or Magnetic Resonance Imaging (MRI) was done after 48h for confirming the diagnosis. The treatment was identical for all patients. For each patient, information was gathered: including demographic data, past medical history, the calculated patients' disability, functional independency, functional status and muscle force that were assessed by Modified Rankin scale, FIM, Bethel index, and Medical Research Council (MRC) scale respectively and also their mortality rate in 48h, 1 week and 3 week after stroke. The EEG was recorded during 48-72h after stroke. All EEGs were interpreted by only one neurologist. The patients were grouped according to EEG findings as follows: group 1 with normal EEG, group 2 with mild abnormality such as background slowing or extreme unilateral or bilateral fast activity. Group 3 contains patients with severe abnormality including epileptic discharges or periodic generalized or focal slowing.

The sample size was determined as minimum 15 patients in each group according to 19 as reference number, with confidence of 90%, the test power of 90% and the probability of 15% dropout.

The data were analyzed by Chi square, independent T, ANOVA, Repeated Measure ANOVA, post HOC Tukey, Kruskal-Wallis and Sphericity tests, in SPSS software version 20.

**Results**

A total of 47 patients, 22 men and 25 women with mean age of 73.09 years (SD=9.01) were enrolled in the present study. Twenty-two had an infarct in left middle cerebral artery territory and 25 in the right side. The groups 1-3, had 15, 15 and 17 patients respectively with means age of 73.20 (SD=9.04), 70.47 (SD=10), and 75.20 (SD=7.84) years. There were no statistical differences between groups in terms of age (p=0.44) and sex (p=0.262).

There were no significant differences between three groups in modified Rankin score, FIM, Barthel index and MRC in first 48h, first week and three weeks after stroke (p> 0.05). The results showed that the patients' disability (MRS) and functional independency (FIM) improved in the study period, particularly in group 1, However, the results were not statistically significant in three groups with calculated P values of 0.178 and 0.19 for MRS and FIM respectively. (Fig.1, 2)

The changes of Barthel index mean were not statistically significant in the three groups. Also, the process of changes did not show any significant difference between three

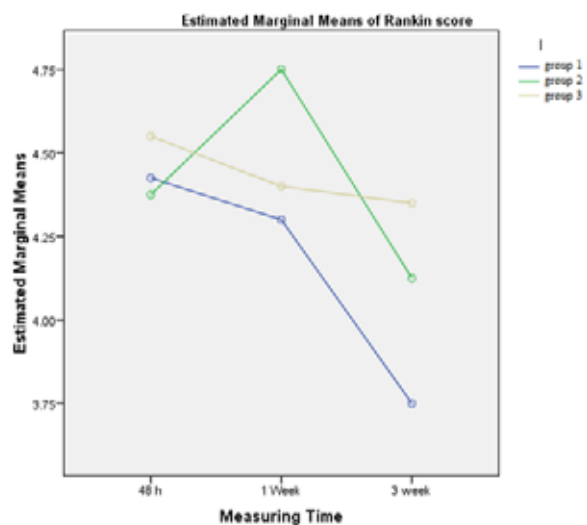


Figure 1 (Above): Estimated marginal means and change of Rankin score in 3 groups according to EEG findings.

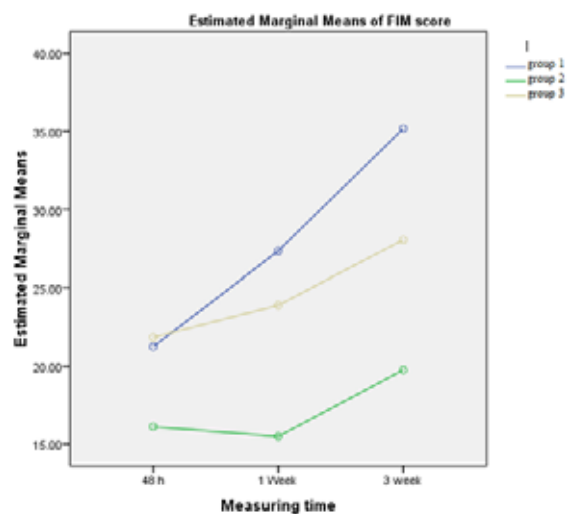


Figure 2 (Above): Estimated marginal means and change of FIM score in 3 groups according to EEG findings.

		Normal EEG	Mildly abnormal EEG	Severe abnormal EEG		
	N	Mean ±SD	N	Mean ±SD	N	Mean ±SD P
MRS change (48h-1 week)	15	0.07±0.59	15	-0.33±0.62	17	0.06±0.75 0.171
MRS change (48h-3 weeks)	15	0.00±1.00	15	-0.47±0.99	17	0.00±0.87 0.359
MRS change(1 week-3 weeks)	15	-0.07±0.96	15	-0.13±1.06	17	-0.06±0.75 0.993
FIM change(48h-1 week)	14	-3.71±11.78	14	0.62±1.66	16	-1.75±3.66 0.094
FIM change(48h-3 weeks)	9	-13.67±12.18	8	-3.63±5.04	15	-6.13±10.46 0.100
FIM change(1 week-3 weeks)	9	-8.22±7.87	8	-4.25±5.04	15	-4.40±9.66 0.392
Barthel change(48h-1 week)	14	-2.14±9.55	12	0.00±0.00	16	-1.56±5.07 0.474
Barthel change(48h-3 weeks)	9	-8.89±15.57	8	0.00±0.00	15	-3.00±9.02 0.195
Barthel change (1 week-3 weeks)	9	-5.56±7.68	8	0.00±0.00	15	-1.33±10.93 0.117
MRC change(48h-1 week)	14	0.14±0.92	13	0.42±0.49	16	0.13±0.47 0.154
MRC change (48h-3 weeks)	9	-0.39±0.85	9	0.03±0.79	15	-0.35±0.96 0.580

Table 1 (Above): Comparison the change of MRS, FIM, Barthel index, MRC in 3 groups according to EEG findings.

groups (P=0.294). Although our observation shows that Barthel index has not been changed in group 2 and 3, there was an improvement in group 1 (Fig.3). Our results showed that the MRC index improved in the three groups but this improvement process was identical in the three groups (P = 0.927) (Fig 4). Also according to Kruskal–Wallis test, the same results were achieved (Table 1). We analyzed all scores separately for men and women. The result showed significant difference between groups for FIM in the first 48h and 1 week after stroke in men, and only FIM 3 weeks after stroke in women (P = 0.039, 0.010 and 0.044 respectively).

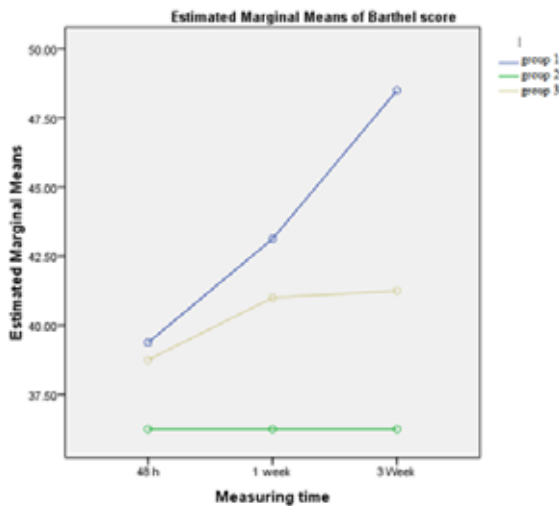


Figure 3 (Above): Estimated marginal means and change of Barthel Index in 3 groups according to EEG findings.

In mortality assessment, no deaths occurred in the first 48 hours. After 1 week, four patients including one patient in group 1, two patients of group 2 and one patient of group

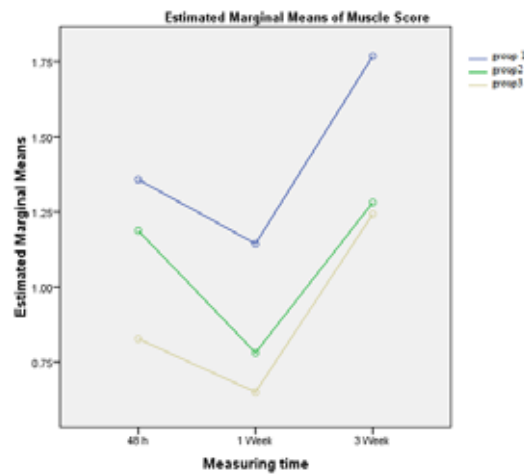


Figure 4 (Above): Estimated marginal means and change of MRC index in 3 groups according to EEG findings.

3 died. Moreover, after three weeks, six patients in group 1 and 2, and two patients in group 3, died. There were no significant differences between three groups. In addition, regarding the mortality rate, there was no significant difference between men and women in our three groups.

### Discussion

Several studies have been undertaken to determine the prognosis of ischemic stroke and a spectrum of clinical and para-clinical prognostic devices have been proposed for this purpose. As there is no strong relationship between clinical improvement and measurable changes in radiography, the simple radiologic methods such as CT scan and MRI are inappropriate for this purpose.

Electroencephalography is an easy, not expensive, almost available, and non-invasive

method that records brain electrical activity. However, the ability of this method to determine the stroke outcome is still controversial. The EEG findings depend on the size and the severity of lesion, its distance from the cortex and the structures involved. Superficial lesions produce focal and limited abnormality, whereas deep lesions cause unilateral and/or bilateral delta activity (Ronald & Timothy, 2012). Therefore, our results suggest that our subjects in group 2 with the background abnormality of EEG had deep involvement and cortical function was spared by collaterals and for the patients in group 3 with focal or periodic EEG changes, cortical injury is more probable.

In our study, we carried out an experiment on 47 ischemic MCA territory stroke patients to investigate the role of EEG in determination of prognosis of ischemic stroke. We reported no significant difference between our three groups in means and changes of disability (by MRS), functional independency (by motor FIM), functional status (by Barthel index) and muscle strength (by MRC) during study period. We also analyzed our findings for men and women separately and our results showed no significant differences.

Only when we investigate the numerical differences of each factor instead of their changes in three groups, a significant difference was reported only for men in the first 48 hours, and one week FIM. Also for women, we reported a significant difference in the first three weeks FIM.

One study determined that the amount of focal slowing of EEG activity during the first 48–72 hours after ischemic stroke, that is the specificity of our group 3, is related to patient's neurological deficit at the time of recording (Bazil et al. 2003). However, according to Kayser-Gatchalain & Neundorfer (1980), the background abnormalities that are observed in our group 2 are more valuable than focal slowing.

Iranmanesh (2008), explained that EEG abnormalities have a significant relationship with the patients' muscle strength at three months after ischemic stroke. Although we concluded that muscle strength improved during the time of study in all groups, this process was identical in the three groups. Therefore, it was not related to EEG abnormality. It seems that improvement continues several months after stroke and the partial perseveration of neural tracks near infarct or

posterior arm of internal capsule, result in future disabilities. Of course if the duration of study was longer, we may have observed significant change in the parameters we assessed.

Cillessen et al. (1994), performed a study in Austria on 55 patients. One year after stroke, they investigated the patients' prognosis. They concluded that acute phase EEG abnormalities have a prognostic value for functional outcome of patient with severe neurological deficit. In the case of moderate neurological deficit, no prognostic value was observed. Therefore, the prognostic value of EEG may be related to severity of neurological deficit. We cannot conclude this because we intended to investigate the short term outcome and on the other hand, in this study qualified Rankin scale was used (moderate: 1, 2, 3 and severe: 4.5 disability) and the difference between results may be due to these methodological differences. Also the results of a Belgian study by Sheorajpanday, Nagels, Weeren, van Putten & De Deyn (2011), suggested that EEG parameters were associated with patients' disability after six months of stroke assessed by MRS.

An Italian study by Giaquinto, Cobiانchi, Macera & Nolfe (1994), concluded that EEG finding has prognostic value in determination of six months Barthel index. In addition to the longer period of this study, multiple EEG(s) were recorded from patients that increase the specificity and sensitivity of test; of course the author said that although EEG is noninvasive method for predicting the patients' outcome but isn't used for routine evaluation, because motor index is a more accurate and faster measure (Faught, 1993).

In our study, we assess the functional independency of patients by FIM scale. We observe that there is no significant difference in FIM scale with EEG variation. However, when we look at men and women in two separate groups, we observe some difference in FIM scale with EEG variation. But because of the small sample size in these groups, our result is not reliable.

In Kitamura, Iwai, Tsumura, Ono & Terashi (1998), almost all patients with normal EEG had improved, Fifteen percent of those with mild to moderate abnormality with asymmetric, focal changes or slow activity in one hemisphere, had sufficient functional independency and those with severe abnormal EEG had poor outcome. This study of EEG in

stroke reflected the functional independence in stroke patients, although no special measure was used for independence assessment such as in our investigation.

De Weerd et al. (1988), performed a study over three years in which the change of clinical status, qEEG and cerebral blood flow (CBF) with xenon inhalation were monitored. The patients were assessed on admission, at months 3 and 36. In almost all patients, the EEG and clinical examination showed improvement with most of the changes occurring in the first 3 months. They concluded that although serial qEEG is valuable in monitoring of patient, it did not have prognostic value, albeit they showed low primary CBF can predict permanent neurological deficit. It seems that the results of this study are in agreement with ours. They both showed that EEG at each interval is correlated to clinical status but is not predictive. Kayser-Gatchalain & Neundorfer (1980) also concluded that EEG is not predictive. Another study emphasized on the value of qEEG in predicting the outcome of stroke (Finnegan et al. 2007).

