SATURDAY 1 OCTOBER		
08:00-09:15	Setup, Registration & Coffee	
09.15-09.30	Welcome and Housekeeping	Joe Lynch, CEO, Parkinson's Association of Ireland
09:30-10:10	An Introduction to DBS – Overview	TBC
10:10-11:05	Theoretical considerations of programming the neuromodulator	Professor Erwin B Montgomery Jr.
11:05-11:25	Questions & Answers	
11:25-11:50	Coffee Break	
11:50-12:40	Practicalities of programming – the Frenchay Experience	Karen O'Sullivan
12:40-13:00	Questions & Answers	
13:00-14:00	Lunch	
14:00-14:45	An algorithmic approach to setting the neuromodulator parameters	Professor Erwin B Montgomery Jr.
14:45-15:00	Questions & Answers	
15:00-15:30	Coffee Break	
15:30-16:10	Two Presentations from People with the Experience of DBS	Ann Keilthy and Christine Nally
16.10-16.50	The work of the Dublin Neurological Institute	Dr Kinley Roberts
16:25-16:50	Panel Discussion and Questions & Answers	
16:50-17:45	Patient forum – DBS – Hopes and Realities Exhibition Viewing including Best Poster Award	

DEEP BRAIN STIMULATION CONFERENCE
Croke Park Conference Centre, Dublin
October 1, 2011

ABSTRACTS and SPEAKER CVs

Frenchay's Experience of Deep Brain Stimulation and Programming

Karen O'Sullivan
Frenchay Hospital, Bristol, UK

October 1st, 2011

Patients with Movement Disorders are referred to Frenchay Hospital for an opinion regarding their suitability for Deep Brain Stimulation (DBS). Referrals are made nationally and internationally. One of two consultant neurologists, with a special interest in Movement Disorders, reviews patients in outpatient's clinic. The consultant will decide whether their medication needs to be optimised. If it is already at an optimal level, funding is requested and an inpatient assessment arranged.

The pre-operative inpatient assessment is carried out over a duration of 3 days, using a holistic approach. On the day of admission, patient's anti-Parkinson's medications are omitted from 18.00 hours and even longer for long acting Dopamine Agonists. The

following day, an assessment is carried out using the Unified Parkinson's Disease Rating Scale (UPDRS), CAPSIT and Tinetti assessment tools. These are performed both in the "off" and "on" states. Patient's cognition is assessed using a Dementia Rating Scale (DRS2). We also establish whether patients are suffering with Dopamine Dysregulation Syndrome. Quality of life questionnaires, including PDQ39, SF36, Non Motor Symptom, Beck Anxiety and Depression questionnaires are completed. A 4 day record of their motor fluctuations is also documented. Once all of the evidence is gathered, the consultant neurologist decides whether the patient is suitable for DBS surgery. If suitable, they are put on the waiting list and have their surgery within 20 weeks. Patients are informed that DBS is not a miracle cure but can provide effective motor symptom control. It may take up to 1 year to achieve maximum symptom control.

For the surgical episode of care, patient's duration of stay is 5 days. Prof Gill and his research registrar plan the target area in the basal ganglia, the day after admission. Patients are given a general anaesthetic (GA) and while they are asleep, a stereotactic frame is attached to their skull and a 3 Tesla MRI scan performed. The target site is meticulously planned using neuroinspire, with millimetre precision. Patients wake up with the frame on, which remains on overnight. The following day, the DBS is inserted under GA. An intra-operative MRI scan verifies of target site. Patients are usually discharged 2-3 days post-operatively.

The DBS is not switched on immediately after surgery. A 3 month trial is taking place where the DBS is switched on and programmed 6-8 weeks after surgery, once the cerebral oedema has subsided and microlesioning effects wear off.

An outpatient appointment is arranged for patients to have their stimulators switched on 6-8 weeks following implantation of DBS. It is important that their anti-Parkinson's medication has been omitted that morning, ensuring that they are in the "off" state. A baseline assessment of tremor, bradykinesia and rigidity is made using the UPDRS rating scale, which is documented. Mobility, speech and dyskinesias are also assessed.

Programming Deep Brian Stimulators is based on the knowledge of the planning and intra-operative MRI scan, knowing the exact position of each individual electrode. The electrode is placed posterior to the Subthalamic Nucleus (STN), in the Zona Incerta (Zi). The middle 2 contacts are positioned in the centre of the target area, Zi. The main aims are to optimise clinical benefit and minimise adverse effects.

