

**INTERNATIONAL SOCIETY
FOR THE HISTORY OF THE NEUROSCIENCES
(ISHN)**

18th Annual Meeting

18-22 June 2013

Sydney University, Australia



INTERNATIONAL SOCIETY FOR THE HISTORY OF THE NEUROSCIENCES
Sydney, Australia, 18-22 June 2013

TUESDAY, 18 JUNE 2013

Lecture Theatre 101, New Law School Building, Eastern Avenue, University of Sydney

- 8:00-9:00 *Registration*
- 9:00-10:00 **OPENING SESSION** (chair: Paul Foley)
- Welcome**
- Cello recital: David Pereira** (see pp. 9-10)
- Max Bennett (Sydney) **Dendritic spines, neurofibrils and synapses in the 19th century**
- 10:00-10:30 **THE FRANK CLIFFORD ROSE MEMORIAL LECTURE**
- John Carmody (Sydney) **Seeking a neurobiological understanding of history: “a modest proposal”**
- 10:30-10:50 *Morning tea*
- 10:50-12:40 **MIND, VISION AND MOTION** (chair: François Boller)
- Stanley Finger (St. Louis, USA), Lawrence Kruger (Los Angeles, USA) **Before the *Thesaurus*: Peter Mark Roget, physician and scientist, and his forays into the neurosciences**
- J. Wayne Lazar (New York, USA) **Visual illustrations reflect developments in nineteenth-century neuroscience**
- Nicholas J. Wade (Dundee, UK), Frans A. J. Verstraten (Sydney) **Sigmund Exner and neural interpretations of motion phenomena**
- Lorenzo Lorusso (Chiari, Italy), Antonia Francesca Franchini (Milan, Italy), Bruno Falconi (Brescia, Italy), Alessandro Porro (Brescia, Italy) **Early motion picture industry and neurology**
- Simon C. Gandevia (Sydney) **Charles Darwin: An additional legacy**
- 12:40-13:00 **POSTER PRESENTATIONS** (chair: Sherry Ginn)
- Julien Bogousslavsky (Glion/Montreux, Switzerland), François Boller (Bethesda, USA) **Jean-Martin Charcot and art**
- Lorenzo Lorusso (Chiari, Italy), Bruno Falconi (Brescia, Italy), Antonia Francesca Franchini (Milan, Italy), Alessandro Porro (Brescia, Italy) **Neuroscientists in 19th century European caricature journals**

- 13:00-14:00 *Lunch (Journal of the History of the Neurosciences board meeting)*
- 14:00-14:40 **INVITED LECTURE** (chair: Hans Pols)
 Nicholas Rasmussen (Sydney) **Amphetamine, the first antidepressant, and the medical reasoning behind it 1930-1950**
- 14:40-15:40 **DIVERSE** (chair: Ian Steele-Russell)
 Jyh Yung Hor (Penang, Malaysia), Yih Chian Yew (Penang, Malaysia), Kazuo Fujihara (Sendai, Japan) **Devic's neuromyelitis optica: Changing concept in disease understanding 100 years later – from Devic's clinicopathological description to anti-aquaporin 4 antibody discovery and new pathological classification**
 Brian Freeman & John Carmody (Sydney) **Description versus experiment in autonomic development – cell migration as *deus ex machina* for the origin of sympathetic ganglia**
 Russell Johnson (Los Angeles) **The gain in pain: Acquiring, preserving, and using ephemera in the John C. Liebeskind History of Pain collection**
- 15:50-16:10 *Afternoon tea*
- 16:10-17:30 **EARLY NEUROSCIENCE** (chair: Lorenzo Lorusso)
 Jeremy C. Ganz (Ulverston, UK) **Edwin Smith Papyrus, Case 8: Ipsilateral hemiparesis – A different explanation**
 Jeremy C. Ganz (Ulverston, UK) **Herophilus and vivisection: Did it really happen?**
 Michael Besser (Sydney) **Galen and the origins of experimental neurology and neurosurgery**
 Gül Russell (Bryan, USA) **From Galenic “humoral function” to “anatomical structure”: illustrating the brain in Vesalius' *Fabrica***
- 17:30-19:00 **RECEPTION: Rare Books – Neuroscience** (*Exhibition Space, Fisher Library, Level 2*)

WEDNESDAY, 19 JUNE 2013

Lecture Theatre 101, New Law School Building, Eastern Avenue, University of Sydney

- 9:00-9:30 **THE CHRISTOPHER U. M. SMITH PRESIDENTIAL LECTURE** (chair: John Carmody)
 Catherine Storey (Sydney) **Queen Square – Finishing school for colonial neurologists**
- 9:30-10:50 **NEUROSCIENCE AND THE ARTS** (chair: Gül Russell)
 François Boller (Bethesda, USA), Anna Mazzucchi (Parma, Italy), Elena Sinforiani (Pavia, Italy) **Influence of aging and of focal lesions on the artistic production and creativity of famous painters**

Sherry Ginn (Concord, USA) “...**The mere action of nerves and brain**”: **Neurology, psychology, and neuroscience in fiction of Anglo-Irish author Sheridan Le Fanu**

Julien Bogousslavsky (Glion/Montreux, Switzerland), Laurent Tatu (Besançon, France), François Boller (Bethesda, USA) **The phantom limb phenomenon and the arts**

Lorenzo Lorusso (Chiari, Italy), Bruno Falconi (Brescia, Italy), Antonia Francesca Franchini (Milan, Italy), Alessandro Porro (Brescia, Italy) **Madness in comic opera**

10:50-11:10 *Morning tea*

11:10-11:40 **INVITED LECTURE** (chair: Yvonne Cossart)

David Burke (Sydney) **Microneurography and its introduction to Australia**

11:40-13:00 **NEUROSCIENCE IN RUSSIA** (chair: Paul Foley)

Yuri Zagvazdin (Fort Lauderdale, USA) **Meningitis in Russian literature: Myths and fears**

Boleslav Lichterman (Moscow, Russian Federation) **Surgical activity at Moscow Institute for Neurosurgery (1929-1941)**

Lilya A. Nazarova (Tashkent, Uzbekistan), Boleslav L. Lichterman (Moscow, Russian Federation), Olim Z. Akromov (Tashkent, Uzbekistan) **The role of neurologists in emergence of neurosurgery in Tashkent (1920-1943)**

Leonid Likhterman, Genrietta Chekhomova, Boleslav Lichterman (Moscow, Russian Federation) **First experience of the institution of the Museum of Moscow Institute for Neurosurgery**

13:00-14:00 *Lunch (International Society for the History of the Neurosciences board meeting)*

14:00-14:25 **INVITED LECTURE** (chair: Paul Foley)

Manuel Graeber (Sydney) **Alzheimer’s disease: History of the original histological slides**

14:25-15:25 **EXTENDED BOUNDARIES OF CLINICAL RESEARCH: GERMAN NEUROLOGY AND BIOLOGICAL PSYCHIATRY 1885 TO 1945** (chair: Frank Stahnisch & Paul Foley)

Ian Steele-Russell (College Station, USA) **The legacy of Johann Bernhard Aloys von Gudden, his contributions to neuroanatomy and psychiatry in the mid-nineteenth century**

Brian Freeman & John Carmody (Sydney) **Haeckel’s influence in developmental biology**

Brian Freeman & John Carmody (Sydney) **Re-evaluating the ethics of the dermatomal maps of the eminent German neurologist and neurosurgeon, Otrfrid Foerster**

- 15:25-15:35 *Afternoon tea*
- 15:35-16:45 **EXTENDED BOUNDARIES OF CLINICAL RESEARCH** (continued, followed by panel discussion)
- Frank Stahnisch (Calgary, Canada) **Eugenics ideals, racial anthropology and the emigration of German-American psychiatric geneticist Franz Josef Kallmann (1897-1965)**
- Paul Foley (Sydney) **Hugo Spatz and German neuropathology between the World Wars**
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- 18:00-19:30 **BEN HANEMAN MEMORIAL LECTURE 2013**
- Friends Room, Mitchell Library*
- Stanley Finger: **Benjamin Franklin and the birth of medical electricity**
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THURSDAY, 20 JUNE 2013

OLD QUARANTINE STATION, NORTH HEAD (see appendix for “How to get to QS”)

- 12:00-13:00 *Lunch: P27 – Governor Bourke Ballroom*
- 13:00-14:30 **INFECTIOUS DISEASE AND NEUROSCIENCE I** (chair: Yvonne Cossart)
- Alison Bashford (Sydney) **Historical background of the Quarantine Station**
- Jennifer Cooke (Sydney) **Cows, cannibals and kuru: how a crime reporter stumbled into the neurosciences**
- Warwick Britton (Sydney) **Leprosy**
- 14:30-15:00 *Afternoon tea*
- 15:00-16:30 **INFECTIOUS DISEASE AND NEUROSCIENCE II** (chair: Paul Lancaster)
- Paul Lancaster (Sydney) **Dr Millard and neoarsphenamine therapy for syphilis**
- Yvonne Cossart (Sydney) **Polio: a twentieth century phenomenon**
- Margaret Burgess (Sydney) **Rubella: its Australian connections**
- 17:00 Return to CBD/Camperdown

FRIDAY, 21 JUNE 2013

**ISHN-MOVEMENT DISORDERS SOCIETY JOINT SYMPOSIUM:
HISTORICAL ASPECTS OF MOVEMENT DISORDERS**

Lecture Theatre 101, New Law School Building, Eastern Avenue, University of Sydney

- 9:00-11:00 **MOVEMENT DISORDERS I** (chairs: C Storey and M Stern)
- Catherine Storey (Sydney) and CG Goetz (Chicago, USA) Welcome/overview of day
- Stanley Fahn (New York, USA) **Dystonia from Oppenheim to the present**
- Francisco Cardoso (Belo Horizonte, Brazil) **Chorea – emergence from olla podrida to movement disorder**
- Brandon Barton (Chicago, USA) **War-related injuries and movement disorders**
- John Steele (Guam) **Historical lessons from Guamanian PD/ALS/dementia**
- 11:00-11:30 *Morning tea*
- 11:30-13:00 **PARKINSON'S DISEASE** (chairs: CG Goetz and B Barton)
- Jennifer Goldman (Chicago) **Parkinson and Parkinson's disease**
- Paul Foley (Sydney) **The history of levodopa**
- Catherine Storey (Sydney) **Early stereotaxic strategies**
- 13:00-14:00 *Lunch*
- 14:00-15:00 **OTHER MOVEMENT DISORDERS** (chairs: F Cardoso and S Fahn)
- Martin Krause (Sydney) **Julius Hallervorden and the value of life**
- Padraic Grattan-Smith (Sydney) **Hystero-epilepsy: Professor Gamgee's account of his visit to the Salpêtrière 1878**
- 15:00-16:00 **ARCHIVAL FILMS OF MOVEMENT DISORDERS**
- Presented by G Aubert (Brussels, Belgium) and CG Goetz (Chicago)
- 16:00-16:20 *Afternoon tea*
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- 16:20-17:20 **ISHN GENERAL BUSINESS MEETING**
- 19:00 **ISHN ANNUAL DINNER** (*Imperial Peking at the Rocks*: see appendix for map)