## Conclusion

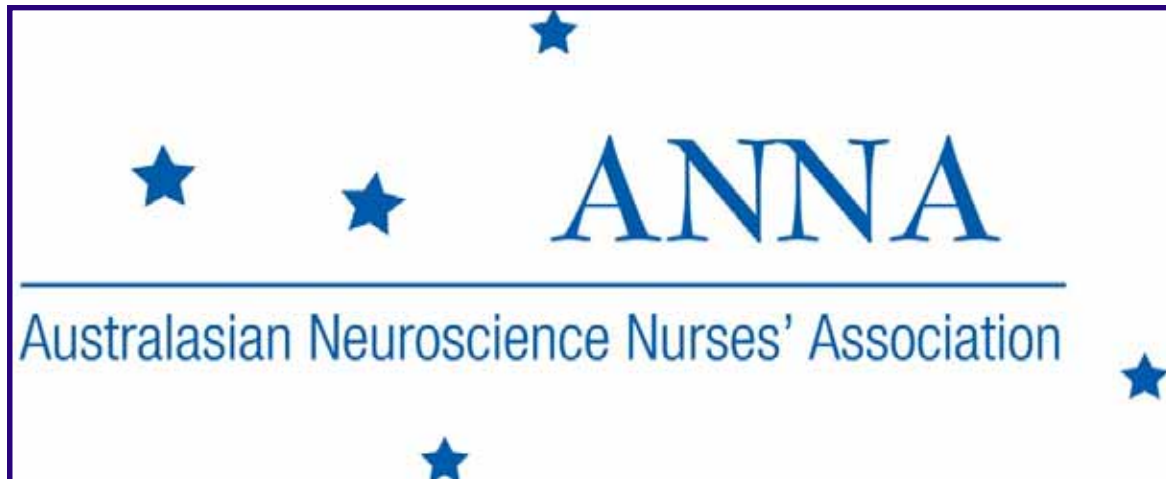
EEG findings cannot determine the short term functional outcome and mortality of ischemic stroke patients. It is suggested to design the future studies with longer duration, and especially compare EEG predictive value with NIHSS.

In our study, there was no significant difference in mortality rate between the 3 groups. Perhaps this limitation was due to the delay in referral of the patient after stroke and the lack of a follow-up system to record rehabilitation progress or mortality. Dogan, Tunc, Ozturk, Kerman & Akhan (2004) suggested that EEG findings can show future mortality of ischemic stroke patients. Perhaps if we elongated the duration of the project and assessed the total mortality rate and long term outcome, our results might be similar to that study.

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### The Louie Blundell Prize

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

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

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

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

For further details and criteria guidelines please visit the ANNA website at [www.anna.asn.au](http://www.anna.asn.au)

# A Case Study: Chronic Lymphocytic Inflammation with Pontine Perivascular Enhancement Responsive to Steroids (CLIPPERS)

Milan Dolezal

## Abstract

Many neurological conditions present diagnostic challenges. Chronic Lymphocytic Inflammation with Pontine Perivascular Enhancement Responsive to Steroids - (CLIPPERS) is a recently described syndrome with a potentially wide list of differential diagnoses. As a result patients might be unnecessarily exposed to excessive investigations, many of which pose significant risk to patient's health. The patient in the following case presented with non-specific neurological deficits and was eventually diagnosed with CLIPPERS. The case illustrates the most common clinical presentation and typical findings on MRI. Improved understanding of this syndrome will increase the likelihood of a timely diagnosis and an initiation of an appropriate treatment plan.

**Key Words:** CLIPPERS, inflammatory neurological disease, central nervous system, demyelination.

## Introduction

There are many inflammatory diseases of the Central Nervous System (CNS) where pathological aetiology is not fully understood. Large numbers of traditional and novel investigations have been employed to identify the underlying pathology and assist with selecting the most appropriate therapy. Ongoing research and the use of the latest case reports enable accurate diagnosis of the increasing number of conditions and successfully treat them.

CLIPPERS is a recently diagnosed inflammatory CNS encephalomyelitis, prominently involving the brainstem and in particular the pons (Keegan & Pittock, 2012). Patients with CLIPPERS often present with gait ataxia, diplopia, pseudobulbar affect, facial paraesthesia and/or dysarthria. Other less common presentations include tinnitus, tremor and vertigo. Due to the non-specific presentations patients usually undergo extensive pathological and radiological workup. However, it is becoming increasingly recognised that certain presentations with the typical radiological findings can lead to the diagnosis of CLIPPERS and efficient treatment can be commenced (Taieb, Duflos, Renard, Audoin, Kaphan, Pelletier, Limousin, Tranchant,

Kremer, De Seze, Lefaucheur, Maltete, Brasat, Clanet, Desbordes, Thouvenot, Magy, Vincent, Faillie, De Champfleury, Castelnovo, Eimer, Branger, UroCoste & Labauge, 2012).

## Case Study

Mr MA is a 58 year old male who presented with a 2 month history of dysarthria, ataxia and right hand paraesthesia. His past medical history includes hypertension, dyslipidaemia and peripheral vascular disease. He stopped smoking 20 years earlier and also has a history of alcohol abuse. His daily medications were Amlodipine, Simvastatin, Clopidogrel and Aspirin. There is no family history of inflammatory diseases of the CNS or autoimmune disorders.

Mr MA's initial MRI in April 2008 showed small enhancing lesions in the right cerebellar hemisphere, brainstem and left internal capsule (Figure 1). His follow-up MRI at 3 months did not show any disease progression.

The main differentials included demyelinating disorders i.e. Acute Disseminated Encephalomyelitis (ADEM), metastatic disease and vasculitis. Mr MA underwent a magnetic resonance angiogram (MRA), CT Chest/Abdomen, trans-oesophageal echo and a detailed blood analysis which did not show any abnormalities except for an increased ratio of T-cell markers - CD4 to CD8 cells and mildly elevated Alpha-2 and beta globulins (Figures 2 and 3).

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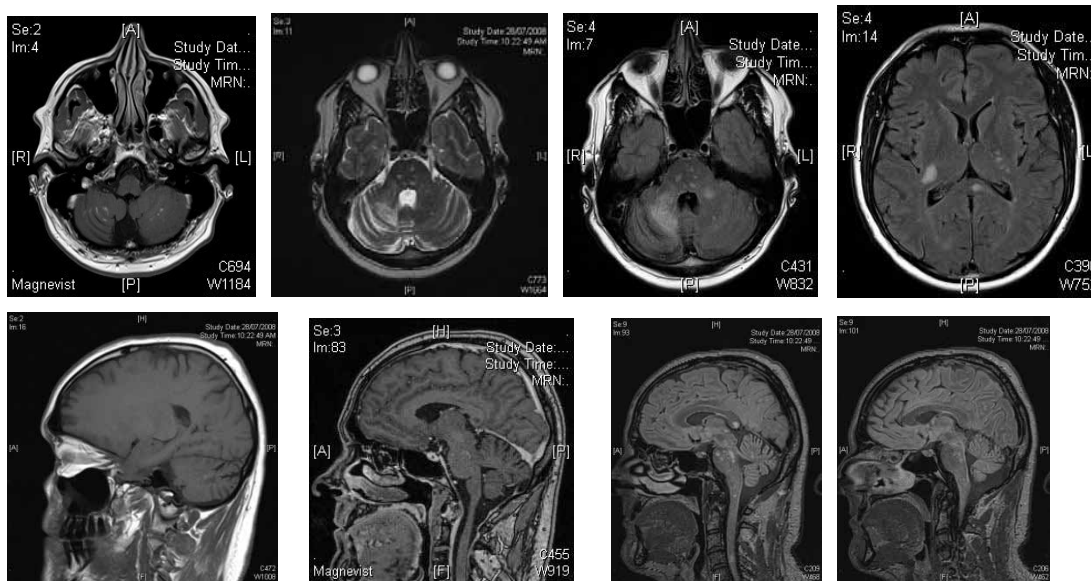


Figure 1 (Above): MRI showing multiple enhancing lesions in cerebellum, brainstem, internal capsule and cervical cord.

TCM			
	Units	Ref Interval	
White Blood Cells	4000	x10E6/L	(4000 - 11000)
Lymphocytes	1100	x10E6/L	(1000 - 4000)
<b>T-CELLS</b>			
CD3 %	86 H	%	(55 - 82)
CD3 No.	946	x10E6/L	(620 - 2200)
CD4 %	75 H	%	(29 - 58)
CD4 No.	825	x10E6/L	(420 - 1410)
CD8 %	11 L	%	(19 - 43)
CD8 No.	121 L	x10E6/L	(220 - 980)
CD4/CD8 Ratio	6.82 H		(0.70 - 2.70)

Figure 2 (Left): T cell markers in peripheral blood.

Immunochemistry & Electrophoresis			
Collected Time	04-Mar-08	04-Aug-08	
Time	07:50	11:38	
PaLMS Lab No	68748911	72207993	
Specimen	Blood	Blood	
External Ref	08-00640112	082170530	
<b>Immunochemistry</b>			
Anti-CCP Ab's	0.5		U/mL (< 7.0)
<b>Electrophoresis</b>			
Total Protein		77	g/L (58 - 78)
Albumin		42.6	g/L (33.0 - 46.0)
Alpha-1 globulins		3.0	g/L (1.5 - 3.0)
Alpha-2 globulins		7.0 H	g/L (3.5 - 6.9)
Beta globulins		13.2 H	g/L (6.0 - 11.6)
Gamma globulins		11.2	g/L (5.6 - 14.1)

Figure 3 (Below): Immunochemistry and Electrophoresis

As Mr MA also reported a history of recurrent conjunctivitis. Behcet's syndrome was considered, however it seemed unlikely as there was no history of previous mouth or genital ulcers and Mr MA's eye exam showed no evidence of inflammation.

Mr MA was given a 10 day course of methylprednisolone. His progress MRI 4 weeks after receiving the methylprednisolone showed marked improvement as well as the

patient reporting a clinical improvement in his symptoms, especially his gait.

At the 2 month follow-up Mr MA had experienced a relapse of his symptoms and a further MRI demonstrated an increase in size of the previously detected lesions as well as several new lesions in the corpus callosum. A lumbar puncture was conducted on Mr MA which showed a slight elevation in the lymphocyte count, however neither electrophore-

sis nor multimodal evoked potentials demonstrated any abnormalities.

Clinically Mr MA experienced further deterioration of ongoing deficits. He developed horizontal eye movement disturbances and weakness in his right arm, predominantly distally. Primary demyelinating disorders were considered including atypical Multiple Sclerosis (MS) and Behcet's syndrome among other differential diagnoses and were investigated with a whole body PET scan. As this did not demonstrate any abnormality, a brain biopsy was performed which showed dramatic infiltration of CD4 and some B cells in the cerebellum. There were no signs of lymphoma, vasculitis or MS.

Based on Mr MA's history and biopsy results, he was started on cyclophosphamide (cytotoxic immunosuppressant) for 4 months which resulted in significant improvements in both MRI and clinical symptoms. The regimen was later changed to a combination of Imuran and Prednisone. Prednisone was gradually weaned from 20mg to 2.5mg and Imuran was increased from 50mg to 125mg over a 2 year period.

Mr MA experienced gradual improvement in all his symptoms whilst on Imuran and Prednisone. In mid-2011, he achieved clinical remission thus Imuran was ceased.

Unfortunately, Mr MA represented in early 2013 to Gosford Hospital with an acute relapse of ataxia, dysarthria and right arm weakness.

In 2010, a study was published in *Brain* that described a newly recognised syndrome (Pittock et al. 2010). It was named CLIPPERS by the authors. Mr MA exhibits the phenotype of this newly diagnosed disorder.

## Discussion

In medicine and in particular, Neurology, there are many conditions with overlapping clinical symptoms that present diagnostic challenges. By following a well-structured line of investigation in cooperation with other specialists and excluding the most serious diagnoses, the appropriate and, in most cases, effective therapy can be established.

Mr MA's case illustrated the importance of remaining up to date with the latest research and case reports in formulating both differential diagnoses and therapeutic approach.

Based on up-to-date publications, a typical presentation coupled with positive radiological and serological findings is a sufficient foundation for starting therapy (Keegan & Pittock, 2012). Brain biopsies can be avoided if there is a clinical response to treatment confirmed on imaging. This is very important considering that in CLIPPERS the pathological changes are usually located in deep brain structures such as the brainstem and cerebellar peduncles of which biopsy carries a high risk of complications.

Due to the progressive nature of CLIPPERS timely initiation of treatment is very important. Simon et al. (2012), reported that pontocerebellar atrophy occurred early in their patients, including those whose therapy was started promptly, and that a delay in the commencement of therapy can lead to significant cognitive impairment (Simon, et al 2012). Early treatment of both the original presenting complaints as well as relapses could reduce the level of disability. Authors of this study also suggest that the name should be amended to Chronic Lymphocytic Inflammation with Pontocerebellar Perivascular Enhancement Responsive to Steroids to more accurately define the latest radiological findings (Simon et al. 2012).