A systematic approach is used, using anatomical and physiological evidence. Stimulation parameters include electrode polarity, amplitude, pulse width and frequency. The electrode contacts can be programmed as anodes (+) or cathodes (-) for bipolar stimulation or as cathodes for monopolar stimulation against the generator case. Each electrode has 4 contacts and each contact is interrogated separately using monopolar settings, determining the efficacy and adverse effects. Initially, basic parameters are used where the pulse width is set at 60 milliseconds and a high frequency of 130 hertz.

Monopolar stimulation is usually the most effective type. However, if patients are experiencing adverse effects then bipolar stimulation may be more suitable, narrowing the spread of current. Adverse effects occur when the electric current is being spread outside the target area into the Internal Capsule. Side effects that patients may experience include parasthesia / pins and needles on the contralateral side of the body, muscle spasm in face and / or light headedness.
Once the electrode configuration has been established on the basic parameters, it may be necessary to adjust the width and frequency of the current. The pulse width is usually 60 - 90us. Patients with tremor predominant Parkinson's often require parameters where the pulse width is wider e.g. 120 – 360 us. If the pulse width is increased, the voltage is reduced. The frequency is usually 130 Hz but can vary from 130 -180 hz. If patients are suffering poor balance, the frequency may be reduced to 120-80 hz. The voltage can range from 1.5 - 3.5 volts. Excessive increases in voltage rarely add significant benefit. The polarity of the current may need to be reversed, where the negative and positive cathodes are swopped improving symptom control. Tripolar parameters (3 live contacts) may be used, but this is quite rare for Parkinson's patients. With tripolar settings the configuration can be 2 negative and 1 positive contacts of 2 positive and 1 negative contacts. This will elongate the spread of the electric current. All 4 contacts (quadripolar parameters) can also be activated. Some patients may benefit from a double monopolar configuration.

Therapeutic and individual impedance checks are carried out to ensure that there are no loose connections in the DBS system. These are done routinely post-operatively and pre and post generator replacements.

It is important to emphasise that everyone with PD is different and that everyone's tolerance to DBS is also different. It is effective symptom control for motor symptoms, not non motor symptoms such as anxiety and depression. 40 - 70% improvement in symptom control is achieved. Deep Brain Stimulation will improve motor symptoms to a similar extent as levodopa. If levodopa does not improve motor symptoms significantly, then DBS will not be appropriate.

Once the best DBS parameters have been established, the patient's medication is reviewed and titrated. Dopamine Agonists generally remain unchanged and levodopa may be reduced by 50%. Dopamine Agonists may be reduced or even discontinued if patients suffer with Dopamine Dysregulation Syndrome. If someone is on low doses of levodopa and having significant "off" time, then it may not be possible to reduce the levodopa. Following DBS surgery, patients experience less "off" time, more "on" time and levodopa can be reduced, reducing dyskinesias.
Patients are followed up routinely at 0.5, 1, 2 and 5 years as either in or outpatients and more frequently if necessary. It is important that they continue seeing their local neurologist.

- ABOUT THE SPEAKER

I'm Karen O'Sullivan and I trained at Addenbrooke's Hospital in Cambridge, where I developed a passion for neurosurgery having worked on an acute head injury unit.

In order to develop my knowledge and gain more experience, I undertook the ENB 148 course in Neurology and Neurosurgery at Atkinson Morley's Hospital in Wimbledon. Following the course, I worked on the Neuro-Intensive Care Unit and then a female Neurosurgery ward.

I have been working at Frenchay Hospital since 1980, where I have had various positions. Initially I have worked as a junior ward sister, senior ward sister and then as a senior nurse within the neurosurgery department.

In 1997, I became a Movement Disorder Nurse, the second post that was created in the country. The main movement disorder that I specialise in is Parkinson's disease but others include dystonia, essential tremor and cerebral palsy. I feel very passionate about my role, which I find both rewarding and challenging. I do my utmost to improve people's quality of life, wherever possible. I feel very privileged to work with such a dynamic and innovative Consultant Neurosurgeon as Prof Steven Gill. I also enjoy being an experienced member of the Movement Disorder Team at Frenchay Hospital. I have gained experience from patients, carers, colleagues at Frenchay Hospital, Medtronic, Parkinson's Disease Course in Cornwall and many national and international conferences

My role has evolved considerably, as the surgical procedures have advanced. I have witnessed patients having holes drilled in their skull under local anaesthetic, lesions made and dramatic improvements in their cardinal Parkinson's symptoms. Then unilateral lesions where made and unilateral DBS inserted, followed by bilateral DBS. In 2001, general anaesthesia came in being. Target areas have varied within the basal ganglia from the thalamus, globus pallidus interna, sub thalamic nucleus and more latterly the zona incerta.