SATURDAY, 22 JUNE 2013

**A CELEBRATION OF NEUROSCIENCE IN AUSTRALIA
AND NEW ZEALAND**

Claffy Lecture Theatre, Level 1, Centre Block, Sydney Hospital, Macquarie Street

- 9:00-11:00 **AUSTRALIA IN THE COMMONWEALTH** (chair: Catherine Storey)
- Catherine Storey (Sydney) **Murder on Macquarie Street: Revisiting the scene of the crime**
- John Carmody (Sydney) **“Temps perdu”: is it a “foreign country”? Fifty years since J. C. Eccles won the Nobel Prize**
- John W. Perram (Sydney) **How did Hodgkin and Huxley do their calculations?**
- Janet McCredie (Sydney) **The thalidomide story: History, neurology, and radiology**
- Ann Scott (Brisbane) **God and Magog: William Richard Gowers 1845-1915: the recruitment and career of ‘one of the greatest clinicians and teachers of clinical medicine in the nineteenth century’**
- A. D. (Sandy) Macleod (Canterbury, New Zealand) **Phylogenetic theories of conversion hysteria during the Great War**
- 11:00-11:20 *Morning tea*
- 11:20-13:00 **NEUROSCIENCE IN AUSTRALIA AND NEW ZEALAND: LOCAL ISSUES** (chair: Hans Pols)
- Neil E. Anderson (Auckland, New Zealand) **To include or not to include: the formation of a neurological society in New Zealand**
- Therese Alting (Sydney) **Huntington’s disease in Australia – “out of the darkness”**
- Richard White and Martin McGee-Collett (Sydney) **Prefrontal lobotomy in a Sydney teaching hospital, circa 1950**
- Paul A. L. Lancaster (Sydney) **Neuropathology from tropical Australia to Antarctica: John Burton Cleland**
- Ian Steele-Russell (College Station, USA) **Sir Grafton Elliot Smith: a neglected polymath**

SOLO CELLO RECITAL – DAVID PEREIRA

Program

1. JS Bach: *Sarabande* from Suite No 5 in C minor
2. Moya Henderson: *Sorry Time*
3. Peter Sculthorpe: *Threnody [In memoriam – Stuart Challenger, † 1991]*

The cello has been described as “the ultimate sensual instrument” and today’s music has been chosen to display that enticing, ruminative – even elegiac – quality of its music. Unsurprisingly, there is an important tradition of works for solo cello. Indeed, this grew out of an earlier tradition of music for solo *Viola da gamba*, where the great name is the French composer and performer, Marin Marais. In the cello repertoire, the enduring name, though, is JS Bach. His six solo *Suites* are not only marvellous works, but they are considered by cellists who accept their challenge as the “Everest” of the instrument.

It could also be said that they are similarly daunting for other composers who write for solo cello – one of the finest of whom was the German Max Reger. Interestingly, this challenge has, in recent years, greatly enticed a number of Australian composers and today we will hear some of the finest results of that attraction. In particular, a number of those works have been reflections on the appropriation, by the 1788 colonists, of the lands of the indigenous Australians.

It is appropriate, therefore, that we first offer homage to Bach *and* that this should be with the *Sarabande* from his *5th Suite* (in C minor). Bach builds the music with a five-note phrase and he reflects on its possibilities in a way which can put the listener in mind of someone considering a piece of sculpture from every possible aspect. “With its throbbing quavers, the *Sarabande* is like a lament from a cantata or a Passion,” as one performer has written of the music.

Moya Henderson is one of Australia’s most individual composers. The title of her short piece, *Sorry Time*, is an expression which is taken from the Australian Indigenous cultures and is used to signify a period of time set aside for grieving. The music grew out of an earlier work, *In Paradisum* (in four parts for unaccompanied choir), which was written in memory of two of her brothers, who had died a few years before *Sorry Time* was written. The cello piece falls into three main sections, each framed by ethereal passages of harmonics. There are moments of intense lyricism as well as more aggressive sections. It is a reflective piece: an expression of the grieving process evoking nostalgia, frustration and anger, emotional outbursts, remembering and final peace. It is dedicated to Diana Carmody.

In today’s context it carries an additional emotional burden. It was commissioned in 1999 by John Carmody who wanted to celebrate the graduation of the young cellist, Clare Rowe, from the Sydney Conservatorium. Her father, Mark Rowe, John Carmody’s great friend and colleague – further, a neuroscientist with a formidable international reputation -- was killed a couple of years ago in a tragic cycling accident.

TUESDAY: OPENING SESSION

Peter Sculthorpe's *Threnody* is also a lament and commemoration. In late 1991 the outstanding young Australian concert and operatic conductor, Stuart Challender, died of AIDS. *Threnody* is dedicated to his memory and was written for a Memorial Service which was held in the Sydney Town Hall, in Challender's honour, shortly after his death. David Pereira was the cellist on that occasion.

It, too, draws on an earlier piece: one of Sculthorpe's major orchestral works, *Kakadu*. This theme is, in the composer's words, "a free adaptation of an Aboriginal lament from Elcho Island, near Australia's northern coast, in the Arafura Sea." Although it is written in one continuous movement, the piece has four sections: *Cantando*, *Con malinconia*, *Risoluto* and *Con rassegnazione*. *Threnody* is widely considered to be one of the monuments of Australian instrumental music.

John Carmody

DAVID PEREIRA

David Pereira is one of Australia's most renowned cellists. He has led a number of our important orchestras and was a long-term member of the internationally-renowned Australia Ensemble (which was based at the University of NSW in Sydney). He has made several solo and chamber-music CDs, some of them featuring music which was especially composed for him. He has been a member of the staff of the Canberra School of Music for many years.

Dendritic spines, neurofibrils and synapses in the 19th century

M.R. Bennett

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Neurofibrils identified after staining, for example with Cajal's reduced silver nitrate (1), were thought by many senior histologists in the latter half of the 19th century to conduct action potentials (2). There was no basis for this popular idea, although it was the impetus for intense study of the 'neurofibrillar network' within neurons by Golgi, Cajal, Freud and others. The neurofibrils, shown with the introduction of ultrastructural techniques to be composed of groups of neurofilaments, were found traversing the entire neuron, soma and dendrites, as expected if they carried action potentials. Although it was Golgi who determined the true structure of dendrites it was Cajal who showed that the dendritic spine was not an artifact of fixation and that spines were the one part of the neuron which neurofibrils did not enter (3). However it was Berkley who first convincingly showed that varicosities and boutons of axon collaterals come into very close proximity with dendritic spines and to suggest that this constituted the principal cortical synapse type (4). This left the major problem, never addressed in the 19th century, of how the action potential was transmitted from neurofibrils in the terminal varicosities and boutons to the neurofibrils in the dendritic shaft, given that they were taken to be separated by the spine. The rich 19th century literature on neurofibrils now seems irrelevant when considering the conduction and transmission of action potentials but the question is still not answered concerning the spatial relationship between the principal filament type in the spines, actin, and the neurofilaments and microtubules of the dendritic shaft (5). We still do not know how the filaments within the spine gain a footing on the filament system of the dendrite shaft that allows for plasticity of the spine manifest in its growth and regression.

1. Potter HD (1971) The distribution of neurofibrils coextensive with microtubules and neurofilaments in dendrites and axons of the tectum, cerebellum and pallium of the frog. *Journal of Comparative Neurology* 143: 385-410.
2. Ramon y Cajal S (1904) *Histology of the Nervous System of Man and Vertebrates*. Vol. 1. Oxford University Press; p. 145.
3. *Ibid.* p. 150.
4. Berkley HJ (1895) Studies on the lesions produced by the action of certain poisons on the nerve-cell. *Medical News* (New York) 67:225-231.
5. Bennett MR, L Farnell and W Gibson (2011) A model of NMDA receptor control of F-actin treadmilling in synaptic spines and their growth. *Bulletin of Mathematical Biology* 73:2109-2131.

THE INAUGURAL FRANK CLIFFORD ROSE MEMORIAL LECTURE

Seeking a neurobiological understanding of history: “a modest proposal”

John Carmody

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History matters because memory matters: biological memory no less than psychological and social memory. We are, in several real senses, “made” by our history and may be destined to repeat at least our biological history, irrespective of whether we “remember” it or not. When, as individuals, we do forget our history, in dementia for example, the effects are diverse and catastrophic – it is the loss of our true personal and communal selves.

The younger generation may, fleetingly, believe otherwise, as it attempts to “kill off” its forbears in its efforts to build its own achievements. Their task is to secure the future, but an older generation with more restricted future understandably feels the imperative to reassemble and reflect upon the past. The ontological “turning point”, whenever it occurs, comes sooner than we realize, but the truth is that – quite apart from its pragmatic implications – we are intellectually impoverished (professionally and personally) if we do not understand the reasons for how we live and labor; or if, as scientists and clinicians, we do not understand why our intellectual advances were so hard-won, nor why they were considered so important when they achieved.

So, if rational medicine – or, as it is now glibly termed, “evidence-based medicine” – is a considered application of our prudent and informed selection from the daunting mass of “the literature”, then history is the ordering and evaluation of human memory. History “matters” profoundly because a person or a society deprived of or disrespectful towards its memory is hardly any longer properly human.

This truth ineluctably raises the nettlesome problem of consciousness. Memory and consciousness are intimately entwined in the human mind, but consciousness remains even more mysterious. Lack of understanding, however, has never diminished the importance of a phenomenon: it still matters. Not only does consciousness remain an intellectual mystery; so does how it is disrupted by anaesthetic and other drugs. In the present state of pharmacological knowledge we can say, at least, that the action of these agents must involve disruption of axonal or synaptic function, or both, most likely at sites on the membrane proteins which mediate either ionic conductance, ionic transport, transmitter release or synthesis – just as consciousness, itself, must be deeply associated with those properties. It is historical fact that 100 years ago we could not even this – in particular, membrane chemistry was far too primitive to allow anything more than speculation that the action of known anaesthetic agents was on membrane lipids. Yet that speculation was valuable and is a reminder that history can matter even when it is falsely understood. Without an understanding of that history, the anaesthetists, neurologists and psychiatrists are obliged to operate with a double handicap: the pharmacological and the conceptual. That may not greatly matter to the patient; to the thoughtful clinician, such an historical lacuna should matter a great deal, if only because an unreflective doctor will eventually become one of diminished skill.