Even with high suspicion of CLIPPERS, other diseases that present with similar clinical features and lesions should be excluded before a formal diagnosis is made. Such diseases include neuromyelitis optica, central demyelinating disorder, neurosarcoidosis, Behcet's disease, lymphoma, central nervous system vasculitis and malignant melanoma. It was also suggested that there could be a link between CLIPPERS and allergic reactions such as atopic dermatitis (Tohge, et al. 2012). This possibility was raised due to increased IgE and T-lymphocytic infiltrate within the perivascular spaces in three of the known cases (Tohge et al. 2012).

## Conclusion

There are still many unanswered questions regarding the pathological processes, clinical presentations, long term prognosis and the management of patients diagnosed with CLIPPERS. However, our understanding deepens with each published study and the long term follow ups of the previously known and the newly diagnosed cases; all of which will provide the pivotal information needed for the improvement of the overall management of CLIPPERS in the future.

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# Difficult Decisions: Tube Feeding for Neurologically Impaired Nursing Home Residents.

Steve Smith

## Abstract

Nursing home residents with degenerative neurological disorders such as Parkinson's disease, motor neuron disease, multiple sclerosis and dementia can be expected to experience problems with eating and drinking as a result of impaired swallowing in the advanced stages of disease. Despite this being an almost inevitable development, the response of health professionals when it occurs is often unplanned, unsupported by current evidence and out of step with the expressed wishes of patients. Nursing home staff and residents' families tend to find the process of deliberating over whether or not tube feeding should be implemented difficult and divisive. Often feeding via a percutaneous gastrostomy tube is implemented despite widely held misgivings. Documenting residents' preferences (often not to be tube fed) in advance is no guarantee of an outcome in favour of the choice made. Consequently patients may endure discomfort and distress over a costly and unwanted intervention that may fail to bring any benefit.

This article considers the extent to which nursing home residents with advanced neurological disorders are tube fed and whether it is unnecessary and unjustified as is often reported. The aim is to contribute to discussion on how best to improve patient-centred care for nursing home residents with advanced neurological conditions who face difficulties with eating and drinking.

**Key Words:** *Tube-feeding, gastrostomy, nasogastric, neurological, swallowing, nursing home.*

## Introduction

Increasing difficulty with eating and drinking due to impaired swallowing is commonly associated with advanced cognitive decline in people with progressive neurological disorders including Parkinson's disease, multiple sclerosis and motor neuron disease, stroke and advanced dementia (Altman et al 2013; Rosenbek and Troche 2013). Whether initiating tube feeding is an appropriate response for a particular nursing home resident in such circumstances has long been acknowledged as an enormously challenging dilemma for those faced with the decision (Gillick 2000; Gjerberg et al 2010).

### **What is the safest and most effective method of tube feeding for eating difficulties in long-term conditions such as neurological disorders?**

Percutaneous Endoscopic Gastrostomy (PEG) 'tube' feeding is widely used in adults

with dysphagia resulting from long-term conditions such as stroke and dementia (Kuo et al 2009). It is generally held to be a safe and effective intervention and suitable as a standard approach to long term enteral feeding (Kuo et al 2009). PEG feeding for people with neurological dysphagia has been reported to have important advantages over nasogastric (NG) tube feeding, which was considered more suited to short term nutritional support (Park et al 1992). Since then Dennis et al (2005) found NG feeding preferable to PEG feeding in the first few weeks of enteral feeding among stroke patients, based on their finding that NG feeding had a lower association with death or poor outcomes. They suggested that long term PEG feeding might negatively impact on dependency as more of the PEG group in their trial remained tube-fed at six months follow-up compared to the NG group. But a more recent systematic review (Gomes et al 2012) combining results from nine randomised controlled studies comparing PEG with NG tube feeding (including Dennis et al 2005) found no significant difference in complications between the two groups, but significantly less intervention failure in the PEG group. The authors concluded that PEG feeding is more effective and safe

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compared to NG with no difference between groups regarding mortality or pneumonia development regardless of underlying diagnosis.

Chen et al. (2013) add support to this view that PEG (or occasionally jejunostomy) feeding is the most suitable approach to long-term enteral feeding. NG tube feeding tends to be poorly tolerated and nasojejun tube feeding is better tolerated but blocks easily and both present problems with maintaining correct positioning which does according to Chen et al. (2013) increase the risk of aspiration pneumonia.

### **What is current practice regarding tube feeding in neurological patients in nursing homes?**

Neurological problems were found to be the most common reasons for PEG feeding in elderly people (40.7% following stroke, 34.7% due to degenerative neurological conditions compared to 13.3% associated with cancer) in an American study carried out in the community (Callahan et al. 2000). In an Australian study of 168 patients a recent stroke accounted for 58% and other neurological impairment for 15% of reasons for PEG feeding (Nicholson et al. 2000).

In a large U.S. cross-sectional study of over 186,000 nursing home residents with substantial cognitive impairment over one third were tube-fed and the extent to which this might be considered an appropriate intervention and factors that may have influenced the implementation remain uncertain (Mitchell et al 2003). There seems to be a lack of more up to date evidence on which to base a judgement of how common tube feeding among people with neurological disorders might be and how often this occurs among nursing home residents and the figure has been relied on more recently (Palecek et al. 2010). Certainly the sight of people with degenerative neurological disorders including forms of dementia being tube fed in nursing homes is not uncommon. This article considers whether tube feeding is effective for people with advanced neurological disease; whether the intervention is often implemented inappropriately and how nursing home staff should support individuals, family members and each other in decision-making with regard to tube feeding.

In the context of advanced dementia, Kuo et al. (2009) report a lack of knowledge about the circumstances that result in decisions to initiate tube feeding or the implementation of

this intervention post insertion. Mitchell et al. (2003) found that being younger, male, non-white and divorced, having a recent functional cognitive decline in the absence of confirmed Alzheimer's disease and not having an advanced directive were factors associated with greater likelihood of tube feeding being implemented. The study also considered whether specific features of care institutions are associated with greater likelihood of tube feeding for residents and having controlled for the above patient characteristics found that this occurred more frequently in homes set within an urban setting, accommodating predominantly non-white residents, having more than 100 beds, and not having a specialised dementia care unit.

### **Is tube feeding implemented unnecessarily?**

According to Mitchell et al. (2009) a third of nursing home residents with cognitive deterioration are tube fed 'despite a lack of evidence' in support of effectiveness of the intervention. In a study that followed 323 residents with dementia living in 22 nursing homes Mitchell et al. (2009) reported that approximately 86% of those studied experienced eating problems among other complications including pneumonia and febrile episodes over a period of 18 months. They found that interventions they described as 'burdensome' including hospital admission, emergency room visit and tube feeding occurred among 40% of the residents in the last few months of life (Mitchell et al. 2009). They concluded that there was much less likelihood of resorting to such interventions among residents whose family caregivers demonstrated an understanding of symptoms that can be expected to occur in late stage dementia, and generally the approach to care for these clients was less aggressive. But can this intervention be effective and appropriate for neurologically impaired nursing home residents even though this does seem to imply that some are tube fed unnecessarily?

### **Is tube feeding effective?**

Most people with amyotrophic lateral sclerosis (ALS) (a form of motor neuron disease in which both upper and lower motor neurons are impaired) experience dysphagia at some point in the disease trajectory and for between a quarter to one third of patients problems with chewing and swallowing are among presenting symptoms (Beghi, 2008). This often leads to the suggestion of enteral feeding aimed at improving nutritional status

for people with ALS but evidence that tube feeding improves survival, nutritional status or quality of life for this group is limited (Katzburg & Benetar, 2011). From their systematic review Katzburg & Benetar (2011), examined 11 controlled studies comparing survival, nutrition and quality of outcomes for 772 people with ALS fed via PEG with those for 2664 controls who continued oral feeding. The review found weak support for increased survival among the tube-fed participants but none of these studies were randomised. Similarly, evidence for improved nutrition among the tube-fed group was weakly positive. Given the possibility of a range of potential confounding factors the authors identified the need for a large RCT in order to claim improved survival and / or improved nutrition associated with PEG feeding with any confidence.

Katzburg & Benetar (2011), were unable to draw any conclusion regarding the optimum point in the disease trajectory to undergo tube insertion. There was no evidence in support of a positive impact on quality of life for caregivers or patients with ALS, and the authors point out that this aspect is widely regarded as the most important factor to be considered when weighing up whether a patient is better to continue with oral feeding or undergo PEG insertion.

Whether or not interventions such as modified food consistency and PEG feeding aimed at increasing nutritional and fluid intake following recent stroke speed recovery is unclear and effectiveness of tube feeding longer term post stroke is even more difficult to determine with any confidence (Geeganage et al. 2012). Evidence from five studies suggest PEG feeding enhanced nutrition in patients and food delivery in longer term was more effective via PEG than via a nasogastric tube but there is a lack of sound support for assumptions that tube feeding following stroke is associated with longer survival, reduced disability or improved quality of life (Geeganage et al. 2012).

Reid & Pantilat (2013), advise that PEG feeding offers people with eating and swallowing difficulties arising from advanced disease including cancer and neurodegenerative conditions no advantage in terms of longer survival, improvement in function or quality of life, the prevention or healing of pressure ulcers or prevention of aspiration pneumonia, and this seems representative of the view generally expressed in the body

of relevant literature.

#### **What is the cost?**

In a study carried out over 10 years to 2009, of over 4000 nursing home residents with advanced dementia the annual cost of care for those fed via PEG for one year post insertion was compared with non-tube fed residents matched for other variables (Hwang et al. 2013). The authors conclude that tube-fed resident health care costs were a little over \$2000 per year higher than controls. They refer to the difference as small but significant and suggest that this is difficult to justify given that best evidence fails to support benefits of tube feeding for this group in terms of health and wellbeing, quality of life or survival.

From their analysis of over 280,000 acute hospital admissions among over 160,000 nursing home residents with advanced cognitive impairment, Teno et al. (2010) supported previous findings that decisions to implement tube feeding generally do not appear to be made on the basis of considered thinking about the best interests and wishes of patients, family views, or those of staff who have come to know the resident over years. The insertion decision is often made rapidly in an acute context in response to crisis such as an infection. The findings are in line with Mitchell et al. (2003) that the absence of advanced directives- and with Mitchell et al. (2009) that understanding of the likely symptoms associated with neurological deterioration – are associated with less likelihood of tube insertion hastily undertaken in response to an acute event.

#### **If evidence doesn't support it, why are nursing home residents tube fed?**

Based on a study of costs and billing to Medicare for 11 patients with dementia who were tube fed compared to 11 who were fed with conventional assistance, Mitchell et al. (2003) proposed that in the United States nursing homes are presented with a financial incentive to favour tube feeding. The tube fed residents required less expensive care from the homes but attracted higher funding. The study was not able to establish whether this incentive had influenced decisions in practice about whether to implement tube feeding. Whether or not the study might have wider international implications cannot be determined but may be a factor worth considering in any local investigation of the rationale behind decisions about tube feeding.

Teno et al. (2010) acknowledge that it is one thing to know that tube feeding is often implemented inappropriately and to understand factors associated with decisions that are widely held to be undesirable, ineffective, costly and against the best interests of patients. However, they suggest that beginning to comprehend how to change practice in the light of this knowledge is a separate matter. Given that it has been reported for many years that nursing home residents with advanced progressive neurological disease would tend to prefer to avoid living longer due to artificial feeding even if it were shown to be effective (O'Brien et al. 1997) it is difficult to appreciate why those in charge of providing care for this patient group seemingly fail to take control of events to ensure residents are not subjected to unwanted invasive procedures with long term consequences. Teno et al. (2010) report a lack of evidence in this area and that family members and health professionals 'grapple' with the issue of what to do in difficult emotionally charged circumstances.

#### **How can decisions about tube feeding be reached?**

Beauchamp & Childress (2001), offer a four-principle framework to facilitate ethical decision-making. To apply the concept to deciding whether tube feeding might be appropriate for a nursing home resident with advanced neurological disease a first step might be to assemble a group including the patient, close family members, nurses and health care assistants closely involved in the person's care, a speech and language therapist, and possibly a clinical psychologist and psychiatrist. If this meeting takes place early enough in the disease trajectory to enable the individual to express his or her wishes clearly the task of the group should be easy, But often this is not the case, and the group has the job of trying to agree what the person would choose if he or she had been able to state personal preferences. The principles to consider are 'beneficence, non-maleficence, justice and autonomy.

Beneficence is to do with acting in the resident's best interests. The group could discuss first whether a decision to implement tube feeding would be likely to benefit the resident. The lack of hard evidence in favour of this can be acknowledged.

Non-maleficence is the principle concerned with ensuring actions or omissions do no harm to the resident. The potential for harm

resulting from inserting a tube weighed against possible harm from inaction can be discussed. Although serious acute harm associated with PEG feeding rarely occurs, long term complications are common (Cervo et al. 2006). Lack of nutritional intake, weight loss and eventual death might be seen as the inevitable consequence of the diagnosis and not a condition brought about through opting against implementing an unnatural feeding method.