I enjoy assessing patients' suitability for DBS and empowering them to manage their chronic neurological condition. A patient disposition is required when programming DBS's. It is important to treat every patient individually using a holistic approach, as every patient with Parkinson's disease is unique.

Theoretical considerations of programming the neuromodulator

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Dr. Sigmund Rosen Scholar in Neurology
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University of Alabama at Birmingham, USA

October 1st, 2011

Effective and efficient Deep Brain Stimulation (DBS) programming is challenging given the myriad of different combinations of active electrode configurations and stimulation parameters such as frequency, voltage or current, and pulse width. Experience demonstrates that there are two general approaches; the first is to try what others reported as the typical configurations and parameters with limited exploration of variations or the second, rationale selection based on anatomical and physiological principles, the latter being what some may describe as theoretical. These approaches have their analogue in the approaches to using medications. The first approach is to imitate the practice of mentors. The second approach is to use principles of pharmacokinetics and pharmacodynamics. The latter include concepts of route and time of absorption, location of action, volume of distribution, rate of metabolism and excretion. It is easy to appreciate that a medication whose half-life, the time it takes to eliminate half of what currently is in the body, is 2 hours is unlikely to be effective if given every 6 hours.

A principled approach to DBS first proceeds from the fundamental mechanism of action. Early in DBS, it was thought that therapeutic high frequency DBS inhibits the target structure. However, this inference followed from the Fallacy of Pseudo-transitivity which is of the form if a implies c and b implies c then a implies b. The logical error was that because high frequency DBS (premise a) produced the same clinical benefit (c) as prior destructive or ablative surgeries (b), then DBS implies reduced activity as would occur with ablation. Subsequent research clearly demonstrates that high frequency DBS excites neuronal elements at the stimulation site. The question then is what are the mechanisms that cause excitation and then how can those mechanisms be exploited for effective DBS.

The fundamental mechanism by which neurons cause physiological functions is by generation of action potentials (although in a few circumstance, it may be graded potentials). Release of neurotransmitters is not the proximate mechanism of DBS, as neurotransmitter release generally requires the production of action potentials. Generation of action potentials depends on opening ionic conductance channels, particularly voltage sensitive (voltage gated) ionic conductance channels. As a few channels open in response to a voltage change in the neuronal membrane (depolarization) the membrane voltage is further reduced causing even

more channels to open. This causes a chain-reaction-like effect that results in the action potential that is regenerates and travels along the neuronal element.

DBS must cause a change in membrane voltage of the neuron. The membrane voltage is generated by relatively greater positive ions outside the neuron and relatively greater negative ions inside the neuron. When the relative negativity of the inside of the neuron is reduced (called depolarizaton), channels, composed of protein subunits, sense the change in voltage and if the change is sufficient, the proteins reconfigure to create an open channel. For DBS, the most important channels are those that allow positive ions from outside the neuron to enter the neuron. This further reduces the negativity within the neuron that opens more channels that cause further reduction in the negativity and even more channels to open and creates the action potential.

The negativity inside the neuron is balanced by the positivity outside the neuron. If the positivity outside the neuron is reduced, then the negativity inside the neuron is reduced and the membrane depolarized. DBS causes a depolarizaton by reducing the positivity outside the neuron. It does so by injecting electrons that become negative ions, onto the surface of the neuron. The additional negative ions reduce the positivity outside the neuron and likewise reduce the negativity inside the neuron.

The key to controlling DBS is to control the number of electrons injected into the brain. The DBS has to “push” the electrons out of the DBS electrode. The amount of electrons pushed out is measured as microcoulombs and the amount of electrons pushed out per unit time, termed electrical current, is measured in microamps. It is the number of microcoulombs that is the critical factor. The voltage provides the force required to push the electrons out. However, the brain tissue resists the injection of electrons (called impedance). The amount of current depends on the voltage and impedance. Thus, for the same voltage, the current will drop by half if the impedance doubles. This complication is avoided if a constant-current implanted pulse generator (IPG) is used rather than a constant-voltage IPG.