Before the *Thesaurus*: Peter Mark Roget, physician and scientist, and his forays into the neurosciences

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Peter Mark Roget is best remembered today for his eponymic *Thesaurus*, a project completed late in his eventful life, after he had “retired.” Hardly remembered at all among historians of science is that he had trained as a physician, practiced medicine, was interested in many branches of the sciences, and approached medical and scientific issues with the mind of a philosopher. More than anything else, Roget’s life was dedicated to systematizing knowledge and helping to organize and run scholarly societies, the most notable being the Royal Society of London. He contributed to the neurosciences with short and long *Encyclopaedia Britannica* pieces, with his physiology books, with journal articles, and with many popular-press publications. In these venues he covered diverse topics, including phrenology, optics, aging, the sensory systems, and the nerves. Although not an empirical scientist or an experimentalist in the modern sense of the word, Roget even discovered a visual illusion of perceived movement that some motion picture historians have hailed as a significant first step on the path to movies.

Visual illustrations reflect developments in nineteenth-century neuroscience

J. Wayne Lazar

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David Ferrier displayed results of his earliest experiments on brain localization of function on line drawings of brains by John Galton in 1873 and on line drawings of brains by Ernest Waterlow in 1874/75. Ferrier turned the line drawings into brain maps by modifying both sets of drawings with circles that indicated unique places on the brains where electrical stimulation was effective in eliciting specific movements. Each circle represented the results of many stimulations of several animals. Nonetheless, the circles had different meanings in the two sets of brain maps. The circles in 1873 indicated “points” of effective stimulation. The circles in 1874/75 had much more meaning. They indicated areas of effective stimulation and represented two-dimensional graphs with only their centers as medians or places of central tendency.

John Bell, who flourished at the end of the eighteenth-and beginning nineteenth century, had figures that contrasted sharply with those of Ferrier. For Bell, the purpose of anatomical illustrations was to aid in dissection and surgery. He argued for realistic drawings—faithful reproductions with all the blood vessels and fascia intact. Bell argued against anatomically unnatural illustrations, explicitly deprecating composite drawings concocted from multiple sources placed together on a standard skeleton to represent an anatomically correct body.

Ferrier’s brain maps resemble Bell’s composites more than they resemble Bell’s drawings from nature; for Ferrier’s brain maps were, in fact, concocted out of multiple stimulations all represented by a circle on a “skeleton” now known as a line drawing. Bell’s realistic drawings were accepted at the beginning of the century while Ferrier’s brain maps were accepted at the end of the century. These comparisons merits attention because they reflect significant changes in acceptable medical illustration and in anatomy and physiology during the nineteenth century.

Sigmund Exner and neural interpretations of motion phenomena

Nicholas J. Wade^a and **Frans A. J. Verstraten**^b

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The waterfall illusion (or motion aftereffect) refers to the modification of motion perception following prolonged observation of a regularly moving stimulus, like descending water. It was described in antiquity but in the last century and a half it has been interpreted in neural terms. Sigmund Exner (1846-1926) added to the range of conditions under which motion perception can be modified and he provided a neural network model to account for the illusory motion. On the experimental side, he demonstrated that the motion aftereffect occurred in depth and that adaptation to linear motions in opposite directions (vertical and horizontal) yielded a diagonal aftereffect. He adapted each eye with opposite motion (both with rotation and with linear motion) and found no motion aftereffect when viewing a stationary test pattern with two eyes, but opposite ones when viewing with each eye separately. Exner's neural model was expressed in terms of influences on eye muscles, but it could be interpreted more generally. Prolonged stimulation in a given direction will lead to fatigue of certain cells but not others; this imbalance will be displayed when a stationary stimulus is subsequently viewed. Exner applied similar concepts to a wide range of motion phenomena, particularly stroboscopic and autokinetic motion. Using light sparks produced by electrical discharges he found that two slightly separated sparks, one appearing more than 50 ms after the other, appeared as a single light moving from one location to the other. On the basis of this observation, he contended that motion was a fundamental sensation that did not require combined elements of location and time. Exner's model of the motion aftereffect was the source of inspiration for many subsequent interpretations and the same logic is applied in recent neurophysiological theories involving adaptation of motion detectors in the visual system.

Early motion picture industry and neurology

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Cinema played an important role for the development of the biomedicine, and there was a particular relationship between the motion picture industry and the emerging neurosciences. Two important motion picture firms collaborated in the development of the application of cinematography in science: Pathé Frères in Paris, and the Lubin Manufacturing Company in Philadelphia.

In 1898 the Société Pathé Frères was founded by four brothers, Charles, Émile, Théophile and Jacques Pathé. Pathé became the most important film equipment and production company in the world, as well as a major producer of phonograph records. Another role played by this company was the establishment at the film studio in Vincennes of a small laboratory where different French neuroscientists worked. An important Pathé collaborator was Jean Comandon (1877-1970), who is considered a pioneer of medical microcinematography. Others physicians, including Paul Sainton (1868-1958) and André Thomas (1867-1963), were involved in the study of neurological disorders using Pathé's tools.

In the U.S.A., Philadelphia had the most representative domestic and international filmmaking empire: Lubin Manufacturing Company. The founder was Lubin Siegmund (1851-1923), who in 1910 began to collaborate with the neurologist Theodore H. Weisenburg (1876-1934) in the production of medical and scientific films.

These collaborations demonstrated the role of cinematography as a tool applied in neurology, and their technical and clinical improvement to have relied upon the best knowledge in both fields. In Philadelphia and in Paris, these pioneers found the best condition to develop an important instrument that has changed our cultural and scientific perceptions, an instrument that still plays an important role because it is based upon vision.

Charles Darwin: An additional legacy

Simon C. Gandevia

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Charles Darwin is best known for his work on evolution through natural selection, a concept presented jointly with Alfred Wallace to the Linnean Society in 1858. However Darwin's writing covers a prodigious array of biological and also geological topics. His creative approach is often lauded as one simply of induction, with his mind a 'machine for grinding general laws out of large collections of facts' (1). In reading Darwin's 'Autobiography' (1) and some of his best-known books, one is struck by his insightful observations not only about methodology and creativity in science but also about the unwitting cognitive biases to which all of us are prey. Many of these types of bias and error were documented by Daniel Kahneman and Amos Tversky (2) beginning in the 1970s, and they have been subsequently studied formally by many others. Darwin, often working (slowly) on several different projects at once, appears to have applied these insights in his approach. He was especially mindful of hindsight bias, observing that when he found evidence contrary to his view, he 'made a memorandum of it at once' because 'such facts and thoughts were far more apt to escape from memory than favourable ones'. Of course, other great scientists and thinkers have written shrewdly about key elements of scientific methods (e.g. Francis Bacon, Richard Feynman), but Darwin may be exceptional here because his insights were collected over many decades and translated overtly into his way of working.

1. Darwin C (1887) *The life and letters of Charles Darwin, an autobiographical chapter*. John Murray, London.
2. Tversky A and D Kahneman (1974) *Science* 185: 1124-1131.

Jean-Martin Charcot and art

Julien Bogousslavsky^a and François Boller^b

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Jean-Martin Charcot, the “father of neurology” in France and beyond, was also the man who established academic psychiatry in Paris, differentiating it from clinical alienism, which dominated medicine in the first three quarters of the nineteenth century. He was known to be both an authoritative and theatrical man, and while most of his present legacy belongs to classical neurology, his fame at the time was mainly due to his work on hysteria, which attracted the non-medical Parisian intelligentsia. In this field, he used artistic representations from previous centuries to illustrate the historical development of this condition, mainly with the help of his pupil Paul Richer, whose skills were such that he became a teacher at the *Beaux-Arts* school in Paris. Charcot himself liked to draw portraits (in particular sketches of colleagues during boring Faculty meetings and student examinations), self-caricatures, church sculptures, landscapes, soldiers, etc. He also used this gift in his work (histological or anatomic specimens, depictions of patients’ features and demeanor) under the influence of his colleague and friend Alfred Vulpian, the founder of modern neurophysiology in France. His most daring artistic experiment was to sketch while under the influence of hashish, but such attempts were not particularly unusual at the time, and Charles Lasègue and other physicians had done this before Charcot. Charcot’s tastes in art were very conventional and he had no connection with the avant-gardes of his time, including impressionism or realism. Indeed, Léon Daudet, son of Charcot’s former friend and famous writer Alphonse Daudet, described Charcot’s home as a pseudo-gothic kitsch accumulation of heteroclite pieces of furniture and materials. However, as Henry Meige wrote a few years after his mentor’s death, Charcot the artist remains “inseparable from Charcot the physician”.

Neuroscientists in 19th century European caricatural journals

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The development of printing techniques in Europe during the 19th century promoted the spread of journals that focused on the art of caricature. The press furthered the popularity of caricature that became hugely because it bridged the literate and illiterate public better than the written word. Caricatures were published on a regularly basis, and the illustrations interested people more than social themes. Great importance was given to the influence of medical cartoons, particularly with respect to laboratory and clinical neuroscience.

The earliest offerings were the French journals *La Caricature* (1830) and *Le Charivari* (1832), both founded by Charles Philipon (1800-1861). In the United Kingdom, *Punch*, or the “London Charivari”, was a weekly magazine of humour and satire established in 1841. The arrival of *Punch* coincided with one of the most important periods in the 19th century science. The magazine also documented the development of “traditional” and “alternative” scientific practices, including mesmerism, homeopathy and spiritualism. Another satirical English weekly magazine which appeared from 1868 was *Vanity Fair*. Colored caricatures were added to the magazine, and over 2000 such caricatures were printed, of which 53 were of physicians. In Germany, the most important satirical journals were *Kladderadatsch* (onomatopoeic for “crash”) in 1848, in Berlin, and *Simplicissimus* (1896) in Munich. In 1848, four satirical journals appeared in Italy: *Il Don Pirlone* (Rome), *L’Arlecchino* (“the Harlequin”; Naples), *Lo Spirito Folletto* (“the Elfin Spirit”; Milan), and *Il Fischietto* (“the Whistle”; Turin).

These magazines reflected the development and contradictions of medical specialists, and the emerging discipline of neuroscience and its protagonists.

TUESDAY: INVITED LECTURE

Amphetamine, the first antidepressant, and the medical reasoning behind it 1930-1950

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In this talk I will describe how amphetamine was discovered, tested, and introduced commercially as the first “antidepressant” medication in the late 1930s. I suggest that the success of this drug in medicine during the 1940s not only set the pattern for future psychiatric drug development, but played an unacknowledged role in the acceptance of neurotransmitter theory.