With regard to the principle of justice, the group might discuss legal and moral considerations such as duty of care, patient rights to refuse treatments, health professional accountability to act in the best interest of patients and to use resources responsibly, avoiding futile interventions assessed as clinically unsound and with costs (financial and human) outweighing likely benefits. The principle of autonomy should be considered. With the caveat that patients cannot choose to insist on receiving treatments that professionals judge to be inappropriate, patient choice is generally accepted as taking precedence over other considerations. The reason for leaving this aspect till last might be to preserve the principle that choice should be informed. The other factors discussed above need to be thought through to enable a considered choice. There should be an assumption of capacity unless evidence strongly suggested otherwise – a point that has been captured within the Mental Capacity Act (2005) in the UK. Wherever possible, the choice expressed by the resident and / or family should be sought and documented early in the disease course, so that at a future time when eating and swallowing become problematic staff should be able to know and be guided by the individual's wishes.

In my own experience as a care home manager employing these principles, the choice tended to be 'no' to tube feeding in these circumstances. But it needs to be remembered that this is a decision usually made well in advance of eating and swallowing becoming problematic. Often reality changes preferences we previously imagined we would favour. An example is that of a good friend of mine who throughout pregnancy let everyone know she wanted to deliver her baby 'naturally' without any pain-relieving drugs. She insisted to her husband and the midwife that even if she pleads for analgesia on the day, it is to be denied on the basis that her wishes expressed while clear-

headed override anything she might yell while emotionally upset and in pain. The midwife made it clear that she would regard my friend as having capacity to make a choice in such a situation and advised not to ask for pain relief if it isn't wanted, and to expect any requests made on the day to be regarded as valid. The husband on the other hand promised he wouldn't allow any pain-relieving intervention no matter what. In the event, my friend cried out for an epidural and got it. Her husband encouraged the choice and afterwards my friend was thankful for what happened. The point is that the goal posts moved when the imagined scenario became a current issue.

An example more specifically related to decisions about tube feeding is that of 'Brenda' (a pseudonym) I have previously discussed in greater depth (Smith, 2011). Brenda told nursing home staff members that when her progressive multiple sclerosis led to her being unable to cut her own food, she would not want anyone to cut it for her, she would choose to discontinue eating. In time it happened that she depended on staff members to cut her food and while Brenda accepted this she made it known that when she is unable to eat solid food she would not accept a softened diet and would go without food. She did later accept a softened diet and insisted though that she would never eat liquidised food. Eventually Brenda chose to accept liquidised food but ensured it was documented that should nutrition become only possible via tube feeding she would not accept this. By the time this last scenario became a reality Brenda had also lost the ability to communicate verbally or in writing.

Staff member opinions became divided over how to interpret Brenda's current wishes. Some were keen to stress that the wish not to be tube-fed was clearly documented and should be respected. Others pointed to Brenda's history of 'moving the goal posts' whenever imagined scenarios turned to reality and felt sure that she would now choose artificial nourishment. Attempts to engage with Brenda, and interpret body language including eye movements tended to reinforce the views held by each individual staff member. Of course resolving an issue of this nature must take account of the range of factors discussed above but the example demonstrates that respecting autonomy can be difficult and contentious.

#### **Will documented choice necessarily pre-**

#### **vent unwanted implementation of tube feeding?**

Poor communication between the nursing home and hospital staff at the time of an admission for an unrelated acute event can lead to hasty inappropriate decisions to implement tube feeding despite any previously documented wishes to avoid this action. Care coordination and sharing of information between hospitals and nursing homes has been found to be often inadequate and in need of improvement and can result in this kind of error of judgement (Delabrière et al. 2014).

#### **What is the way forward?**

Recent developments such as the appointment of specialist dementia nurses in acute hospitals who liaise between patients, families, nursing home staff and professionals in the hospital, and working closely with speech and language therapists with expertise in neurodegenerative disease-run training programmes for staff and for families may help to reduce the likelihood of impromptu decisions such as whether tube feeding might be appropriate in specific circumstances. Although many people with advanced degenerative neurological conditions may not have dementia, some features such as impaired swallowing and communicating ability are clearly relevant to this issue and it seems reasonable to assume that they might all benefit from the advocacy skills of a specialist dementia care nurse.

In my own region of East Anglia in the UK, one such collaboration between dementia care specialist nurse and speech and language therapist (SALT) with neurodegenerative disorder expertise has resulted in an ongoing multidisciplinary training programme. An educational package contains a leaflet targeting family members who may be concerned that something should be done when their loved one's food and drink intake is reducing along with the dying process (Smith & Morrow, 2010). The leaflet reinforces practical advice such as offering pureed food and thickened fluid if advised by the SALT, and suggests trying alternating temperature (hot and cold) and taste (spicy, sharp, sweet) to stimulate eating. Good mouth care and a calm environment without distractions are promoted. Those thinking about whether tube feeding might be an option are encouraged to consider what the individual would have wanted; what is expected to be achieved and whether the expectations are realistic given the evidence



available; whether tube feeding is really in the interests of the person and whether some benefits of human contact and stimulation from food might be lost.

I would argue that the quality of engagement between professionals and family members on these issues is as important as the information conveyed in the discussions and reinforced in literature such as the leaflet referred to above.

Reid & Pantilat (2013), suggest that the language used by health professionals when speaking to families about making this choice is a critical factor and suggest that it is important to adopt a skilful and reassuring approach. They advocate open questions such as 'how are you finding managing to eat?' and 'What do you hope for in the future'. This allows topics- such as tube feeding for example- to come up in conversations without being perceived as having been suggested or recommended by health professionals. Also, a grasp of the unfortunate inevitability of the disease trajectory and final outcome regardless of any efforts to persist with giving food is reinforced. The phrase 'artificial nutrition' can aid appreciation of the unnatural and invasive nature of tube feeding (Reid & Pantilat, 2013).

## Conclusion

To date available evidence cannot support effectiveness of tube feeding for nursing home residents with advanced degenerative neurological conditions including movement disorders and dementia and /or with a history of stroke in terms of any benefits assessed so far such as increased survival or improved quality of life. Yet the intervention continues to be initiated for this group. Advanced directives, education for patients and family members about the inevitability of loss of ability to eat and drink in advanced disease stages and improved communication between nursing home and hospital staff when transfers occur are among factors that may decrease the likelihood of inappropriate initiation of tube feeding.

Research has identified associated factors but cannot explain why PEG tube feeding is often implemented for this group who generally express a wish to avoid it when given the opportunity to assert a preference. This article has sought to offer a structured approach to decision-making and to highlight approaches conducive to patient-centred practice.

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## Neuroscience Methods, Nursing and Patients

Joseph Lee

### Abstract

Wherever they work, in all contexts neuroscience nurses have an outlook towards patients and their families, coupled with professional expertise in healthcare of the brain and central nervous system. For those in research laboratories, institutes, and academic medicine, direct patient management is less immediately evident in comparison with the nursing profession. However, progress in medicine and neuroscience has relevance for patient care. This article seeks to highlight some connections between research and patients, drawing links between several neuroscientific methods and how they relate to patients' lives. Many methods today are highly dependent on complex technology. However, it is possible to look beyond this to appreciate the human dimensions. There are five areas that were explored:- the lowest physical level of molecules and genetics where advances are occurring; then, a look at the realm of neurons and networks and a case of autoradiography; thirdly, the whole brain and some neuroimaging methods. This is where many patients encounter the world of neuroscience in its medical and nursing settings, as well as in the final two areas: brain damage and neurosurgery. The article concludes with a few comparative observations, and a cautionary note. In all, what is shared ground with neuroscience research especially clinical research, and neuroscience nursing, is patients looking for the best outcomes from healthcare.

**Key Words:** *Neuroscience methods, brain, patients, neuroscience nursing*

### Introduction

Whether in the acute setting, theatre, rehabilitation or community, neuroscience nurses are focussed on patients and families as well as health care related to the brain. In Australia, of the 48,000 each year who will have a stroke, it is estimated that about one third of survivors are affected by depression. Stroke nurse specialists have "the ability to deliver all information and education at a level appropriate to the individual's educational, psychological, social, and cultural requirements," says neurologist Graeme Hankey, Royal Perth Hospital, Perth (as cited in Christodoulou, 2012, p. 211).

For colleagues working in research laboratories, patient management is less evident, at least immediately compared with the nursing profession. However, there are connections. Smith (2006) argues that "inevitably, nursing clinical practice is shaped by medical advances, policy directives like the European Union's Working Time Directive limiting

junior doctors' hours, supply and demand of health professional staff and economic considerations" (p. 1070). Nevertheless, neuroscience nursing research is allied with progress in neuroscience. Specialized neuroscience intensive care nurses promote the link with high-quality neuroscience research (Chatfield, 2008). There were more neuroscience research studies published in nursing journals in the 1990s than from 1960-1988, and the scientific methods increased in complexity during the decade (Dilorio, Yeager, Donahue, Wasserman, Postier & Broderick, 2004).

Interestingly, neuroscience nurse researchers also tended to use self-reports which is reasonable given that more studies were examining psychosocial variables (Dilorio et al. 2004, p.66). Again there is a focus on patients. In setting updated research priorities, the Neuroscience Nursing Foundation (USA) identified five programs: nursing care outcomes in acute care settings, health promotion, end-of-life nursing care, improving quality of life, and special populations (Dilorio, Hinkle, Stuijbergen, Algase, & Smeltzer, 2011).

This article seeks to highlight some links between neuroscientific research methods and

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upholding a vision for patients. Medicine needs an epistemology able to recognise patients and clinicians as persons, because reductionist concepts in science and medicine are generally unquestioned (Henry, Zaner & Dittus, 2007). Many methods have high technological standards. Naturally there is technology in nursing too. Besides the intracranial pressure (ICP) monitor, there are new technologies for the neuroscience intensive care unit that provide crucial information on intracranial dynamics to maximise outcomes in this critically ill patient population (Bader, 2006). We begin at the lowest physical level.

### Molecules and Genetics

Genetic neurological disorders include Huntington's disease and hereditary ataxias. Gene profiling of the human brain can accurately identify the multiple candidate genes in complex psychiatric disorders (Atz, Walsh & Cartagena, 2007). Scientific innovations have promoted the characterisation of three important genetic units: the genome, the transcriptome, and the proteome (Morrison, Kinoshita, Johnson, Uo, Ho, & Veenstra, 2002; Guo, Fu & Van Eyk, 2007). The genome depicts the full set of genes encoded by an organism's DNA. The transcriptome takes in the full complement of mRNA transcripts that are transcribed from the genome of a cell. The proteome depicts the full complement of proteins expressed by a cell in a point in time. For example, the use of high-sensitivity and rapid-throughput techniques for wide proteome analysis include the study of proteomes of a brain tumour *glioblastoma multiforme* (Melchior, Tholey, Heisel & Huber, 2009).

Some study the metabolome: molecules in a system covering an assortment of small molecules like glucose, ATP, cholesterol, and biogenic amine neurotransmitters (Quinones & Kaddurah-Daouk, 2009). Studying disease pathophysiology, biomarker identification and pharmaceutical action in this way means there are benefits from a systems approach. Whereas classical biochemistry usually centred on single metabolites, metabolomics encompasses quantitative data gathering on a large range of metabolites. This can reveal insights into drug exposure and disease (Smith, 2013).

At the level of 1 ångström (Å) or  $1 \times 10^{-10}$  metres (Sejnowski, 2003, p. 263), the molecular constituents of the genome are only a very tiny part of a brain. The "person" appears remote unless perhaps that entails

"personalized medicine" (Tremblay & Hamet, 2013; Wolff, Brown, Buryanek & Rytting, 2012), which is centred on genomic information and the uniqueness of human individuals. Clinical researchers such as Samuel Berkovic, who was named a Companion of the Order of Australia (AC) in the 2014 Australia Day Honours, identified gene mutations and molecular mechanisms for epilepsy. Speaking about his subspeciality, "the fundamental question that many patients and families want answered is, what has caused this disease?" (cited in Williams, 2010). One can say it was "a genetically predestined thing, you can live with that, you can't do anything about it, well not at the moment, but at least you've got closure" (p. 570).

### Neurons and Networks

There are numerous methods operating at the level of cells and tissues. Patch clamp techniques enable single-channel currents to be studied from tiny patches of membrane or minute cells (Sakmann & Neher, 1984) with technological advances (Stuart, Dodt & Sakmann, 1993). It is used to study brain disorders such as epilepsy (Oliva, Berkovic & Petrou, 2012). *In vitro* (glass) experiments (Steriade, 2001; Hájos & Mody, 2009) which examine brain slices also have had technological upgrades. A lesser-known method uses radioactivity. Radiography involves irradiating an object with high energy radiation to produce an image on photographic film. However, in *autoradiography*, a radioactive isotope is instilled into the specimen and its decay is detected using photographic techniques (Tsoi & Wong, 2002). The binding and distribution of radiolabelled chemicals in benzodiazepine receptor system has been studied by *in vitro* postmortem autoradiography on whole hemisphere brain slices of Alzheimer patients and control subjects (Gulyás, Makkai, Kasa, Gulya, Halldin, 2009). Similarly, the understanding of essential tremor pathophysiology is underdeveloped with relatively scarce clinicopathological studies, compared to other central nervous system diseases. Autoradiography was used to study human tissues and reveal a neurochemical difference in individuals with essential tremor, versus controls or patients with Parkinson's disease (Paris-Robidas, Brochu, Sintès, Emond, Bousquet & Calon, 2012); since essential tremor is frequently misdiagnosed as Parkinsonian tremor. The research environment is the cellular world. Like the first section above on molecules and genetics, the technology and the engineering know-how surpasses the size, mass and status of one

brain cell - a micro-level part of tissues in the brain of a human person.