The electrons injected by the DBS pulse will spread out from the electrodes. There will be fewer electrons (or negative ions) in the brain further away from the electrodes. The number of electrons (or negative ions) per unit brain tissue is called the current density. Thus, the size and shape of the distribution of the current densities is critical to determining how many neuronal elements will generate action potentials.

The size and shape of the current density distribution is controlled by the electrical current used (or the voltage divided by the impedance if a constant-voltage IPG is used) and the electrode configuration. Monopolar stimulation, where the DBS contact is negative (cathode) and the IPG case is positive (anode), provides a large though less intense distribution of current densities whereas bipolar (where the DBS lead contains both the negative and positive contacts) produces a smaller but more intense distribution of current densities. Pulse widths also are important because the number of microcoulombs injected depends on how long the electrical current is applied, that is the pulse width. Also, neuronal elements have different sensitivities to pulse widths. Large diameter neuronal elements require shorter pulse widths than small diameter neuronal elements.

Generally, DBS efficacy is related to stimulating sufficient numbers of the desired neuronal elements while avoiding stimulation of undesired neuronal elements. Many DBS targets have nearby structures which when unintentionally stimulated produces adverse effects. Thus, it is critical to understand the regional anatomy of the DBS targets.
The issue of DBS frequency is more problematic and does not relate to the effectiveness of activating neuronal elements directly. Rather, the effects of frequency appear to be dependent of the specific systems and symptoms being treated. Evidence to date suggests that the frequency dependence is a function of the network of oscillators being affected by the DBS particularly on the frequencies of the neuronal oscillators mediating the various symptoms.
As the number of DBS indications and targets expand, these general principles will continue to benefit the programmer. An algorithmic approach incorporating these principles is introduced in the abstract entitled “An algorithmic approach to setting the neuromodulator parameters”.

- *ABOUT THE SPEAKER*

Dr. Montgomery received his undergraduate degree in biochemistry (1972) and medical degree (1976) from the State University of New York at Buffalo. Subsequently, he completed an internship in internal medicine, residency in neurology, fellowship in movement disorders/neurophysiology and assistant professorship at Washington University in St. Louis. He then joined the Department of Neurology and the Program in Motor Control at the University of Arizona where he was an associate professor.

Dr. Montgomery joined the Department of Neurology at the Cleveland Clinic Foundation in Cleveland Ohio in 1997 to establish the clinical and research program in Deep Brain Stimulation (DBS). His interest in central nervous system physiology goes back to medical school where he studied cerebellar physiology with Dr. Gary Allen in the laboratories of Sir Johns Eccles. He re-focused on basal ganglia physiology and pathophysiology while at Washington University and that interest continues to the present.

Until 2009 when he joined the faculty at the University of Alabama at Birmingham, he studied the basal ganglia in the non-human primate model of Parkinson's disease. Over the last few years, his interest has shifted to exploiting DBS in humans to address basic,

translational and clinical research. He is now focusing primarily on the mechanisms of action of DBS, particularly as a probe to understand the physiology and pathophysiology of the basal ganglia-thalamic-cortical system.

Dr. Montgomery found that a thorough understanding of basic neuronal physiology greatly facilitated effective and efficient DBS programming, formalized in his book ***Deep Brain Stimulation: Principles and Practice, Oxford University Press, 2010.*** *He currently is the Dr. Sigmund Rosen Scholar in Neurology and Professor of Neurology at the University of Alabama at Birmingham. Dr. Montgomery has published over 80 peer reviewed articles and has received several research grants.*

An algorithmic approach to setting the neuromodulator parameters

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October 1st, 2011

An algorithm is simply as set of instructions with branch points for decision-making. One algorithm may be "do what you are doing if its working or do something different is it is not." The algorithmic approach presented here is based on a principled approach to DBS management. Those principles were introduced in the abstract "Theoretical considerations of programming the neuromodulator".