TUESDAY: DIVERSE

The gain in pain: Acquiring, preserving, and using ephemera in the John C. Liebeskind History of Pain collection

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In 1999, we introduced the John C. Liebeskind History of Pain Collection to ISHN as a resource for oral history interviews, organizational archives, and personal papers of individual researchers and clinicians. Since then the focus has evolved to capture and highlight printed ephemera, which present specific challenges for acquisition, preservation, access, and use. For many researchers, as well as librarians and archivists, the world of ephemera is uncharted territory; we will make it more familiar and accessible.

Devic's neuromyelitis optica: Changing concept in disease understanding 100 years later – from Devic's clinicopathological description to anti-aquaporin 4 antibody discovery and new pathological classification

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Devic's neuromyelitis optica (NMO) is an inflammatory disorder preferentially causing severe, bilateral optic neuritis and longitudinally extensive transverse myelitis.

In 1894, Eugene Devic in Lyon, France published a case report of a 45-year-old woman who developed acute bilateral blindness and paraplegia. Autopsy of the woman showed severe demyelination and necrosis of the optic nerves and spinal cord, with no brain lesion. Devic's student, Fernand Gault, collected 17 cases of similar conditions and published this as his doctoral thesis in that same year. The term "neuromyelitis optica" was coined in those publications. Being interpreted or misinterpreted by subsequent generations of neurologists to be a monophasic disorder, and without brain lesions, this disease concept of NMO has confused and perhaps misled neurologists for the next 100 years or so. In neurology textbooks, NMO was classified as demyelinating disease or as variant of multiple sclerosis.

In 2004, an autoantibody specific for NMO was reported, and the following year, this autoantibody was identified to be anti-aquaporin 4 (AQP4) antibody. Since this discovery, the disease spectrum of NMO has broadened. Limited forms of NMO, and those with specific brain lesions, that were seropositive for anti-AQP4 antibody, were recognized as NMO spectrum disorders. So-called opticospinal multiple sclerosis in Asia was also found to be NMO. Previously thought a monophasic disorder, clinical studies showed that relapse was in fact frequent in NMO.

Subsequent studies further revealed that the primary pathological event in NMO is astrocytic damage, with demyelination as a secondary event, and that anti-AQP4 antibody is pathogenic. As a result, NMO should thus be classified as an astrocytopathic disease targeting AQP4 rather than a demyelinating disease like multiple sclerosis.

This history of NMO illustrated that a century after the initial description, new clinical and experimental evidence can dramatically change our understanding of a unique neurologic disease.

Description versus experiment in autonomic development – cell migration as *deus ex machina* for the origin of sympathetic ganglia

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Since the 1840's there has been no unanimity on the origin of sympathetic ganglion cells: some believe that autonomic postganglionic neurones cells arise *in situ* from mesoderm, some think the ganglia are formed by cells migrating from the neural tube, and others think the source is migrating neural crest. When cell migration is claimed, speeds of movement are rarely calculated and no inertial reference frame is given. In the absence of a null-point, speeds of cell displacement during ontogeny, when the whole organism is growing rapidly, are arbitrary and do not provide evidence for active migration. Investigations of the normal development of the human embryo at the Anatomy Institute of Göttingen University, mainly from the 1940's onwards, provide no evidence for the independent migration of cells. Whole embryos were reconstructed rather than isolated regions or organs; growth movements were determined with reference frames for velocities. This kinetic analysis permits hypotheses about biomechanical forces involved in development. History teaches us that mechanics matured as a discipline long before chemistry, so it is just as logical to seek a biomechanical framework for development as a biochemical one. In any case, biomechanical hypotheses are not contradicted by findings from independent analyses of chemical (i.e., so-called molecular biological) events. Developmental biomechanics provides no evidence that neural crest cells, cortical neurones, melanocytes, germ cells, etc. migrate independently in a normal embryo. On the other hand, the outcomes of experimental manipulation of embryos are generally explained using the contrivance of cell migration. Observing the normal growing organism provides a firmer footing for interpreting development than interfering experimentally with it. The influential dogma, that an embryo has three germ layers each with cells of determined potential, is rendered less secure the more one investigates the origin of postganglionic sympathetic neurones.

Edwin Smith papyrus, case 8: Ipsilateral Hemiparesis - A different explanation

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The injury in case eight includes a skull defect, a deviation in the position of the eye, though the direction is not stated, and an ipsilateral hemiparesis.

Since the patient could walk in the presence of a skull defect, this would suggest a depressed fracture without generalised cerebral damage. There would seem to have been a concomitant skull base fracture as evidenced by bleeding from the nose and ears. Moreover, this bleeding indicates that the trauma was fresh. So what could be the mechanism of an ipsilateral paresis in an ambulant patient with a fresh injury and a skull defect?

The papyrus helps us some more. The sole is described as turned over with the tips of the toes contracted to the ball of the foot. Of the hand it is said “with the nails in the middle of his palm.” These are the descriptions of contractures and contractures do not develop until many weeks after a trauma induced hemiparesis.

One explanation of the ipsilateral hemiparesis has been a contra coup lesion. This is unlikely in the presence of an ambulant patient. A contra-coup occurs where there is diffuse cerebral damage and this would have to be extensive to produce a hemiparesis. With an ambulant patient this is unlikely. A newer explanation is that the patient had a swelling under the trauma which was compressing the brain stem against the opposite tentorial edge producing an ipsilateral paresis; a familiar situation. This is also unlikely for two reasons. Such a paresis would only occur in a deeply unconscious and not an ambulant patient. Moreover, the open skull defect makes such an explanation less likely.

It is suggested the hemiparesis was from some earlier cause unrelated to the trauma.

Herophilus and Vivisection: Did it really happen?

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The evidence that Herophilus of Alexandria (335-280 BCE) performed vivisection on condemned criminals is based on the writings Celsus (ca. 25 BCE - ca. 50 CE) and Tertullian (155-222). Celsus lived nearly 300 and Tertullian over 400 years after the events in question. Their sources are unclear. However, what makes Celsus so believable is his standing and the fact that he did not disapprove of the vivisection of criminals.

However, every surgeon knows a lightening anesthetic may be signaled by an increase in leakage of blood in the operating field. This will be more pronounced if the muscle relaxation is less adequate and the patient begins to breathe against the endotracheal tube. It seems clear from the writings of Galen, amongst others, that Herophilus dissected humans, but Galen does NOT state that they were alive at the time.

Criminals in general are not known for patience and altruism. Those condemned to death had no benefit from undergoing vivisection. One cannot but imagine that they would resist energetically. This would increase the difficulty of performing precise dissection. The inevitable extra bleeding would cover up the anatomy. This together with the noise from the suffering victim would make vivisection as a method for studying human anatomy a lot less effective than the dissection of a newly dead corpse. It is suggested it is more likely Herophilus dissected human corpses, itself a revolutionary departure from the norms of his time.

Nonetheless, there is no doubt that Herophilus has been widely believed to have performed vivisection and this has hindered the acceptance of his true achievements. Herophilus' influence on future generations has been damaged by the rumors about him. This has contributed to a delay in the acquisition of reliable anatomical knowledge until many centuries later.

Galen and the origins of experimental neurology and neurosurgery

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Galen (born AD 129) remains one of the major figures in the history of medicine which he dominated for 15 centuries. His vast output of writings, more of which survives than for any other ancient writer, are very wide ranging in scope, covering all aspects of theoretical and clinical medicine as well as significant areas of philosophy. Although a dogmatic authoritarian, he was a careful, accurate observer and original thinker. His public demonstrations of anatomy using vivisection in animals were astonishing demonstrations of Galen's skill and knowledge.

Galen made extraordinary advances in anatomy which were not challenged until the time of Vesalius. His dissections were of animals describing a wealth of accurate detail and the depth and range of his anatomical works attest to the crucial importance he placed on anatomy in his medical and philosophical world. As well as meticulous dissection, it was Galen's mindset as a medical scientist that gave him access to structural and functional physiology which he was then able to describe in exquisite detail.

Galen's experimentation on animals divulged the anatomy of the spinal cord, the sensory and motor nerves with their connection to the CNS, and the nerve supply to the larynx. His experimental methodology in vivisection work on animal brains, together with his observations on brain injured patients, laid the groundwork for the modern concept of brain localisation and foreshadowed brain stimulation in awake craniotomy.

Galen brought anatomy and physiology together in his study of the brain and nerves using experimental methodology which arguably represent the zenith of neurological investigation in antiquity. His description of his experiments on the exposed animal brain is remarkable.

From Galenic “humoral function” to “anatomical structure”: Illustrating the brain in Vesalius’ *Fabrica*

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During the Renaissance, anatomy was transformed from a tradition solely reliant upon the authority of the written word by incorporating veridical illustrations, as epitomized in Vesalius’ *De humani corporis fabrica* (Basel, 1543). This paper will explore in its historical context the question of how and why this change occurred. The roots of the change will be traced to the humanist revival of the classical tradition. Visual artists could now create exact likenesses of solid objects that “deceived the eye.” In anatomy, the human body could be studied directly through dissection, inspired (not suffocated) by the recovery of Galen’s anatomical writings. It will be shown that what made the change possible was the bringing together of the skills of both the anatomists and the artists in order to represent the dissected “fabric” of the human cadaver.

Earlier manuscripts and printed works had envisaged function within the Galenic humoral theory, “naming the parts” or mapping the “uses of the parts” for mnemonic purposes. With Vesalius, however, a new role was assigned to illustrations: specifically, the teaching of anatomy. The illustrations represented anatomical structures with increasing detail and accuracy, exemplified by those of the brain in both the *Fabrica* and the *Epitome*. Although not alone, Vesalius provided the first comprehensive and detailed description of how illustrations should serve anatomy by complementing direct observation in dissection.

His standpoint was not primarily the product of the need alone to provide a visual record to compensate the limited availability of cadavers. An analysis of Vesalius’ views between 1538, when he started lecturing in Padua, and the Preface to the first edition of the *Fabrica* provides evidence of a gradual development. It will be argued that his views emerged as a result of cumulative empirical experience. A comparative approach in dissection, using both human and animal preparations paved the way for observations of greater detail. This required the accurate representational skills of Renaissance artists to be finally conveyed through the medium of printing. With the *Fabrica*, morphology became divorced from humoral function, and enduring paradigms established that dominated until the nineteenth century.

THE INAUGURAL CHRISTOPHER U. M. SMITH PRESIDENTIAL LECTURE

Queen Square – Finishing school for Australian neurologists

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The Royal Australasian College of Physicians (RACP) was established in 1938. Prior to that time, any Australian aspiring to be a physician took the well-worn path back to the “mother-country” – the United Kingdom, to gain membership of the various Royal Colleges. For those who wished to practice as neurologists, that path invariably led to Queen Square, London.