### The Whole Brain

The brain at the macroscopic level is most familiar to healthcare practitioners and patients. Several standard methods are relatively recent: computed tomography (1972), positron emission tomography (PET, 1975), single positron emission tomography (SPECT, 1976), and magnetic resonance imaging (MRI, 1980) (Gray & Orton, 2000). There are some other methods.

Used since 1929, electroencephalography (EEG) is suited to analysing quick neural events e.g. in executive control, due to its high temporal resolution (Astle & Scerif, 2009). EEG coherence analysis has been used to investigate alterations in functional connectivity in the cortex during normal and abnormal development and in various brain states and pathologies (Knyazeva & Innocenti, 2001). There was a girl with severe bilateral lesions of the primary visual area at 3 weeks and abnormal vision; researchers found no interhemispheric coherence (ICoh) response until 9 years, whereas in control group children there was visual stimulation generated higher occipital ICoh at 6 to 7 years old.

Such abnormal functional brain connectivity is a possible cause of developmental brain disorders in child cognitive dysfunctions (Murias, Swanson & Srinivasan, 2007). One EEG study found changed functional connectivity especially in the frontal brain regions: a static state of deficient connectivity in attention-deficit hyperactivity disorder (ADHD) plus a stimulus-induced state of overconnectivity between and within frontal hemispheres. The person also emerges through cognitive-affective studies. Some associate affective disorders such as found in depressed persons and relatively decreased left resting brain activity (Sutton & Davidson, 2000). However others contend that hypothesised relationships between EEG and variables are not empirically validated (De Raedt, Franck, Fannes & Verstraeten, 2008).

Magnetoencephalography (MEG) was introduced around 1968 (Salmelin & Baillet, 2009). Small magnetic fields ( $10^{-14}$  Tesla) are produced by the electrical activity of neurons and MEG records these (Llinás, 2000). Different to EEG scalp voltages recorded in microvolts, the typical magnetic induction produced by neural currents is extremely small, hence requiring complex technology for detection

(Baillet, Mosher & Leahy, 2001). MEG can be used in very young children, sleeping infants (Astle & Scerif, 2009), even to study foetal brain function *in utero* (Matuz, Govindan, Preissi, Eswaran, 2012). It is a valuable diagnostic tool in the clinical situation, e.g. patients with brain tumours and epilepsy (Dai, Zhang, Dickens & He, 2012) and cortex study after hemispherectomy (Yao, Qiao, Shu & Xu, 2013). For patients, the encounter with the neuroscience world is brought closer by neurodegeneration, head trauma, brain injury, other diseases such as metastatic cancer, or other reasons. The scanners while technologically advanced do not 'see' the patient's family, their mental state, the responsibility for care, or financial and time implications. It is largely clinicians and nurses who diagnose and manage the human side.

### Brain Damage

Concussion in sports is a notable issue (Evans, 2013). Consider head injury and neuropathological damage associated with boxing. While the knockout (KO) punch is dramatic, "the cumulative effects of multiple sub-concussive head blows appear to be the primary cause of neurologic injury in boxers, especially for a subset of professional boxers with extensive fight histories" (Heilbronner, Bush, Ravdin & Broshek, 2009, p. 12). There are links between neurological, psychiatric, or histopathological signs and symptoms of encephalopathy or brain disease, and the number of bouts fought. Heilbronner et al. (2009) found three kinds of neurological trauma: 1. acute neurologic injuries 2. persistent groggy states and post-concussion syndrome; 3. chronic traumatic encephalopathy (punch-drunk syndrome or dementia pugilistica).

The theme of the person as patient continues in the treatment of brain injury, neurodegenerative diseases, and cancer (Kaal & Vecht, 2008). Lesions studies emanate from neuropsychology's study of brain damage in patients (Allman & Scott, 2013). These "loss-of-function" methods test whether a particular brain region is necessary for a certain cognitive process (Tsuchida & Fellows, 2009). Damasio & Damasio (2000) point out that while associations between neuropsychological dysfunction and specific areas of the cerebral cortex have been validated, and localisation of damage are ascertainable from neurological signs, these links, nonetheless, do not entail that the functions affected by the lesion were particular to the tissue destroyed by that lesion. Lesion-symptom mapping results are correlatory; they do not prove a nec-

essential role of any implicated brain regions (Fellows, Heberlein, Morales & Wu, 2005; Utz, Humphreys & Chechlacz, 2013). Cognitive, behaviour and motor deficits are life-limiting realities for human beings. Personhood is related to brain injury (Au, 2000), which draws in the psychosocial, family and socio-cultural considerations. The methods are effective means to assist in health care.

### Neurosurgery

Critical care and intensive care settings are indeed necessary for life-saving interventions, though patient research and technological tools can involve neurosurgery. Intractable epilepsy is commonly treated by surgical removal of the anterior temporal lobe of the brain (Binder, Swanson, Hammereke & Sabsevitz, 2008). In brain tumour resection, preservation of neurological function after procedures is needed for patients' quality of life (Klijn, Hulscher, Balvers, Holland & Ince, 2013). The aim is to maximize tumour resection yet to minimize permanent damage to functionally important brain tissue. Electrocortical stimulation mapping is the gold standard for real-time functional mapping of the brain surface. Other procedures like laser speckle imaging have been able to detect increases in cortical microcirculatory blood flow induced by motor activity during awake neurosurgery.

While there is data about aphasia prior to malignant brain tumour resection, there is less information about language outcomes - the kind and severity of aphasia - after tumour resection (Davie, Hutcheson, Barringer, Weinberg & Lewin, 2009). Figures suggest that one-third to half of patients who have had a left hemisphere brain tumour resection experience aphasia. To discover patterns of aphasia subtype and severity, Davie et al. (2009) analysed the Western Aphasia Battery (WAB) scores retrospectively, of individuals tested during the acute recovery phase following neurosurgery for resection of malignant brain tumours. They found that in the acute recovery phase following brain tumour resection, aphasia was generally mild (63% of patients) and that the most frequent subtype was anomic aphasia [difficulty naming people and things] (48% of patients), irrespective of tumour grade or lesion location.

Moreover, the pattern of aphasia severity was different in patients who underwent brain tumour resection and those who experienced stroke. Patients assessed after stroke

showed higher rates of global aphasia (20-40%) and lower rates of anomic aphasia (9-28%), while brain tumour resection had low rates of global aphasia (3%) and high rates of anomic aphasia (49%). The lesser global aphasia rates after brain tumour surgery are possibly explained by reorganisation of language during tumour growth, the helpful use of pre-operative neuroimaging, and intra-operative awake cortical mapping techniques that enable surgeons to minimise injury to crucial language regions during tumour resection (Davie et al., 2009).

To find functionally important brain regions, directly stimulating the cortex is a way to map the brain. A newer method is transcranial magnetic stimulation (TMS), a noninvasive means to stimulate the brain using a short, intense magnetic field produced by an electric current passing through a magnetic coil, located at the head and on a tangent to the scalp (Hallett, 2007). A review concluded that so far, navigated TMS is the only brain mapping modality that is able to stimulate the brain and record the output in a painless, non-invasive manner. "Its accuracy for delineation of the motor cortex is comparable to that of DES [direct electrical stimulation]. However, DES cannot be replaced by a non-invasive method due to its unique capability to stimulate subcortical structures accurately and to monitor function during surgery" (Takahashi, Vajkoczy & Picht, 2013, p.6).

Another useful technique is hemispheric anaesthetisation by amobarbital (Wada & Rasmussen, 2007; Whitman, Morrison, Becske, Barr & Carlson, 2012), also known as the Wada test or intracarotid amobarbital procedure (IAP). The clinical use of IAP in neurosurgery patients causes each hemisphere to be functionally disabled by anaesthetisation caused by sodium amobarbital injected into the carotid artery. IAP and pre-operative procedures aim to minimise injury to crucial brain regions during neurosurgery are themselves dependent on technology.

### Conclusions

The Australasian Neuroscience Nurses' Association (ANNA), *Professional Standards for Neuroscience Nurses (2013)* uses the term "patient" 58 times, indicating its professional focus. Traditional categories of bench versus bedside, laboratory versus clinic, biomedical versus nursing, can be somewhat fluid given research collaborations. At the

Dimension	Neuroscience	Neuroscience nursing
Goals	Scientific, medical, diagnostic	Patients, expert patient management, families, person-centred.
Areas	"anatomy and physiology", genome, neuron, synapse, cortex, neurochemistry, neurobiology, neuroimaging, diseases, drugs, etc.	"advancement of neuroscience nursing thus enabling optimal patient centred care" (ANNA, Mission), but also immersed in neuroscience disciplines
Example brain cancer	Next discovery from research in neuro-oncology and treatments will most likely come from researchers.	Diagnosis of a brain tumour causes complex reactions, particularly for a high-grade diagnosis which is different to other cancers, due to the expected, considerable cognitive changes experienced by the patients and witnessed by their families (Lucas, 2013).
Teams	"...major breakthroughs in understanding the brain will continue to emerge from the labs of individual investigators." (Anonymous 2013)	Varies with nursing contexts: clinical and trauma settings, community, research, education
Technology	Indispensable in most methods	Used as instruments in delivery of care
Size	From molecules upwards to systems	Body systems, family systems
Wider links	Translational neuroscience, with caution (see below)	Neuroscience nursing research, collaborations

**Table 1** (Above): Some comparisons of neuroscience research and neuroscience nursing.

peril of oversimplification, we can try to generalise some characteristics in a table, without implying any restrictions (Table 1).

The goal of bringing neuroscience insights to improve treatment and preventive strategies for neurobehavioral disorders is known as Translational neuroscience (Whitten, 2013). Research remains crucial. Insel & Landis (2013) find that the biggest barrier is the need for a deeper understanding of how the brain works so as to understand brain disorders. In the last 25 years there has been stunning progress, yet much of this has yet to change the lives of millions burdened with CNS disorders like Alzheimer's disease and autism. Insel & Landis (2013) also note the mounting concerns there are in the USA about the trend in basic science grant applications, which mention a disease then promote the proposed research's translational impact. Such 'translational blurbs' may be "misleading and not recognise the divide between what is basically understood and is needed for clinical application" (p. 564). Drawing from that, we can see how associations between the laboratory, clinical trials, deployment in healthcare, patients and families, will be evidence-based, subject to funding, requiring much effort and long hours leading to fruition.

Neuroscience nurses may not see the clinical endpoints of neuroscientific research in their professional practices. Neuroscience has been described as a "splintered field," with some 10,000 laboratories worldwide, "at any large neuroscience meeting, one is struck by the pace of discovery, with 50,000 or more practitioners heading away from each other in all directions, in a sort of scientific Big Bang" (Koch & Reid, 2012, p. 397). The one constant is human persons and their brains, being treated as patients in health care systems, with technological methods, by professionals interested in optimum outcomes. Neuroscience nurses are closest to these patients and their families, while clinical research, subject to financial backing, looks for causes and new and improved treatments. Together such outcomes can be reached and bettered.

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# Protein Requirements in Traumatic Brain Injury: A Systematic Review.

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## Abstract

Traumatic Brain Injury (TBI) occurs in events where sudden blunt or piercing trauma damages the brain. TBI has been associated with higher nutritional requirements as a result of hypermetabolism and hypercatabolism during the acute phase of initial trauma. Early nutrition is essential to ensure energy and protein requirements are met, minimising the consequences of hypermetabolism and hypercatabolism. Conflicting ranges have been reported in the literature on protein requirements for TBI making it difficult to practise evidenced based nutrition. A systematic literature search was conducted using four research databases. All study designs were included with defined selection criteria. Study comparisons were made between defined protein requirement ranges, clinical outcomes and rates of mortality and morbidity. Using the National Health and Medical Research Council (NHMRC) guidelines, 14 studies were appraised. The literature suggests a protein range of 1.5-3.0g/kg body weight (BW)/day or at least 15-20% protein calories to meet protein requirements in TBI patients. Protein exceeding 2.5g/kg is suggested to provide minimal clinical benefit. Conclusions recommend ongoing qualitative research and clinical trials to determine administered protein ranges for clinical benefit for TBI patients.

**Key Words:** Traumatic brain injury, protein requirements, nutrition support, nutrition intervention, nitrogen balance, parenteral/enteral nutrition.

## Background

Traumatic brain injury (TBI) occurs when there is sudden blunt or piercing trauma to the brain (Segaran, 2007). Data from the Australian Institute of Health and Welfare indicates that rates of hospitalisation for males per 100,000 population were two and a half times those for females in 2004-2005 with the most common causes of brain injury being falls, transportation and assault (Harrison, Henley et al. 2008).