The key principle underlying this algorithmic approach is that efficacy is a function of stimulating sufficient neuronal elements, typically axon terminals and axons as these have the lowest threshold to stimulation, while avoiding stimulation of unintended neuronal elements that produce adverse effects. DBS efficacy can be pursued by increasing the number of neuronal elements using at least three different approaches. First, the stimulation electrical current (or voltage) can be increased. This will increase the current densities close to the electrode and extend the spatial extent of effective current densities. Second, pulse widths can be increased to activate smaller neuronal elements within the distribution of the current densities.
Third, bipolar stimulation (where the DBS lead has both negative [cathode] and positive [anode] contacts) produces a more intense, though smaller, distribution of current densities. In monopolar configurations, the current densities fall off as the square of the radius (r) from the electrode (~1/r2). In bipolar configuration, the current densities fall of as the cube of the radius (~1/r3). However, in bipolar configuration the current densities increase as the square of the distance (d) between the positive and negative contacts (~d2). Thus, wide bipolar creates a more intense distribution of current densities compared to narrow bipolar. This is the reason why DBS leads with more widely separated contacts are preferable.

The most neuronal elements will be activated with the largest and most intense distribution of current densities. The largest distributions can be accomplished by using monopolar configurations with maximal stimulation electrical currents (or voltages) and multiple negative contacts. Further, more neuronal elements will be activated within the distribution of current densities by using maximal pulse widths. However, maximal parameters increase the risk of stimulating unintended neuronal elements producing adverse effects. The effort then is to reduce the number of neuronal elements being stimulated, emphasizing the unintended neuronal elements, by reducing stimulation electrical current (or voltages), narrowing the pulse width, using single negative contacts, and using narrow bipolar configurations. Further, unnecessarily large stimulation parameters risk the premature exhaustion of the implanted pulse generator's (IPG's) battery.

The design of any algorithm should first emphasize efficacy with minimum adverse effects; however, some consideration of preserving the IPG battery life should be given. The key is the amount of electrons, measured in microcoulombs, that are removed from the battery. This is proportional to the electrical current (the microcouloumbs per second), the number of negative or positive contacts, the duration of electrical current measured in the pulse width, and the frequency at which the pulses are applied. In addition, the electronics of the IPGs is a factor. Most IPGs have to engage a separate electronic circuit when the stimulation voltage (in the case of constant-current IPGs, it is the current times the impedance) is greater than the battery voltage. Depending on the efficiency of the electronic circuit, stimulation voltages higher than the battery voltage will cause a greater drain on the battery. Unfortunately, some IPGs have battery voltages that are less than the typical stimulation voltages.

A doubling of the stimulation electrical current (or voltage), doubling of the number of positive or negative contacts, doubling the pulse width or doubling the frequency all will double the drainage from the battery, approximately. However, operationally, it is rare that one would double the frequency while it is not uncommon to double the pulse width. Thus, when titrating upwards to increase efficacy, it is generally preferable to start with monopolar or wide bipolar configurations. With respect to the stimulation parameters, it is preferable to change the stimulation current (or voltage) first and trying to keep the stimulation voltage less than the IPG battery voltage, then increase the frequency, and then increase the pulse width. Finally, if efficacy is still a problem, multiple negative contacts can be used.

To relieve adverse effects, one can move the distribution of current densities (the electrical field) away for the neuronal elements that, when stimulated, produce the adverse effects. This will depend greatly on the placement of the DBS lead and the individual's unique regional anatomy. The results from the intra-operative microelectrode recordings can provide some estimation of the individual's unique regional anatomy. Caution is advised in using the stereotactic coordinates and the various atlases for interpretation of the regional anatomy. There is considerable individual variation that is not captured in the atlases.

Another method for establishing the individual's unique regional anatomy is to perform a monopolar survey. Stimulation is conducted through each contact and the current (or voltage) is increase to some effect, whether improvement in symptoms or some adverse effect. From the patterns of adverse effects one can construct the patient's unique regional anatomy and many configurations can be excluded quickly.

An algorithm for DBS programming can be found at the following web site.

http://www.uab.edu/images/neurology/EBM/PDF/Algorithm%20and%20Checklist%20for%20electrode%20configuration%20Appendix%203.pdf

The algorithm contains two nested algorithms. There is an outer algorithm that is used to determine the electrode configurations and an internal algorithm for adjusting the stimulation parameters within a specific electrode configuration.

There are several advantages in strictly following an explicit algorithm of sufficient detail. First, the algorithm is efficient as it is based on physiological principles. Second, completing the algorithm ensures that potential therapeutic configurations and parameters are not missed. Third, it provides documentation in the event of some future problem or change in programmers; previously attempted ineffective configurations and parameters do not have to be repeated.

Any algorithm is only as good as its user. There may be the temptation to bypass certain steps or to give up prematurely and resort to the previous treatments. However, these previous treatments failed as if they worked the patient would not have had DBS surgery. Persistence is key.