In October 1950, with only four physicians who practiced exclusively as Neurologists in Australia (and all in Melbourne), the Australian Association of Neurologists (AAN) was formed. Young graduates of the post-war era embraced the neurosciences. With no opportunities to train on home soil, the majority of these young men sailed off the England, with the hope of gaining a place at Queen Square. Here they were assured of a wealth of clinical experience delivered by the ‘great’ names of Neurology.

In the early 1970s, the RACP changed the rules of membership and introduced a structured 3-year post-examination period of specialist training. By now there were excellent neurology departments throughout Australia that could offer comprehensive training. At the same time a position was set up at Queen Square to be filled annually by a nominee of the AAN (later ANZAN). This has resulted in a continuous link between Australian Neurology and New Zealand, and the National Hospital, Queen Square.

Influence of aging and of focal lesions on the artistic production and creativity of famous painters

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This presentation reviews the changes produced by age and by focal cerebral lesions on various aspects of artistic painting, particularly creativity and actual production. Aging in trained painters is often accompanied by a decline in creativity, which in turn is due to the cognitive decline related to aging. It has been argued, however, that aging does not cause a decline, but only changes in style and content. The two views are not mutually exclusive and we will present examples illustrating both aspects. We will also show that, in addition to cognitive changes, impairment of sensory organs, especially vision, and of the bones and joints, may also produce marked changes in an artist production and style. Examples will include Old Masters as well as contemporary painters.

Focal lesions such as strokes significantly affect painting and produce changes in painting style. This, however, varies according to the hemisphere affected. Painters with left hemisphere lesions would be expected to have major style changes because of the communicative disorder and right hemiparesis. Actually the changes are often relatively subtle. They usually find ways around their motor weakness. As for aphasia, there is surprisingly little correlation between its severity and a style change. Left hemisphere damaged painters mainly show an inability to represent tridimensionality, with a frequent evolution towards naïf art and expressive techniques involving decorative or geometric features.

Painters with right hemisphere lesions tend to show greater loss of depth and distance and also an impoverishment in the use of colors. Their main problem, however, is visuospatial in nature, leading to the neglect of the left side of the canvas. There is also evidence of constructional apraxia. These data lend support to the frequently stated view that the left hemisphere tends to more analytic while the right hemisphere mainly has synthetic abilities.

“...The mere action of nerves and brain”: Neurology, psychology, and neuroscience in fiction of Anglo-Irish author Sheridan Le Fanu

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Examination of the stories and novels of Anglo-Irish author Sheridan Le Fanu can be appreciated especially for the insights he provided into the psychology of his characters. However, his psychological thought should not be considered in the modern sense, as Le Fanu died in 1873 before psychology was “formally” established in 1879. His psychology is more reminiscent of the theories proposed during the early years of the 19th century with respect to the role of the ego and how it interacts with the non-ego. Le Fanu sought to show how a human being’s conscious perceptions could interact with their unconscious perceptions. The connection between these two aspects of the psyche could break following trauma, sickness, ingestion of drugs, or even from guilt.

A voracious reader, Le Fanu immersed himself in reading works related to the occult and magic as well as religion and theosophy. He was greatly influenced by the mystic-scientist Emanuel Swedenborg, a fact readily observed in much of Le Fanu’s fiction. A number of Le Fanu’s works indicate a belief that guilt or fear can manifest as a semi-independent existence capable of interacting with evil forces, of which his protagonists were unaware. In many respects this idea predates the concept of psychological dissociation as we know it today. At the time, it reinforced the prevailing view of evil, demonic forces at work, attempting to undermine man’s sanity.

Le Fanu’s short stories and novels also provide information about the emerging medical specialty of neurology; his characters frequently suffer from various “brain fevers” and other nervous maladies, such as hysteria, which were the subject of intense debate and analysis in England and continental Europe at the time. A superficial reading of Le Fanu’s fiction shows that he was familiar with early views about the actions of the nervous system. However, a more in-depth exploration indicates a man cognizant of contemporary scientific research along with a willingness to include the results of this research in his fiction. This presentation will examine several of Le Fanu’s short stories, correlating the behavior of each story’s protagonist with contemporary scientific knowledge.

The phantom limb phenomenon and the arts

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The term “phantom limb” was introduced by Silas Weir Mitchell in 1871, but the phenomenon had been noted earlier. We present two artists, a writer and a pianist, who, following amputation of their right arms, experienced a life-long phantom limb phenomenon, influencing the history of literature and music. They illustrate subtleties of the phenomenon, including lost limb perception, pain, and sensation of movement.

The life and works of Blaise Cendrars (1887-1961), one of the greatest French authors of the 20th century, were profoundly influenced by the phantom limb phenomenon. He was Swiss, but he enrolled in the French Foreign Legion at the onset of the First World War. Following shrapnel wounds, his right forearm was amputated, and he began to suffer from stump pain and phantom hand phenomena which persisted until his death. He became a left-hand author and wrote extensively about this, providing us with vivid descriptions of the life-long suffering that accompanies this condition.

Paul Wittgenstein (1887-1961) had been exposed to music since early age and was a trained pianist when in 1914 he was wounded in his right arm, which had to be amputated. Because of family ties and because he wanted to keep playing, many composers wrote music for him, including Benjamin Britten, Paul Hindemith, Sergei Prokofiev and Richard Strauss. Wittgenstein often indicated that he could “feel the playing movement of the fingers of his missing right hand”, and that it influenced and helped his left hand. Maurice Ravel wrote and dedicated to him his ‘Concerto for the Left Hand’ (1932), one of the masterpieces of 20th century music. Upon hearing Wittgenstein perform, Ravel was horrified not only by his poor technique but also because of unauthorized score changes. He only accepted having the Concerto played again publicly when he finally found a more suitable interpreter.

Madness in comic opera

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Representation of madness in opera is usually presented for dramatic purposes, especially in the 19th century when the madness caused by love was transformed into pathological madness by Gaetano Donizetti (1797-1848) in *Anna Bolena* and *Lucia di Lammermoor*, Vincenzo Bellini (1801-1835) in *Puritani*, and Alan Berg (1835-1935) in *Wozzeck*.

There had been comic interpolations in “opera seria”, and in the 18th century a new form of comic opera arose in southern Europe, especially in Naples: the characteristic of *Commedia dell’Arte*, presenting situations associated with ordinary people, closer to everyday life. A particular musical style was developed with an elaborate *cantabile* and melodic expressiveness that would spread throughout Europe.

The principal composers of *comic opera* included Francisco António de Almeida (c.1702-c.1755), Wolfgang Amadeus Mozart (1756-1791), Giovanni Paisiello (1740-1816), Domenico Cimarosa (1749-1801), Gaetano Donizetti and others who included in some of their operas the presentation of madness in comic situations, such as “*Spinalba*”, “*The Marriage of Figaro*”, “*Nina, ossia la pazza per amore*”, “*La finta malata*”, and “*Pazzi per progetto*”.

These musical pieces are a mirror of the popular conceptions of madness, based on scientific knowledge of the time and expressed by major composers.

Microneurography and its introduction to Australia

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Microneurography is an invasive physiological technique for recording the action potentials of peripheral nerve axons during natural activity in awake cooperative human subjects. It was developed in Sweden by Karl-Erik Hagbarth and Åke Vallbo, whose students (particularly Gunnar Wallin, Erik Torebjörk, Roland Johansson) popularised the technique which then spread to a number of countries, notably Germany, Japan, Australia, USA, France, Chile and Spain. The experimenter inserts an insulated tungsten microelectrode manually through the skin of an experimental volunteer to impale the underlying nerve trunk. The tip of the electrode enters a fascicle innervating muscle or skin, and is then manipulated to bring the desired neural activity into focus. Recordings can be made from single axons of large myelinated afferents or efferents, from unmyelinated C fibres (afferent or sympathetic efferent), and occasionally from small myelinated afferents, thermal (cold) and nociceptive (pricking pain).

The first studies in Australia followed a visit by Hagbarth in 1973, involved multi-unit recordings from cutaneous afferents in the median and radial nerves, and were supported by the first of many grants from the National Health & Medical Research Council (1974-1976). Single unit recordings did not occur until 1977. Since then Australians have published recordings from muscle afferents (in motor control studies), from cutaneous afferents (in studies of cutaneous sensation), from joint receptors (in studies of kinaesthesia) and from unmyelinated sympathetic efferent fibres innervating skin and joint, the latter from Melbourne as well as Sydney, often in association with Gunnar Wallin, one of the “second generation” of Swedish experts. There have been many notable firsts that have elucidated normal physiological function, but there have also been major insights into the pathophysiology of certain motor disorders and into the contribution of sympathetic nervous system activity to hypertension.

Meningitis in Russian literature: Myths and fears.

Yuri Zagvazdin

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Meningitis remains a devastating infection of the central nervous system and collects its deadly toll all over the globe despite availability of curative agents in the new millennium. The lack of effective remedy made ravages of its epidemic form in the United States during the 19th century so horrific that Dr. Andrew T. Still, the founder of the American osteopathic medicine, called it “whirlpool of death”. This “Charybdis” would not spare any continent, nationality or social class and left very distinct imprints of morbid fear in the minds of Russian people. These marks of fear are recognizable in several myths about the etiology of the malady. The most widespread of them is a belief that exposure to freezing air generates meningitis. The Russians also thought that the disease can be instigated by mental stress. The very same sentiment can be found in some Victorian novels where the condition appeared under the veil of the term “brain fever”. During that era, medical authorities commonly discussed extreme temperatures, sunstroke or emotional disturbances as probable causative factors for meningeal inflammation. By the beginning of the 20th century, these false assumptions vanished from the textbooks when the infectious nature of most cases of the disease became undisputable. Nevertheless, head covering in cold weather is still perceived as an important protective measure against meningitis in Russia and other countries of the former Soviet Union. Reflections of the illness by Dostoevsky, Paustovsky, Solzhenitsin and contemporary Russian writers in novels, short stories, newspaper articles, and even some items of fashion suggest that meningitis has a unique place in Russian society. The goal of this paper is to investigate how some of these reflections and the death of Semion Nadson, an outstanding Russian poet of the 19th century, possibly contributed to the perpetuation of myths about the causes of meningitis.

Surgical activity at Moscow Institute for Neurosurgery (1929-1941)

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A 25-bed neurosurgery clinic was opened in January 1929 at the Roentgen State Institute in Moscow under the directorship of Professors Nikolay Burdenko, a surgeon, and Vassily Kramer, a neurologist. A separate research neurosurgery institute was established there by special decree of the Soviet government (Sovnarkom) in October 1931. This was followed by a decree of Russian Ministry of Health (NKZ RSFSR) in January 1932. Professor Burdenko was appointed Institute director, and Dr. Efim Rossels and Prof. Kramer as his deputies. The Institute opened de facto in Spring 1934 as the Central Neurosurgery Institute. It got a separate four-storey building for 100 beds at ulitsa Ulyanovskaya.