TBI can be further clinically categorised into primary and secondary brain damage. Primary brain damage occurs at the time of initial injury. Secondary brain damage is preventable neurological damage occurring in the first few hours or days after the initial injury. The aim of treatment within the critical phase post TBI is to minimise secondary brain injury and improve neurological outcomes. The Glasgow Coma Scale (GCS) score indicates cognitive function and is regularly used as a tool in categorising TBI. GCS score determines the severity of TBI; mild 13-15, moderate 9-12 and severe injury 3-8.

Nutrition priorities in acute injury phase include assessing nutritional requirements, which may be increased as a result of metabolic response to injury, and providing adequate nutrition to meet these needs (Ghajar 2000). Numerous studies have identified a proportional association between the severity of TBI and increased nutritional requirements (Rapp, Young et al. 1983, Clifton, Robertson et al. 1986, Vizzini and Aranda-Michel 2011, Dhandapani, Dhandapani et al. 2012, Dickerson, Pitts et al. 2012) due to hypermetabolism and hypercatabolism.

Nutritional therapy in brain injury is challenging as a result of changes in metabolic and immune responses and gastrointestinal function; impacting on oral intake due to reduced digestive function, higher risk of aspiration and absence of cognitive/mobile function because of the nature of TBI (Frankenfield 2006). Furthermore, research shows discrepancies in the extent and duration of hypermetabolism making it difficult to predict energy requirements individually for each patient with TBI (Rapp, Young et al. 1983, Vizzini and Aranda-Michel, 2011).

However, it has been shown that optimal nutrition support can assist rehabilitation of TBI patients (Ghajar, 2000), although exact quantitative protein requirements continue to be debated in the literature and varying recommendations exist (Segaran, 2007)

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There is limited evidence-based literature on appropriate protein requirements for TBI patients and most of the studies have limitations, making the results difficult to translate into the clinical setting (Bruns and Hauser, 2003).

### Research Objectives

To analyse and investigate recommended guidelines on protein requirements in patients with TBI and conduct a systematic literature search on protein requirements in TBI.

- Appraise the literature using the National Health and Medical Research Council (NHMRC) guidelines, Critical Appraisal Skills Programme (CASP) guidelines and Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) statement.
- Prepare a summary report detailing the process and findings of the literature review, with particular attention to measured study outcomes.
- Incorporate results of the systematic review in the development of a neurosciences database for ICU patients presenting with TBI.

### Method

#### Search strategy

A systematic review was conducted to identify relevant studies. Three electronic databases were searched: Medline, The Cochrane Library and Google Scholar. Combined MeSH terms varied depending on the literature database. A manual search on the references of selective studies was additionally conducted for inclusion of supplementary eligible studies. The search strategy is outlined in Figure 1.

#### Selection criteria

This systematic review was limited to English research articles. Legal hearings, non-English studies, letters to the editor and commentaries were excluded. Only studies on adult (>19 years old) populations with an identified traumatic, severe, moderate or mild brain injury, as defined by each study's authors, were included. Paediatric, animal studies and studies on stroke were excluded.

#### Method of review

The NHMRC guidelines, CASP guidelines and PRISMA statement were used to critically appraise included studies. Extracted data included authors' names, year of publication, study design, interventions,

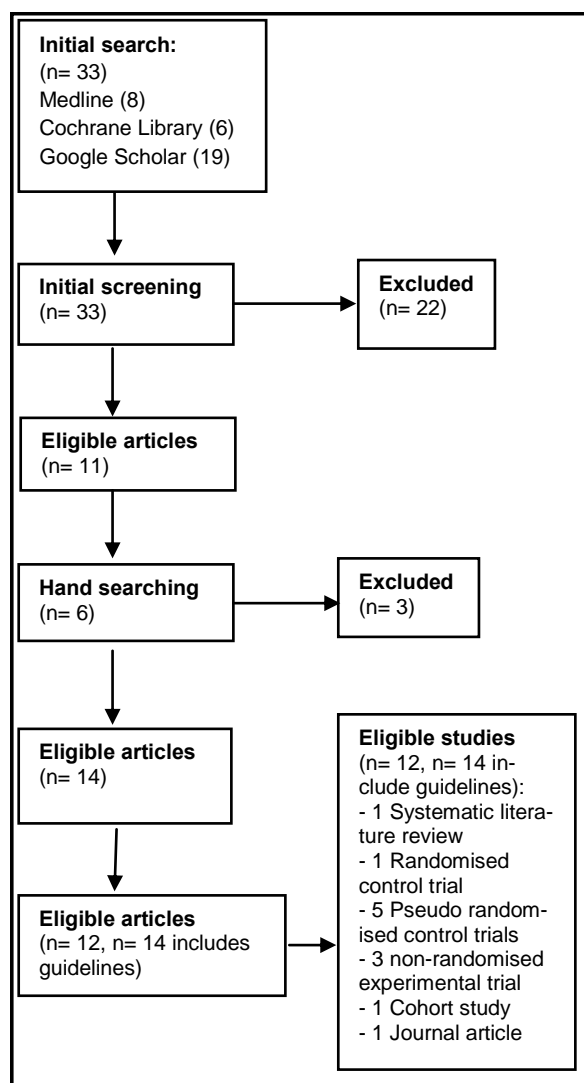
	Searches	Results	Search Type
1	'Brain injuries'	34762	Advanced
2	Exp Brain Haemorrhage, Traumatic	436	Advanced
3	1 or 2	35103	Advanced
4	Nutritional Requirements	17185	Advanced
5	Proteins	184814	Advanced
6	Critical Care	24510	Advanced
7	Intensive Care	15306	Advanced
8	Rehabilitation	16491	Advanced
9	4 and 5	424	Advanced
10	6 or 7	39497	Advanced
11	3 and 9	1	Advanced
12	3 and 5	58	Advanced
13	From 12 keep 17,35, 41, 42	4	Advanced
14	Metabolism and nutrition in patients with moderate and severe traumatic brain injury: a systemic review" (Article Title)	1	Advanced
15	Nutritional Support	2901	Advanced
16	3 and 15	31	Advanced
17	protein.tw.	2238118	Advanced
18	16 and 17	7	Advanced
19	From 18 keep 1-2, 4-6	5	Advanced
20	16 not 18	24	Advanced
21	Limit 20 to "all adult (19 plus years)"	6	Advanced
22	From 21 keep 1-2	2	Advanced
23	Traumatic brain injury nutrition support protein (including related terms)	10640	Basic
24	Limit 23 to "all adult (19 plus years)	2670	Advanced
25	From 23 keep 2-3	2	Advanced
26	From 24 keep 1-3, 5, 8-10, 12-13, 15, 17-22...	17	Advanced
27	13 or 14 or 19 or 22or 25 or 26	25	Advanced

Figure 1 (Above): Search Strategy

clinical outcomes, study interventions and limitations of the study.

### Study outcomes

Primary study outcomes included clinical and neurological improvements in GCS score, nitrogen balance, disability and mortality. Observed secondary outcomes include the length of stay (LOS) and infectious complications. Study outcomes were assessed to evaluate validity of administered protein ranges to aid clinical recovery in TBI. Furthermore, the Northern Sydney Local Health District TBI management guidelines and other best practice protocols will be revised to determine feasibility of further research.



**Figure 2** (Above): A flow diagram showing study selection for inclusion of eligible studies in systematic literature review.

## Results

### Study characteristics

The search strategy yielded 33 results. After initial screening, 22 studies were excluded, resulting in 11 studies (including guidelines) for review. Figure 2 shows reasons for exclusion. Further hand searching identified 6 additional studies, of which, 3 were included for review. Therefore from an overall pool of 39 potential studies, 25 were excluded and 14 studies (including guidelines) included for systematic review.

An extensive literature search identified 1 Randomised control trial (RCT) (Borzotta, Pennings et al. 1994), 5 pseudo RCT's (Rapp, Young et al. 1983, Hausmann, Mosebach et al. 1985, Young, Ott et al. 1987, Grahm, Zadrozny et al. 1989, Falcao de Aruda and de Aguilar-Nascimento 2004), 3 nonrandomised, uncontrolled experimental designs (Clifton, Robertson et al. 1986, Dhandapani, Dhandapani et al. 2012, Dickerson, Pitts et al. 2012), 1 cohort study (Bochicchio, Bochicchio et al. 2006), 1 systematic literature review (Vizzini and Aranda-Michel, 2011) and 1 journal article (Rajpal and Johnston, 2009).

The range and mean sample size of all reviewed studies was 20-249 and 64.3 subjects respectively, excluding journal articles and systematic literature reviews. Ten studies had defined selection criteria.

The CASP guidelines were used to determine the validity of the studies for inclusion in the systematic review. Level and strength of evidence was assessed in each study in accordance to the NHMRC evidence guidelines. The PRISMA checklist was used to evaluate the study design and methods of systematic literature reviews.

### Participants

Common eligibility criteria across all studies included GCS score, definition and diagnosis of TBI, age, consensus, severity of TBI, route and timing of feeding. Four studies were not experimental and did not require selection criteria.

Participants had a GCS of less than 8 with a range of 4-12. Some studies included other neurological injury scoring systems (Injury Severity Score, Abbreviated Injury Scale Score and/or APACHE II scores) additional to a GCS score to determine severity of TBI and assess neurological outcomes. The age range of subjects was 18-60 years. The ma-

majority of the study population were males involved in motor vehicles accidents.

#### **Nutrition intervention:**

##### *Protein and energy requirements in TBI*

Studies commonly showed both energy and protein were observed and trialled jointly in TBI in attempt to identify the effectiveness of nutrition support- involving both protein and energy, not exclusively protein.

##### *Nutrition Intervention*

Patients were commonly commenced on enteral nutrition upon admission unless parenteral was more suitable due to clinical complications. Where nutrition support was inadequate, enteral nutrition was further supplemented (Bochicchio, et al. 2006, Dickerson, Pitts et al. 2012). Most studies followed the Brain Trauma Foundation (BTF) guidelines to initiate enteral feeding within 72 hours (Falcao de Arruda and de Aguilar-Nascimento, 2004; Bratton, Chestnut et al. 2007, Rajpal and Johnston, 2009). Decisions regarding implementing either enteral or parenteral nutrition depended on individual study objectives or clinical decision by either the physician or medical team.

##### *Follow up*

2 RCTs stated follow up procedures (Young, Ott et al. 1987, Dhandapani et al. 2012). The Dhandapani et al. (2012) study involved a selection of 95 subjects with a GCS score between 4-8 within 24 hours of admission. Primary outcomes were measured at 3 and 6 months post injury either directly or via telephone. Other secondary outcomes were measured weekly. Young et al. (1987) examined 51 subjects admitted with a GCS score of 4-10 within 24 hours of admission. Biochemical and anthropometric outcome measurements were measured. Subjects were studied for 18 days post injury with follow-up measured at 3, 6 and 12 months post injury. The remaining studies did not include follow-up.

##### *Study outcomes*

Study outcomes included GCS score, nitrogen balance, anthropometric measures for malnutrition, degree of disability/motor function and risk of mortality and morbidity. GCS score was used most frequently to assess neurological progress and recovery. Young et al. (1987) found improved nitrogen balance and GCS score when allocated protein values of 1.35 and 0.91 g/kg BW/day were administered to enterally and parentally fed

subjects respectively. Clifton et al. (1986) aimed for a target percentage range of 16-22% dietary intake being protein calories and showed clinical improvements in GCS score and nitrogen balance although nitrogen balance commonly remained in a negative state during acute admission. A study by Dickerson et al. (2012) administered protein ranges of 1.0-3.0g/kg BW/day. Results indicated improved clinical outcomes with 54% of subjects achieving a positive nitrogen balance when protein values of 2.0-2.5g/kg BW/day is administered. In comparison, 38% of subjects achieved positive nitrogen balance given a protein range of 1.5-1.99g/kg BW/day and 29% achieved similar outcomes with a protein range of 1.0-1.5g/kg BW/day. Graham et al. (1989) observed improved nitrogen retention and reduced infection rates during admission length of stay when experimental nitrogen intakes were double the values of the control group (11.1g/day vs. 5.6g/day). Dhandapani et al. (2012) provided 2.0g/kg BW/day of protein showing mild increases in subjects' total body protein levels and identified declining weight and nutritional status when early feeding was delayed. Hausmann et al. (1985) trialled protein values of 1.4g/kg BW/day in TPN vs. 2.4g/kg BW/day combined TPN and enteral nutrition with results of higher nitrogen balance values when the TPN solution was lower in protein, compared to the combined TPN and enteral method. However there was no found statistical difference in mortality and creatinine excretions between the two feeding regimes. Vizzini and Aranda-Michel (2011) reviewed literature and found provision of 2.2g/kg BW/day correlated to positive nitrogen balance however subjects receiving 1.5g/kg BW/day had negative nitrogen balances. Overall conclusions from Vizzini and Aranda-Michel suggest 2.0-2.5g/kg BW/day are recommended for nitrogen loss prevention. Bochicchio et al. (2006) calculated and administered protein values based on double the individual's normal protein requirement. Rajpal and Johnston's (2009) review found a study identifying a value of 2.2g/kg BW/day improved net protein synthesis and an overall range of 1.5-2.0g/kg BW/day to facilitate wound healing. Borzotta et al. (1994) allocated 2.5g/kg BW/day with a significant improvement in GCS score and nitrogen balance. Borzotta concluded that a protein intake of 2.2g/kg BW/day showed better clinical outcomes in GCS score and nitrogen balance than a protein intake of 1.5g/kg BW/day.