Deep Brain Stimulation- the experience of the Dublin Neurological Institute

Dr. Kinley Roberts MRCPI
Neurology Registrar
Dublin Neurological Institute

Deep brain stimulation (DBS) is an effective neurosurgical procedure for idiopathic Parkinson's disease (IPD), essential tremor (ET) and primary dystonia[1, 2, 3]. It is a symptomatic, non-curative, elective procedure that involves the stereotactic surgical implantation of a battery-operated neurotransmitter into specific deep brain nuclei. DBS is thought to mediate its effects through stimulation of neural tracts and disruption of neural networks[4]. The ultimate aim of surgery is to decrease disability and improve the quality of life of the chronically ill patient. DBS is a lifelong therapy that requires lifelong maintenance and follow-up.

Multiple trials have demonstrated the effectiveness of DBS in the treatment of IPD and ET[1, 2]. Recently, DBS has been used in the treatment of primary dystonia[3], obsessive compulsive disorder[5], depression[6], Tourette's syndrome[7], and headache[8]. Huntington's disease[9], Alzheimer's disease[10] and morbid obesity[11] may also prove to be suitable for DBS in the near future.

Given the absence of a DBS program in Ireland, Irish patients are referred abroad to the UK, Europe and the United States. Unfortunately, patients have to travel repeatedly for pre and post-operative care in the years following their operation. Many of these patients experience increased levels of stress, anxiety and hardship as a result of these trips. The safe pre and post-operative care of these complex patients is compromised by the absence of such care in Ireland. Moreover, both the DBS surgery and subsequent post-operative care abroad incurs substantial costs to the state.

A designated DBS clinic takes place at the Dublin Neurological Institute (DNI) on a monthly basis. We provide pre-operative and post-operative care to patients who have had DBS surgery in either Ireland or abroad. Patients are often referred from other neurological centres for consideration of DBS as a suitable treatment option. These patients are then referred overseas. Patients with IPD, ET and dystonia are assessed by the multidisciplinary team in place including a consultant neurologist, neurology registrars, a DBS nurse specialist, the Medtronic® DBS clinical nurse specialist and a physiotherapist. In recent years we have referred some patients with more unusual conditions for DBS also, including Rapid Onset Dystonia Parkinsonism and Fragile X Tremor Ataxia Syndrome (FXTAS).

In 2006, a collaborative effort was started between the DNI, the Mater Private Hospital and the Walton Centre to develop DBS surgery in Ireland. The multidisciplinary team (MDT) consisted of a consultant neurologist (TL), an Irish consultant neurosurgeon with DBS training (GQ) and support from the functional neurosurgery unit at the Walton Centre (that had provided DBS training for GQ) by a consultant neurosurgeon (PE) and functional neurosurgical theatre sister at the Walton centre (PB) Preoperatively, patients were assessed by: a senior neuropsychologist (GF), the Medtronic® DBS clinical nurse specialist (DOB), a senior speech and language

therapist with expertise in IPD (FS), a specialist senior neuro-physiotherapist (EH), a consultant radiologist (EK), and a national expert in MRI imaging (PG). The MDT was assisted by specialists in neural engineering and implanted devcies (RR) and information technology (POS, SK).

Three patients were selected (2 IPD, 1 ET) suitable for DBS. Each patient was fitted with a Medtronic® deep brain stimulator which was activated on day 1 post-operatively.

This talk will focus on the outcome of the three Irish patients who underwent DBS in Ireland as well as the experience of some of our patients who have undergone the same procedure abroad.

Key

TL Professor Timothy Lynch GQ Mr. Gavin Quigley PE Mr. Paul Eldridge PB Ms. Patricia Beirne
GF Ms. Gillian Fortune DOB Ms. Deirdre O'Brien FS Ms. F. Shinkins EH Ms. Elaine Harrington
EK Mr. Eoin Kavanagh PG Mr. P Gilligan RR Mr. Richard Reilly POS Mr. Peadar O' Scolai SK S. Kelly

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- *ABOUT THE SPEAKER*

A graduate of Medicine in 2010, Dr. Roberts is Neurology Registrar at the Dublin Institute of Neurology.

- *PEOPLE WITH A NEUROLOGICAL DISORDER*

Christine Nally and Ann Keilthy detail their experiences of DBS for Dystonia and Parkinson's respectively.