The aim of this presentation is to analyze surgical activity at the neurosurgery clinic and Institute from 1929 to 1941.

It is based on archival sources from the Museum of the Burdenko Neurosurgery Institute, including surgical logbooks and reports on surgical activity, as well as published materials.

Results. According to the surgical logbooks, there was a more than a threefold increase in the number of surgeries (there were 120 operations in 1929 (not all of them were neurosurgical) and 369 in 1939). The mortality rate was high; for example, there were 34 cases of acoustic neurinoma operated within a six year period (from 1929 to 1935); 13 patients died after surgery. Of 28 patients operated on for cerebellar tumors, only 11 survived. A detailed analysis of surgical activity from 1929 until 1941 will be provided.

Conclusions. Although postoperative mortality at the Central Institute for Neurosurgery in the 1930s was high, major surgeries for CNS tumors were already performed there during this period.

The role of neurologists in emergence of neurosurgery in Tashkent (1920-1943)

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Introduction. The Turkestan University in Tashkent was established in 1920 by Russian professors sent from Moscow by the Soviet government. The university neurological clinic was headed by professor Mikhail Zakharchenko (1879-1953). His assistant Dmitry Bogorodinsky reported a series diagnosed cases of spinal tumors which were operated at surgical university clinics. Spinal cord tumors were the subject of Bogorodinsky's habilitation thesis defended in 1946. The first department of neurosurgery was opened in Tashkent in 1943 due to the influx of patients with head injuries into military hospitals located in Tashkent during the Second World War. The aim of this presentation is to trace neurosurgical activity in Tashkent from 1920 to 1943.

Material and methods. Case records of patients from a neurological clinic of the Tashkent medical institute (former medical faculty of Turkestan University) referred to surgical clinics from 1920 to 1943 were identified and analyzed (Central State Archive of Technical and Medical Documentation, F.96, op.96).

Results. During that period there were 1,381 patients at the neurology clinic; of these, 187 patients (13.5%) were referred to surgeons. This number gradually increased (22 patients in 1941). The most frequent diagnosis was suspicion of brain tumor (81 patients; 43.3%), followed by syringomyelia (39 cases; 20.8%), spinal tumors (37 cases; 19.7%), haematomyelia (11 cases; 5.9%), spondylolysis (5 cases; 2.7%), and head injury (4 cases; 2.1%). Fifty six cases were not operated for various reasons, and had been sent back to the neurology clinic; 17 of them died.

Conclusions. Neurosurgery in Tashkent emerged due to the cooperative efforts of Russian neurologists and surgeons.

First experience of the institution of the Museum of Moscow Institute for Neurosurgery

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The museum is the only one in Russia dedicated to neurosurgery history. It was opened in May 2002 and located on the first floor of a building built in art nouveau style for a boarding school at the turn of the 19th to the 20th centuries. The museum hall has 140 sq. m. Thanks to private sponsors it was possible to hire professional museum designers to construct the interior. The exposition is centered around a writing desk of one of the founders and the first director of the institute, Nikolai Burdenko (1876-1946), a famous Soviet surgeon. It is elevated above the floor and surrounded by transparent walls, thus resembling a Russian table glass. The outer side of the “table glass” is dedicated to a biography of Nikolai Burdenko and the early years of the Moscow Neurosurgery Institute (1929-1946). In front of the glass walls there are low glass stands for three-dimensional artifacts, including books and instruments from 1930s to the 1940s.

The post-Second World War story continues clockwise along the walls of the museum room. There are nine large arched glass stands on the podium for two-dimensional artifacts decorated with enlarged background reproductions of paintings and engravings of medical subjects from the 16th to 18th centuries. In front there are eighteen low glass stands for three-dimensional artifacts. Interspaces between the stands are occupied by busts of outstanding neurosurgeons of the Institute made of bronze, marble, or plaster of Paris. The exposition is divided into periods according to the directorship. The total number of displayed artifacts at the museum is 1100.

The museum is open to the public by appointment. There are guided tours for neurosurgery residents and participants in regular refreshment courses in neurosurgery. The museum organized several temporary exhibitions dedicated to jubilees of the institute employees, their participation in the Second World War, medical medals etc. Lectures on neurosurgery history are also delivered at the museum.

Alzheimer's disease: History of the original histological slides

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Alois Alzheimer was the first to describe neurofibrillary tangles, but amyloid plaques had been discovered earlier. Alzheimer nevertheless deserves to be called a pioneer because what he reported as an entity had not been recognised before. Quite fittingly, the genetic cause of his first case involves a prototypical Alzheimer gene, presenilin 1.

The discovery of the mutation marks the end of a 20 year long research project that would not have been possible without the following individuals: Kohshiro Fujisawa stimulated the original search; Henry deF. Webster mediated his contact with Munich; Parviz Mehraein had rescued archival material; Georg Kreutzberg's department assisted with reagents from Walther Spielmeyer's time; Richard Banati provided a crucial missing link; the enthusiasm of Asao Hirano upon seeing Johann F.'s slides triggered my renewed search for the sections of Auguste D.'s brain; and Ulrich Müller, in whose laboratory the mutation was identified. The promised accompanying online atlas of Alzheimer's well preserved and inspiring research material (1) has been delayed by years because I had to devote my time to matters of principal importance for human brain banking and the development of neuropathology (2). The atlas project, however, remains alive.

The rediscovered histological slides are still of practical value. They allow the conclusive rejection of ill-founded aetiological hypotheses that were published before the original slides had been rediscovered, e.g. Auguste D. representing a case of metachromatic leukodystrophy or of vascular dementia. Perhaps of even greater relevance today when looking at a prominent scientific trend, there is also no evidence of inflammation in the original cases of Alzheimer's disease.

Society changes slowly, but a few years into its second century, Alzheimer's disease is on its way to becoming the most important human disease due to its frequency and economic impact. In the years to come, it will likely serve as a driver for change in many areas of society.

1. <http://ibro.info/wp-content/uploads/2012/12/Alzheimer-Alois-2003.pdf>.
2. <http://blogs.plos.org/speakingofmedicine/2011/12/21/ensuring-respect-for-the-donated-brains-of-children>.

The legacy of Johann Bernhard Aloys von Gudden, his contributions to neuroanatomy and psychiatry in the mid-nineteenth century

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Von Gudden was born in Kleve, in lower Rhineland near the Dutch frontier, in 1824. In 1843, he began his studies in philosophy and medicine at the university in Bonn. For his doctoral dissertation, von Gudden studied torsional eye movement under the supervision of Volkman at Halle. He received his medical degree in 1848, and in the same year passed with distinction the state medical examination in Berlin. Thereafter, he obtained a position at the Siegburg asylum as an assistant under the supervision of Jacobi, one of the leading German psychiatrists. From 1851 to 1855, he worked with Roller in the Illenau asylum near Achern, the first modern psychiatric hospital in Germany. In 1855 he was appointed the director of Werneck, a newly established asylum in northern Bavaria. In October 1869, von Gudden became director of the newly founded Burghölzli psychiatric hospital in Zurich, Switzerland, and in 1870, was appointed co-editor of *Archiv für Psychiatrie und Nervenkrankheiten*. In 1872, he assumed the directorship of the Oberbayerische Kreis-Irrenanstalt in Munich, and subsequently became a full professor of psychiatry at the University of Munich.

Von Gudden devoted himself to the study of neuroanatomy, a rapidly developing science in the 19th century. He is perhaps best known for his studies on partial decussation of the optic paths, a subject that occupied him for around 30 years. His method of producing secondary atrophy of central structures after the removal of sense organs or cranial nerves in young animals ushered in a fresh advance in experimental neurology. In fully grown animals, from which eyes had been removed when they were young, he showed not only crossed and uncrossed optic fibres but also a supraoptic commissure and the transverse peduncular tract. Both of these tracts now bear his name. He was the first to describe the interpeduncular nucleus and the tegmental nuclei, known to all who work in the midbrain today as the dorsal and ventral nuclei of von Gudden.

As a psychiatrist he introduced the humanitarian principle of “no restraint” to German asylums. Although he did not attempt to develop a systematic nosology, he nevertheless founded an influential school of psychiatrists (including Forel, Kraepelin, and Nissl). For many years von Gudden served as consulting psychiatrist to the Bavarian royal family, and drowned together with his patient, King Ludwig II, whose sanity had been questioned.

In the presentation I shall place von Gudden’s double career in the perspective of German scientific and medical contributions in the nineteenth/twentieth centuries.

Haeckel's influence in developmental biology

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Ernst Haeckel (1834–1919) won't go away: recent hagiographic accounts of his work demand a re-examination of why a forger of scientific diagrams still exerts such an influence, e.g., as when he is acclaimed as "the father of evo-devo". The presentation will review Haeckel's embryological forgeries and discuss their sequelae. It will be shown that not everyone who challenges Haeckel is a religious zealot, a creationist, a believer in 'intelligent design', or is even necessarily attacking Darwin's legacy, impressions given by some who rebut criticisms of Haeckel. The fact is that Haeckel altered drawings of embryos by other scientists to suit his theories. Yet although the accuracy of Haeckel's embryology diagrams was questioned from the 1860's onwards and although Haeckel admitted his falsifications in 1908, it was not until the 1960's that Haeckel's claims (e.g., "... ova and embryos are, at certain periods of their development, all perfectly alike") and his so-called Biogenetic Law (ontogeny is an abbreviated recapitulation of phylogeny) could be tested scientifically. The large, standardised 3D reconstructions of embryos at the University of Göttingen were pivotal in testing and disproving Haeckel's Law with respect to human development. Nevertheless, Haeckel's preconceptions about recapitulation continue to influence the teaching of developmental biology and neuroscience with the use of inappropriate concepts and terms.

Re-evaluating the ethics of the dermatomal maps of the eminent German neurologist and neurosurgeon, Otfried Foerster

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Mapping of dermatomes in humans was carried out from about 1908 to 1926 by the pioneer Breslau (Wrocław) neurologist and neurosurgeon, Otfried Foerster, who sectioned dorsal roots in spastic patients; today his maps are ubiquitous.