## Discussion

The resting energy expenditure (REE) for head-injured patients has been debated amongst studies. General consensus in practice shows that patients with TBI have increased energy requirements on par with burns and other traumatic injuries (Clifton, Robertson et al. 1986). Bratton and Chestnut et al. (2007) reported a weight loss of 15% per week as a result of hypermetabolism, hypercatabolism and potential underestimation of nutrition requirements, leading to significant nitrogen losses and muscle wasting. Many studies determined daily protein requirements by assessing urinary urea, nitrogen intake and the nitrogen balance equation (Rapp, Young et al. 1983, Hausmann, Mosebach et al. 1985, Clifton, Robertson et al. 1986, Young, Ott et al. 1987, Grahm, Zadrozny et al. 1989, Borzotta, Pennings et al. 1994, Falcao de Arruda and de Aguilar-Nascimento 2004, Dickerson, Pitts et al. 2012). Other methods used to calculate protein requirements include the Harris Benedict equation and the Kjeldahl method (Dhandapani et al. 2012). In the studies evaluated, protein was reported as nitrogen intake/day, protein intake/day or protein calorie percentage.

Protein values vary greatly in the literature. The majority of studies recommend a minimum of 2.0g/kg BW/day (Hausmann, Mosebach et al. 1985, Borzotta, Pennings et al. 1994, Bochicchio et al. 2006, Dhandapani et al. 2012, Dickerson, Pitts et al. 2012). It has been observed that higher protein intake may be associated with improved GCS score, nitrogen balance, decreased LOS and reduced infections (Borzotta, Pennings et al. 1994, Dickerson, Pitts et al. 2012).

A protein range of 2.0-2.5g/kg BW/day may achieve improvements in nitrogen balance, anthropometry, infection rates and neurological function (Borzotta, Pennings et al. 1994, Bochicchio et al. 2006, Rajpal and Johnston 2009, Dhandapani et al. 2012, Dickerson, Pitts et al. 2012). Young, Ott et al. (1987) indicated administration of protein values of up to 3.0g/kg BW/day may be beneficial if nitrogen excretion is excessive, however Dickerson et al. (2012) suggested protein supplementation above 2.5g/kg BW/day shows minimal clinical benefit.

The BTF guidelines have outlined some studies identifying benefits in glutamine, arginine, and omega-3 fatty acids for aiding recovery

and reducing infection rates. In particular, studies on glutamine have shown benefits in wound healing, sepsis and recovery (Falcao de Arruda and de Aguilar-Nascimento 2004). Recent research however has shown glutamine may actually increase mortality in hospital at 6 months and is therefore contraindicated in this setting (Heyland et al. 2013). There are currently no studies investigating the effect of branch chain amino acids on clinical recovery following TBI (Falcao de Arruda and de Aguilar-Nascimento 2004).

Many RCTs have investigated the effect of enteral versus parenteral nutrition on clinical outcomes. The 2007 BTF guidelines support enteral feeding over parenteral however the guidelines are based on limited evidence (Rapp, Young et al. 1983). Parenteral nutrition is indicated when enteral nutrition alone cannot meet nutritional requirements or if the patients' gut is not functioning (Bratton, Chestnut et al. 2007, Heidegger et al. 2013). One study showed TBI patients solely on parenteral nutrition received higher protein and energy intake with better clinical outcomes than TBI patients on enteral nutrition (EN) (Young, Ott et al. 1987).

With regard to timing of feeding, some studies have indicated commencing a nutrition intervention within 72 hours is associated with improved clinical outcomes and reduced mortality (Falcao de Arruda and de Aguilar-Nascimento 2004, Bratton, Chestnut et al. 2007, Rajpal and Johnston 2009).

The BTF guidelines highlight clinical strategies to treat and manage TBI and may be used as a guide for practice in an area with limited evidence. The nutritional component of the guidelines include routes of feeding, estimated energy and protein requirements, clinical outcomes and further areas of required study relating to management of TBI (Bratton, Chestnut et al. 2007).

The BTF guidelines (2007 3<sup>rd</sup> edition) suggest increased caloric and protein requirements in patients with TBI due to hypermetabolism and hypercatabolism. The guidelines recommend energy requirements of 100-140% of REE with factors such as ventilation, state of coma and posturing influencing nutrition needs. The guidelines also recommend protein requirements of approximately 15-20% of total caloric intake to reduce nitrogen loss (Bratton, Chestnut et al. 2007).

A correlation has been established between meeting nutritional requirements via early enteral nutrition and improved outcomes. When nutrition support (either EN, PN or both) did not meet patients' nutrition requirements results showed deterioration in nutritional status and declining nitrogen balance and anthropometric measurements- reflected in low body protein levels (Bratton, Chestnut et al. 2007, Vizzini and Aranda-Michel 2011, Dhandapani et al. 2012, Dickerson, Pitts et al. 2012). A study by Hausmann, et al. (1985) showed when enteral nutrition was supplemented with parenteral nutrition in order to meet estimated requirements, only nitrogen balance showed slight improvements, there were no significant statistical differences in overall clinical benefits (Hausmann et al. 1985). The majority of these evidences are of Grade C level, therefore should be used with strict caution.

### Limitations

There were many limitations in this systematic review, in particular the lack of clinical trials and the varying results from the limited evidence. The initial literature search performed was too heterogeneous resulting in many broad literature articles on brain injuries. The search was repeated using significantly less MeSH terms which further restricted the results. The researchers included all relevant studies from both these literature reviews and by hand searching to ensure all papers meeting inclusion criteria were included.

Differences in study design, selection criteria, diagnosis of TBI, outcome goals and outcome measurements (different measurement techniques and equipment for nitrogen and protein requirements and status) limit the reliability and consistency of this systematic literature review. Study designs will always vary. Therefore each reviewed study is limited by individual study design, outcome measures and how each hospital manages TBI.

In the reviewed studies there was no publisher bias in favour of significant desirable results. All reviewed studies reported significant experimental results and discussed experimental limitations linked to increased bias. Funding or sponsorships were mentioned in respective studies to minimise bias. Clinical studies funded by external stakeholders stated relevant products included in the experimental design and acknowledged

limitations linked to sponsors. However, external stakeholder funding remains as a limitation and experimental bias would also be in question. There were limited studies which included detailed follow-up procedures otherwise follow-up was not stated in methodology.

It was difficult to adequately assess protein supplementation and improved outcome. Definitive protein requirements are rarely studied individually and are generally published alongside energy values. A lot of the studies may be considered too old to change practice and more up to date research is required. Nutrition requirements of individualised TBI cases will hinder consistency of research. Unfortunately most of the studies have methodological flaws and are considered to be of low grade evidence, which impacts on the conclusions derived from gathered literature articles.

Current methods of protein provision are guided by the patient's individual clinical and anthropometric status. This method of individualised feeding proves difficult to conduct cohort studies and the nature of TBI adds a degree of difficulty in commencing experimental trials. This area of practice requires assistance of peers and consultation with other experts in this area to define the importance, role and need for increased protein requirements for TBI.

### Recommendations

More research into the area of TBI and nutrition is required to determine definitive requirements for this vulnerable patient group. This may be achieved by conducting clinical trials across multiple sites administering protein ranges from 1.2-3.0g/kg BW/day taking into consideration feasibility, resources and study design.

A national or joint Australia-New Zealand questionnaire seeking feedback from clinical dietitians and medical professionals about their nutrition practice in TBI may assist units in providing a good starting point for clinical practice.

Empirical evidence shows that patients with TBI are surviving with modern medical, nursing and allied health interventions. It is important to raise the question on where nutrition research in TBI is best implemented, whether in critical care, acute care or rehabilitation.

## Conclusion

Due to the lack of evidence the authors are unable to suggest definitive protein requirements with patients presenting with TBI. Clinicians will need to continue to use individualised treatment plans and clinical judgement when assessing patients' nutritional requirements. Ongoing research is required to quantify recommended protein requirements in TBI.

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## Guillain-Barre Syndrome in a 15mth old: A Case Study.

Lissy Joseph

### Abstract

This case study explores the presentation, diagnosis and treatment of an 18 month old boy with Guillain-Barre Syndrome (GBS). It will mainly focus on the applied anatomy and physiology, treatment, key nursing interventions and management of a child with GBS with critical analysis using evidence based literature.

The case study traces a paediatric patient's journey from presentation to the emergency department through to discharge and follow up. It considers some nursing interventions for the patient and family which may alleviate stress and suffering. Although many aspects of GBS remain a mystery, it is clear from this case study that timely diagnosis and quality nursing care can improve the experience of patients and families.

**Key words:** *Guillain-Barre Syndrome, plasmapheresis, immunoglobulin*

### Introduction

The child selected for this case study is referred to as Jacob (pseudonym) who was diagnosed with Guillain-Barre Syndrome (GBS). Jacob presented to the emergency department (ED) at the Children's Hospital with his parents. He was previously admitted to another hospital with a five day history of irritability. A detailed history of his illnesses over the past two months was obtained from his mother. This, combined with the physical examination and investigations, warranted admission to the neurosciences ward with suspected GBS.

Jacob is a 15month old boy who had reached normal developmental milestones and was reported as being previously healthy. Previous medical history did not suggest any autoimmune or neuromuscular disorders in any of the family members. Jacob was born full term with no complications, had no allergies and was not on any regular medications. He lives with his parents and sister and attends child care three days a week.

Jacob was brought to the ED by his parents because he was unwell on and off for the past two months with pharyngitis, otitis media

and upper respiratory infections which had been treated with oral cephalosporin. His last infection was three to four weeks ago. More than 50% of patients with GBS had an infection, mostly respiratory, within six weeks of the start of illness (Randall, 2010). Viral and bacterial infections, Hodgkin's disease and systemic lupus erythematosus were found to be some of the risk factors for GBS (Pluta, Lynn, & Golub, 2011).

Jacob was irritable two days prior to presentation, and his mother noticed swollen gums attributing this to tooth eruption. However, Jacob would scream when attempting to be placed in a sitting position. He also had intermittent colicky pain every hour lasting 15-45 minutes. He would become particularly distressed by nappy changes. In the three days prior to presentation to hospital, Jacob was lethargic, activity intolerant and preferred to lay supine. He was unable to weight bear. Sarkar, Menon, Sarbapali, & Pal (2011) suggest that in children the most common symptom of GBS is limb weakness. Some of the signs and symptoms are pain, limb weakness, ataxia, paraesthesia and sensory loss (Pluta, et al., 2011).

On admission to ED observations were normal for his age. Heart rate was 114 beats per minute, respirations were 25 breaths per minute, oxygen saturation 98% on room air. Temperature was 36.8°C. Neurological assessment was attended using modified paediatric Glasgow Coma Scale (GCS) (Wong & Hokenbury, 2003) and he scored 13/15.

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There is no specific diagnostic test available to confirm the diagnosis of GBS. Diagnosis is usually based on clinical appearance thorough history, combined with elimination of other neurological conditions (Randall, 2010). Jacob was admitted to another hospital at first, where investigations included bloods, abdominal ultrasound, urine, bilateral femur and hip X-rays. All investigations were found to be normal. He was febrile up to 40°C on original presentation. He was treated with intravenous fluids and antibiotics for suspected meningitis.

A lumbar puncture was performed under aseptic technique after obtaining written consent from Jacob's parents and he was found to have normal intracranial pressure (ICP). Cerebrospinal fluid (CSF) protein level was 4.24 g/L, which is high. Normal CSF protein is below 0.4g/L (The Royal Children's Hospital, Melbourne, 2013). CSF proteins are increased in the second week of the disease process (Khan, 2004). Lumbar puncture is performed to find the typical pattern of elevation in protein without raised monocytes (Randall, 2010).

Magnetic resonance imaging (MRI) of Jacob's brain was performed under general anaesthesia with no abnormalities detected. MRI of the spine showed cauda equina nerve root enhancement. This signifies inflammation consistent with the diagnosis of GBS. Anterior nerve root enhancement is more typical in GBS. (Gaillard & Singh, 2013).

Electromyographic (EMG) and nerve conduction studies (NCS) were not done. EMG and NCS are useful in determining the dangers of respiratory dysfunction (Durand, et al., 2006). The blood test will decide if the symptoms are triggered by another factor. An electrocardiogram (ECG) was performed to look for arrhythmias (Lugg, 2010), of which there were none. Jacob's results were reviewed by the Neurology Fellow and after being seen by the Paediatric Neurologist, Jacob was admitted to the Neurology ward with suspected GBS, and acute post inflammatory polyradiculoneuropathy with spinal involvement on the basis of the MRI report.