As an undergraduate, Foerster impressed Heidenhain; later as protégé of Wernicke, he went to Paris to work under Dejerine and then with Marie and Babinski; he published a brain atlas with Wernicke in 1903. He admired English neuroscientists Hughlings Jackson and Sherrington and later developed a rapport with Cushing, and Penfield, who worked with him in Breslau, and with Fulton. In 1934 the Rockefeller Foundation helped construct his research institute. Despite these connections, virtually his entire output was published in German. A major exception was a long paper in *Brain* (1933, 56: 1-39), "The dermatomes in man", based on Foerster's Schorstein Lecture at London Hospital in 1932. The young South African surgeon and, later, Australian anatomist Dr Maurice 'Toby' Arnold was disturbed by what he read and saw in that paper ("wasted bodies") and heard in Foerster's Hughlings Jackson Centenary Lecture in London in 1935. He later told Australian colleagues that Foerster was evasive about "how he happened to come by so many conveniently spinally injured patients". In *Brain*, with brevity that would be entirely unacceptable today, Foerster simply wrote, "I need not discuss here the circumstances under which such a selected procedure may be undertaken". Other comments about Foerster (e.g. "Local anaesthesia, together with a dose of 'stern Prussian discipline', was used to keep the patient still" (1); "He completely disregarded physical pain [in himself]" (2); "He helped his patients, but they had to pay the price by being subjected to physiological experimentation"; (2)) suggest it is appropriate to review the ethics of his dermatomal mapping.

1. Tan T-C and PMcL Black (2001) The contributions of Otfried Foerster (1873–1941) to neurology and neurosurgery. *Neurosurgery* 49:1231-1236.
2. Wartenberg R (1953) Otfried Foerster (1873–1941). In: E Haymaker (ed) *Founders of Neurology*. CC Thomas, Springfield; pp. 422-425.

Eugenics ideals, racial anthropology and the emigration of German-American Psychiatric geneticist Franz Josef Kallmann (1897-1965)

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Biological psychiatry research during the first half of the 20th century had often been based in a number of interrelated disciplines, such as neurology, neuroanatomy and -pathology, as well as experimental biology. The German-American psychiatric geneticist Franz Joseph Kallmann is an example of a highly innovative and multidimensional researcher from clinical neuroscience, who functioned exceptionally well in both scientific cultures – early in the field of neuromorphology in Germany, as well as during his forced exile on the other side of the Atlantic – despite the marked differences in the contexts of scientific pursuit between Berlin and New York. Such innovative ideas, however could be an ambiguous advantage for émigré neuroscientists, since it might also easily lead to incommensurable scientific views, and sometimes marginalization in existing research programs.

Kallmann received his MD from the University of Breslau in 1919, and following his postgraduate training periods in Berlin with psychiatrist Karl Bonhoeffer and later the neuropathologist Hans Gerhard Creutzfeldt (1885-1964) Kallmann collaborated for a long period with Ernst Rüdin (1874-1952) at the German Research Institute for Psychiatry in Munich in investigations of sibling-inheritance of schizophrenia, becoming a pioneer in the field and protagonist of genetic research on psychiatric and neurodegenerative conditions. In late 1936, Kallmann emigrated to the US, where he worked for the New York State Psychiatric Institute, and published his hallmark study “The Genetics of Schizophrenia”, based on data he had assembled from the Berlin district pathological institutes of the city’s public health department. Kallmann resumed his role as an international player in psychiatry and genetics, for instance becoming co-founder (and later president: 1950/51) of the American Society of Human Genetics, and director of the NYSPI in 1955.

Despite such initial successes, however, there was an interesting twist in Kallmann’s emigration story: although Kallmann’s work had been well received by American and international geneticists, the idea of genetic differences hardly took hold in American psychiatry. To a large extent this was a consequence of the strong influence of another group of émigrés, the psychoanalysts, who dominated clinical psychiatry in the US until the 1960s. The psychoanalysts also established an important new cultural and philosophical direction, in which genetics played no significant role, and was often regarded as a dangerous form of knowledge in light of Nazi medical atrocities. Kallmann’s example, however, can be interpreted as a double fracture of the historical events. Medical scientists and clinicians were among the very social protagonists who pursued their research aims under the aegis of Nazi philosophies in the health care system, and they were also among the major theorists of racial hygiene, which largely subsumed medical genetics as a scientific foundation for the ideals and applications of eugenics.

Hugo Spatz and German neuropathology between the World Wars

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The German neuropathologist Hugo Spatz (1888-1969), who studied with both Kraepelin and Spielmeyer, is today best remembered for being one of the eponyms for Hallervorden-Spatz disease, a neurological condition he and his then new colleague and friend Julius Hallervorden described in 1922. Continued use of this disease name has been criticized in recent years, largely on the basis of the attitudes to so-called 'euthanasia' expressed by Hallervorden in an interview conducted by Leo Alexander at the end of the Second World War, as well as more recent investigation of the use of material from euthanased patients at the Berlin Institute for Brain Research, of which Spatz was the director between 1937 and 1945. This paper will present an overview of Spatz' highly productive career both before and after 1933, and will critically address the issue of whether judgements concerning his behavior during his directorship of the Institute for Brain Research have been overly severe.

Historical background of the Quarantine Station

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This paper introduces the 1000 or so inscriptions on the Quarantine Station site, made by internees from the 1830s through to the mid-1980s. An interdisciplinary team of archaeologists and historians are currently working to explore the context of these inscriptions, and their meaning as a form of mark-making.

Cows, cannibals and kuru: how a crime reporter stumbled into the neurosciences

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Advances in prion disease research have been greater in the last 20 years than in the preceding four decades. Spectacular events contributed: the rise and fall of the epidemic of bovine spongiform encephalopathy (BSE) and the official end of kuru, a mystifying disease that nearly wiped out cannibal tribes in the eastern highlands of Papua New Guinea.

Confined largely to scientists, researchers, anthropologists and neurological specialists, the world of prions, including Creutzfeldt-Jakob disease (CJD), began its leap from the cul-de-sac of science to the research superhighway in the early 1990s as reports of fears about BSE crossing the species barrier into humans appeared more frequently in the British press as the BSE epidemic escalated. On another front, articles and letters published in specialist medical journals marked the progress of iatrogenic CJD via corneal and dura mater grafts, stereotactic electrodes, neurosurgery and injections of human pituitary growth hormone and gonadotrophin. It took years for the implications to ripple across the disciplines.

Meanwhile, after a portentous phone call in November 1992, this reporter, more familiar with shootings, stabbings and a Royal Commission into drug trafficking, faced a steep learning curve in many things “ology”. Endocrinology, microbiology, forensic pathology, gynaecology and neurology were just some. As variant CJD emerged from 1996, there was much ado about animals like mice, chimpanzees, hamsters, macaque monkeys, elk, deer, mink, the odd moose and, of course, cattle. The result was many newspaper stories, a book, new friends and acquaintances and a post-graduate qualification in HPS.

Charting the twists and turns of the various prion disease epidemics has led to an interaction with the neurosciences that now enters its 21st year and this presentation. It’s like the Hotel California:

“You can check out any time you like, but you can never leave.”

Leprosy as a neurological disease

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Leprosy is a chronic disease of skin and peripheral nerve caused by infection with the slow-growing, intracellular pathogen *Mycobacterium leprae*. Until 1873 when Hansen discovered *M. leprae* in the tissues of leprosy patients, leprosy was considered a chronic neuro-degenerative condition with a possible genetic cause. Although *M. leprae* was the first bacterial pathogen associated with a human disease, the inability to grow *Mycobacterium leprae* culture has limited the study of the disease. There is wide range in clinical manifestations of leprosy and this confounded attempts at classification of the disease. In 1966 the landmark study of Ridley and Jopling demonstrated that leprosy was a spectrum disease in which variations in the immune response to the relatively inert *M. leprae* were responsible for the diverse manifestations of the disease. Over the last 30 years there has been rapid expansion in our understanding of the molecular and cellular basis of leprosy, including the sequencing of the *M. leprae* genome, the basis of drug resistance and the identity of the major antigens of *M. leprae*. In the case of the peripheral neuropathy, the receptors on Schwann cells for *M. leprae* have been defined, although the molecular basis for the tropism of *M. leprae* for Schwann cells is still unclear. The pattern of immunopathology and early manifestations of nerve damage have been identified. There have been considerable advances in the treatment of leprosy resulting in reduction of the prevalence of the disease; however there are still over 250,000 new patients per year. Over a third of these will develop nerve function impairment, often in the context of acute leprosy reactions. These leprosy reactions are caused by fluctuations in the immune response to the pathogen causing deterioration even whilst receiving effective antimicrobial therapy. The current therapies for nerve function impairment and leprosy reactions are inadequate, and there is the need to develop more effective interventions. Paradoxically, thalidomide, which is teratogenic because of its effect on peripheral nerves, was retained as therapeutic agent because of its efficacy in Erythema Nodosum Leprosum. The diagnosis of leprosy still has profound impact on patients, and understanding the social dimensions of leprosy is essential for the control and eventual eradication of this disease.

Salvarsan for syphilis in Sydney

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In the early 20th century, syphilis persisted as a significant clinical problem in Australia. Deaths from syphilis were highest in 1908-1910 (121 per million), then declined to 29 per million in 1946-1950. These deaths were attributed mainly to the predominant neurological diagnoses of tabes dorsalis (progressive locomotor ataxia) and general paralysis of the insane.

Two major clinical trials were conducted in Sydney using the new arsenical drug, compound 606 or Salvarsan, discovered in the laboratory of Paul Ehrlich in 1910. In a 16-month period in 1911-1912, 500 patients were treated at the infectious diseases Coast Hospital in Sydney. About one in four had tertiary syphilis. The majority of patients received two intravenous injections of Salvarsan. Evaluation of the effectiveness of treatment by the Wassermann reaction was hampered by concurrent use of mercury in many patients and by the difficulty of maintaining contact with patients. In the other clinical trial of Salvarsan on more than 350 patients treated in 1911-1912 at Royal Prince Alfred Hospital, most of the in-patients and out-patients had severe secondary or tertiary syphilis. Numerous patients had previously been treated unsuccessfully with mercury but usually did not receive this concurrently when Salvarsan was used. Individual case histories of patients with tabes indicated the benefits of this new treatment. Recurrences of syphilis were uncommon and usually due to relatively small initial doses of Salvarsan. The two clinicians who led the Salvarsan trials were Reginald Millard, Medical Superintendent of the Coast Hospital from 1908 to 1933, and Edmund Molesworth, Honorary Physician for Disease of the Skin at Royal Prince Alfred Hospital.

These clinical trials of the 'magic bullet' – *magische Kugel*, Ehrlich's term for an ideal therapeutic agent – in Sydney confirmed the efficacy of Salvarsan and marked the beginning of effective chemotherapy.