'GBS is a rare autoimmune neurological disease in which the body's immune system produces antibodies against its own nerves resulting in damage to them.' (Pluta, et al., 2011, p. 319) .The name derives from two French doctors who established and described this syndrome for the first time in two

soldiers during World War I. Worldwide incidence of GBS is 1.5/100,000 (Randall, 2010). The incidence of GBS in Australia is one to two cases per 100,000 annually (Khan, 2004). Out of all cases of GBS, 90% are seen in the Western world (Vucic, Kierman, & Cornblath, 2009). GBS is an acute inflammatory demyelination polyradiculoneuropathy which may lead to tetraparesis (Ghildiyal, Shradha, Iqbal, Varma, Smita, Manish & Tiwari 2012). Acute inflammatory demyelinating polyradiculoneuropathy is the most commonly recognised form in the Western world. However, Jacob is of Greek ethnicity. No ethnic prevalence occurs for GBS, however there is a male to female ratio of 1.5:1; male predominance is seen especially in older patients (Andary, 2012). GBS is infrequently detected in infancy and unusual in children less than two years of age (Bloch, Akhavan, & Avarello, 2013).

#### Types of GBS include:

- Acute inflammatory demyelinating polyneuropathy (AIDP) typically causing ascending paralysis.
- Acute motor axonal neuropathy (AMAN)
- Acute motor and sensory axonal neuropathy (AMSAN), which disturbs the motor features of the nervous system with good prognosis and is the more severe form of GBS (Bloch, et al., 2013)
- Miller Fisher Syndrome (MFS) - a rare variant of (GBS). It is described by external ophthalmoplegia, ataxia and areflexia. The incidence of MFS as a proportion of GBS was reported to be 1 - 5 % in Western countries and considerably higher in Eastern Asia - i.e. 19% in Taiwan and 25% in Japan (Das, Biswash, Karim, Nig Ahmed, Islam, Mandal, Abu & Sarkar 2012).

The actual cause of GBS is not known. 60% of patients had a history of upper respiratory infections, 27% of patients had no ongoing illness. The most commonly implicated infection is *Campylobacter jejuni* – one of the major causes of gastroenteritis (Hitt, Daleo, Stafstrom, Fitzgerald & Rose 2013). The bacteria or virus infection causes the body's immune system to attack the myelin sheath of the peripheral nerves causing nerve damage, resulting in defective sending of signals between nerves and muscles (Khan, 2004). Many studies attempted to show a link between vaccinations particularly influenza vac-

cine and GBS but this is still in dispute (Bloch, et al., 2013). Researchers from the UK report that the influenza vaccine does not escalate the danger of GBS (Boggs, 2014).

GBS is a post infectious immune-mediated ailment. Various notorious infectious mediators are believed to tempt creation of antibodies that cross-react with precise gangliosides and glycolipids that are spread throughout myelin in the peripheral nervous system. (Andary, 2012). GBS is an acute inflammatory demyelination polyradiculoneuropathy which may lead to tetraparesis (Ghildiyal, Shradha, Iqbal, Varma, Smita, Manish & Tiwari, 2012). In GBS, an immune-mediated reaction activates damage of the myelin sheath near the cranial and spinal nerves. The demyelination course is accompanied by oedema and inflammation of the peripheral nerves. Collection of lymphocytes and macrophages are apparently accountable for the definite stripping of the myelin from in between the nodes of Ranvier. The demyelination of axons results in loss of salutatory conduction. In addition the inflammation can end in axonal injury, which is linked to a decrease in fullness of muscle potential which indicates a lesser recovery. Demyelination is patchy and it arises gradually (Hickey, 2003).

### Nursing Care

Nursing care was focused on maintaining a patent airway, providing adequate nutrition and pain relief safely, regular neurological assessment, and addressing autonomic dysfunction. Another area of concern was parental anxiety. On admission to the ward Jacob was very irritable and crying when attended to by nursing staff. Jacob was observed to have weakness in all four limbs. Respiration, heart rate, ECG and oxygen saturations were continuously monitored. All observations, including GCS, were documented hourly. Respirations were assessed for quality, rate and he was observed for any abdominal breathing or cyanosis. Dysautonomia is a symptom of severe forms of GBS, which can cause life threatening cardiac arrhythmia (Doorn, 2008). Monitoring is of paramount importance especially to look for fatal complications such as respiratory failure requiring intubation, haemodynamic unsteadiness and autonomic dysfunctions (Bloch, et al., 2013). Jacob generally had a GCS of 14/15 with both pupils equal and reacting to light. The earliest sign of improvement or deterioration in neurological status is a change in level of consciousness, which is assessed by observ-

ing the child's response to the environment (Hockenbury, & Wilson, 2007). The GCS assesses eye opening, verbal response and motor response in order to obtain a score out of fifteen. A modified paediatric GCS identifies that verbal and motor responses must be related to the child's age in order to obtain an appropriate score (Hockenbury & Wilson, 2007). Neurological observations involve assessing pupils for size, shape, equality and reactivity to light (Crisp, 2009). Prompt acknowledgment of variation in GCS is vital (Hickey, 2003).

Constant description of all cares, investigations and the treatment plan were given to Jacob's family. As he was woken every hour, his family were given an explanation regarding the significance of repeated neurological assessments and other vital signs, which allow the nursing staff to observe the child's neurological status and intercede if any irregularities are identified. Regular communication and involvement between family and nursing staff can ensure feelings of safety and good care (De cort, 2011).

Parents were involved in the care provided. Both parents were distressed about the situation. However, nursing staff answered all their queries regarding every nursing intervention and explained to them the support networks available at the hospital with services including social work and chaplaincy. Jacob was started on intravenous immunoglobulin (IVIg) as per hospital policy after obtaining verbal consent from his parents. IVIg is known to block the receptors on macrophages avoiding an attack on the Schwann cells and myelin (Lugg, 2010). IVIg is simple to give and has fewer side effects with the end result similar to plasmapheresis. IVIg is now considered as the initial treatment of choice (Bloch, et al., 2013), commencing at 1 g / kg in two divided doses. It was revealed that IVIg has the possible chance to considerably minimise the morbidity and mortality (Pluta, et al., 2011). IVIg probably hastens the recovery compared to supportive therapy alone (Hughes, Swan & van Doorn 2012). If this fails, plasma exchange is recommended. Prednisolone 10 mg twice a day was started along with omeprazole 10 mg daily to counteract the potential gastrointestinal side effects of steroid therapy. Corticosteroids can be used as a combination therapy (Grant, Briscoe, Mezei, & Krassioukov, 2011).

An intensive care team review occurred in view of Jacob's increased work of breathing, dysautonomia and potential for respiratory failure, as phrenic nerve involvement was of concern. A blood gas was done, but there was no indication of carbon dioxide retention. GBS can weaken the phrenic nerve and cause respiratory failure (Grant, et al., 2011). Jacob was observed to have progressive weakness in his lower limbs and experienced immense pain particularly in his lower back. The pain team recommended treatment with gabapentin 5 mg / kg. Regular pain relief with paracetamol and ibuprofen continued in combination with occasional oxycodone.

Acknowledgement of pain is essential particularly in patients who are unable to converse with 89% of GBS patients experiencing severe pain (Doorn, 2008). Amlodipine 1 mg daily was commenced as Jacob developed hypertension. Autonomic involvement can cause blood pressure variations (Grant, et al., 2011). Weaning of prednisolone and increasing the dose of gabapentin were initiated gradually. Nursing measures were commenced to alleviate the distress and irritability. Treatment included supportive care and treating the immune response combined with the syndrome (Doorn, 2008). Many cases of paediatric GBS do not require intubation but one cannot foresee this without close observation over time (Sarkar, et al., 2011). Distraction techniques were used during all nursing interventions especially while obtaining blood pressure as he was quite irritable during that procedure. Once the pain had settled, the physiotherapist became involved and started gradual activity in discussion with his mother. Pain relief was initiated prior to the physiotherapy. Physical therapy for passive muscle movements avoids atrophy of muscles and contractures (Lugg, 2010).

Jacob's parents were educated about chewing and swallowing problems associated with GBS and initially nursing staff observed his eating and drinking. A review by the speech pathologist was carried out and this confirmed Jacob was able to swallow and chew safely. Swallowing is also to be carefully observed to determine any cranial nerve involvement and potential airway risk (Lugg, 2010). The dietician was involved due to Jacob's lack of oral intake. Insertion of a nasogastric tube was discussed, but Jacob's parents insisted on not inserting the tube as he had been through so much trauma and pain. Jacob's parents persisted with encour-

aging increased oral intake, in which they succeeded. Nursing staff strictly recorded the intake and output, monitoring bowel movements and providing laxatives when required. Jacob was discharged after a two week stay in hospital.

While critically analysing the nursing care provided to Jacob, it was noted that he did not receive his regular analgesia as his parents often declined due to the added stress of these interventions. It was evident that some of the nursing staff were not very knowledgeable regarding the disease process of GBS especially the intensity of pain. When this was recognised, nursing staff were provided with appropriate education. Parents were given explanations about the regular use of pain relief for better pain control, and support and encouragement was given at all times, especially before invasive procedures. This seemed to alleviate anxiety.

Another problem noted was regarding the measurement and documentation of blood pressure (BP). Sometimes a high BP reading was recorded because Jacob was crying and moving during the procedure. When this was reported to the Team Leader, nursing staff were advised to do a BP when Jacob was more relaxed. With regular physiotherapy Jacob's peripheral weakness improved steadily and he was able to gradually weight bear and walk while holding his parent's hand. His blood pressure also stabilised and he did not require antihypertensives on discharge. Outpatient physiotherapy was organised for him when discharged. On follow up with neurology team Jacob was able to walk unaided.

## Conclusion

The general prognosis for Jacob was good as 80% of patients will recover completely within three to six months. However GBS results in severe disability in 14% of patients annually. Loss of full strength, tenacious pain and requirement of professional modification occurs in 40% of patients due to the acquired disabilities. Mortality is about 4% (Rajabally, 2012). The fact that so many cases of GBS arise following a viral or bacterial infection advocates that definite characteristics of some virus and bacteria could trigger the immune system inappropriately. Certain proteins or peptides in viruses and bacteria may be similar to those seen in myelin, and the generation of antibodies to neutralise the conquering viruses or bacteria could produce

the attack on the myelin sheath Researchers are examining for those characteristics to learn how to stop this disorder and to make improved treatments accessible when it strikes (National Institute of Neurological Disorders and Stroke (NINDS)).

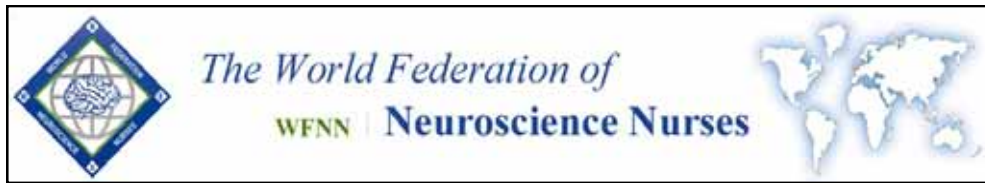
In conclusion after reviewing recent literature on GBS, it is agreed that much more research is needed to find out the exact cause of the disease, so that a vaccine to prevent GBS may be invented with time. Researchers are working for the development of superior management choices. It is reassuring that researchers found no proof of bigger risks regarding the influenza vaccine and GBS.

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There are a number of new initiatives that the new WFNN Executive have been discussing since the Japanese Congress last year. These include:

- Development of a members-only section of the website dedicated to professional development.
- Development of a topic-based online discussion group that will allow for meaningful exchanges worldwide.
- Production of a teaching video to demonstrate the techniques of neurological assessment with subtitling in as many languages as we can arrange.
- Webinars that allow nursing knowledge across the world.
- Development of a WFNN membership list so that promotion of events & surveying of members can be streamlined.

There are many ways that we can promote the financial health of the organisation including increasing available revenue through grant funding and holding additional educational events in non-congress years. We are actively working on grant funding and have explored the option of additional educational events such as:-

1. **Northern California Meeting:** (September 2015) - hosted by the University of California Davis, located in Sacramento. This would be a 2 day conference, with optional trips to the Napa Valley Vineyards and San Francisco.
2. **Hawaii Conference:** partnering with Queens Medical Center in Honolulu. This would be a 1.5 day conference to provide an update on neuroscience nursing topics such as TBI, Stroke, Neurodegenerative disease and skills clinic for neurological assessment.
3. **Partnering with Royal Caribbean Cruises:** potential for a 4 day cruise from Florida to the Bahamas with 1.5 days of conference time. A great way to combine business with vacation time.

The first event will be the Northern Californian meeting in September 2015. Final dates will be known soon and will be distributed to members through the ANNA & WFNN website. The aim of these additional events are to complement the WFNN Congress as well as local National meetings.

Cheers, Vicki

## Calendar of Events

### 2014:

- **NNPDSC Conference**  
Sydney  
[www.aci.health.nsw.gov.au](http://www.aci.health.nsw.gov.au)
- **BANN Conference**  
Palace Hotel  
Manchester, England  
Date: 17-18 October

### 2015:

- **European Association of Neuroscience Nurses Congress** Belgrade, Serbia.  
Dates: 13-16 May  
[www.eann2015.rs](http://www.eann2015.rs)
- **American Association of Neuroscience Nurses Conference** 21-24 March  
Nashville Convention Center,  
Nashville TN. USA .  
[www.aann.org](http://www.aann.org)
- **WFNN Interim Conference**  
Northern California Meeting  
September 2015 *TBC*  
[www.wfnn.org](http://www.wfnn.org)

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