Polio: a twentieth century phenomenon

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Occasional cases of polio were seen in the ancient world, and still occur in unvaccinated countries , but a relentless rise in the number of cases in children of the mid-twentieth century western world spurred laboratory research into the nature of the infection and public health measures for support, treatment and ultimately control. Polio had a major impact on the development of scientific ideas and also on social history. The virus was discovered by Landsteiner who injected autopsy spinal cord material into the brains of rhesus monkeys. The natural history of the disease was elucidated by studying the disease in monkeys. The long term paralysis was explained by the inability of neurones to regenerate, but the exquisite specificity of the virus for motor neurones is still not fully understood. Therapeutic advances were slow to eventuate and heavy splinting and artificial respiration in “iron lung machines” were widely used, and widely criticised. After President Roosevelt was afflicted, funding for disease specific research was raised by public subscription to the “March of Dimes”. The cultivation of the virus in cell culture by Enders and his colleagues paved the way for vaccines. Controversy arose about the relative merits of attenuated and killed vaccines, and the Cutter incident invoked regulation of vaccine safety. Clinical trials in Third World countries where the virus, but not the disease, was common provoked outrage, but in the long term led to the global eradication campaign which is still proceeding. Three Nobel Prize winners Landsteiner, Burnet and Enders made landmark discoveries about poliomyelitis, but the disease is only mentioned in Enders’ citation.

Rubella: its Australian connections

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The view that rubella (German measles) was a disease of little importance was changed dramatically in 1941. In the spring and summer (September to February) of 1940 a severe epidemic of rubella occurred in Australia. Six months later Dr Norman Gregg noted that “an unusual number of cases of congenital cataracts made their appearance”. Prompted by the mothers’ histories of rubella in their pregnancies, Gregg reviewed his and his colleagues patients and published a paper describing 78 children with cataracts born in early 1941 with 68 of the mothers having experienced rubella in early pregnancy. The children frequently had other defects including microphthalmos, retinopathy, microcephaly, intrauterine growth retardation, congenital heart disease, developmental delay and spastic paresis. Later some were also found to have congenital nerve deafness. It took time for this clinical association to be accepted outside Australia and in 1944 an annotation in *The Lancet* stated that Gregg “cannot yet be said to have proved his case”. However, confirmation gradually appeared from other countries and in 1947 the *New England Journal of Medicine* drew world attention to the importance of the findings.

This clinical association was made before laboratory methods were developed for the identification of rubella virus in 1961. In 1964, there was another world wide outbreak of rubella. During this outbreak it was found that the virus was still present in the babies at birth and often for many months afterwards resulting in the development or progression of some defects such as cataracts, deafness, hepatitis and encephalitis after birth.

Rubella vaccines became available in 1969. Vaccination has led to the near elimination of rubella and congenital rubella in Western Europe, the Americas, Australia and New Zealand but has not yet been implemented in most low income and some middle income countries.

Dystonia from Oppenheim to the present

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Immediately after Hermann Oppenheim reported four Jewish children from Eastern Europe with what he termed *dystonia musculorum deformans*, neurologists around the world recognized the condition and began using this terminology. Dystonia was coined because Oppenheim was impressed with hypertonia on some occasions and hypotonia on others. Contemporary neurologists in Oppenheim's day, as neurologists today, were unimpressed with the alternating muscle tone and concluded that the twisting muscle contractions and spasms were the main clinical hallmarks of the disorder. From 1929 until the end of WWII, dystonia was considered a syndrome, secondary to encephalitis, Wilson disease, cerebral palsy, and other brain injuries, rather than a separate entity. Ernst Herz in a trilogy of papers published in 1944 resurrected dystonia as a distinct disease in its own right, and not just a syndrome secondary to an environmental brain insult. In 1975, C. David Marsden proposed that blepharospasm, spasmodic dysphonia, torticollis, and writer's cramp, were part of the dystonia spectrum and they became known as focal dystonia. In the following year the Dystonia Medical Research Foundation (DMRF) was founded by Sam and Fran Belzberg, and this foundation spurred research in all of the dystonias, including contracts and grants to initiate studies on epidemiology and genetics. These efforts were fruitful, and today many of the genetic forms of dystonia are labeled from DYT1 to DYT25. Progress in genetics has been especially rewarding; for example, in the past 12 months four gene mutations were reported. The definition and classification has changed over time as new progress in understanding dystonia has unfolded; the latest version is to be published in July 2013. Physiology of dystonia appears to be a combination of central inhibition, particularly surround inhibition, and increased plasticity. Treatment has improved with use of selective CNS-active medications, botulinum toxin injections into dystonic muscles, and stereotaxic surgery with stimulation of specific brain targets. New views of brain network and neurotransmitter involvement is currently being investigated, with basal ganglia, cerebral cortex and cerebellum considered as key areas. One can speculate that research related to the abnormal proteins generated from dystonic gene mutations will eventually shed light on the biochemical mechanisms of dystonia, from which new effective therapies will be discovered. Because gross and microscopic examination of the brain had not revealed neurodegeneration with nerve cell loss in the primary dystonias, the ability to reverse the symptoms and signs is highly likely at some point in the future.

Chorea – Emergence from *olla podrida* to movement disorder

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The first descriptions of chorea date back to the Middle Age when people in Europe celebrated the feast of Saint Vitus dancing before his statue. In the XVIth century Paracelsus coined the term chorea to describe the movements of those participating in the outbreaks of “dancing mania”. In 1687 Sydenham described the illness that would be named after him. Despite the accuracy of his description, he did not use the term chorea. In most of the medical literature of the XIXth century the term chorea was used without a precise definition of its meaning. This includes Charcot's writings, where there is no clear distinction between Sydenham's chorea and Huntington's disease (HD). Therefore the expression *olla podrida*, used by Olser, summarized well the imprecision behind the term chorea. He, along with George Huntington and William Gowers were responsible for establishing the concept that chorea was a unique movement disorder with a myriad of causes. The discovery of the HD gene in 1993 was a feat made possible by the discovery of a large number of patients in a remote area of Venezuela by Américo Negrette. A multinational team led by Nancy Wexler and James Gusella studied these individuals.

War-related injuries and movement disorders

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Since the beginning of recorded human civilization, the history of war has been inextricably tied to observations about neurological function. However it was not until the 20th century, with major improvements in survival rates, bigger wars with larger numbers of soldiers, improved neurosurgical treatments and more survivable brain insults, that meaningful observation and research about the acute and long-term effects of wartime neurological brain injuries has been possible. Basal ganglia injuries, movement disorders, and trauma have a controversial relationship: without a discrete lesion in the basal ganglia structures, the strength of association between a history of brain injury (traumatic or toxic) and the later development of a movement disorder such as Parkinson's disease is difficult to ascertain. Secondary movement disorders may appear similar to the primary/idiopathic forms of the disease, trauma may hasten or exacerbate the presence of idiopathic movement disorders, and while head trauma may increase the risk of neurodegenerative parkinsonism, epidemiological studies show mixed findings. The writings and discussions between historical figures regarding Parkinson's disease's relationship to war experiences shed light on this argument, a discussion that actively continues today. War experiences have also precipitated psychogenic movement disorders and unusual post-traumatic movement disorders of central and peripheral causes.

Historical lessons from Guamanian PD/ALS/dementia

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For more than two centuries, successive generations of Chamorro families on Guam and a few resident migrants of other ethnicity suffered a progressive and fatal neurodegenerative disease which was manifest in some siblings as ALS, in others as atypical parkinsonism, and in yet others as dementia. Its time of onset was variable, varying from adolescence to late senescence, and its latency after leaving Guam was as short as two years or as long as 54. It is also associated with a linear retinopathy, which may be a marker of etiology of a disease that occurs only on Guam and in patients with an identical disease who reside in the Kii peninsula of Japan.

We have learned that the disease Kurland and Hirano termed the ALS/Parkinsonism-dementia complex is a polyproteinopathy which includes all the abnormal proteins of each of the classical and universal degenerative disorders, including 3R and 4R tau, beta-amyloid, alpha-synuclein, TDP-43 and ubiquitin.

It is unlikely that this disease of two Pacific isolates is due to genetic inheritance, plant toxins, or geochemical mineral and metal abnormalities. But while we are certain what did not cause it, we have not yet learned what did.

The disease began to decline in those born after 1925 and ended with those born before 1951. But because the age in onset was delayed by latency, the annual incidence did not alter until 1955, and it has steadily declined since then as the age of onset increased. At the present time only dementia still occurs in very elderly subjects.

The disease Zimmerman called "an obscure malady" in 1945 is about to end.

Knowing the cause of this disease in these distant places and why it ended gives hope that other universal and related neurodegenerations could end in the same way, without therapy.

Parkinson and Parkinson's disease

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This session explores the life and times of James Parkinson (1755-1824). Although he may be best remembered in the medical profession for his *Essay on the Shaking Palsy* (1817) describing cases of paralysis agitans, Parkinson was also a prolific and respected writer on diverse subjects including politics, social reform, mental health, chemistry, and geology. His writings, whether scientific or political in nature, demonstrate his keen sense of observation, breadth of knowledge, and devotion to humanity. His six patient case series, published as the celebrated *Essay on the Shaking Palsy* and its relationship to past and current concepts of Parkinson's disease and its etiopathogenesis will be discussed. Parkinson outlined many of the motor and non-motor symptoms of this disorder that are recognized today. He proposed the medulla spinalis as the neuroanatomical site responsible for the shaking palsy, discussed symptomatic treatment options, and even suggested the need for neuroprotective therapies. Clinico-pathological correlations were lacking at the time of the *Essay* and did not emerge until later in the 19th century. Work of Charcot and his pupils, Ordenstein and Brissaud, allowed for further clinical characterization of paralysis agitans, the coining of the eponym Parkinson's disease, and early neuropathological investigations.

The history of levodopa

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The 1960 discovery of the striatal dopamine deficit caused by degeneration of the substantia nigra, ultimately resulting in reduced thalamic activation of the cortex, remains the major watershed in the history of therapy for parkinsonism. The importance of nigral degeneration had been first clearly identified in 1919 and recognized by the late 1930s, but this initially had no impact upon concepts related to the pathophysiology and therapy of parkinsonism. It was only in 1957 that Swedish pharmacologist Arvid Carlsson found that the listlessness induced by reserpine-induced catecholamine depletion in rabbits could be reversed by restoration of CNS dopamine levels through administration of the catecholamine precursor L-DOPA; he concluded that a dopamine deficiency might also underlie parkinsonian akinesia, for which there was no treatment. In Vienna, pharmacologist Oleh Hornykiewicz discovered that dopamine levels were indeed reduced markedly in the basal ganglia of parkinsonian patients, leading to the initiation of a daring human experiment in 1961: neurologist Walter Birkmayer intravenously administered L-DOPA to a severely afflicted parkinsonian patient, with results that exceeded expectations, ultimately leading to the installation of L-DOPA as the 'gold standard' medication for parkinsonism. The path leading to this discovery was, however, not as linear as it appears in retrospect, with a number of curious side stories and issues enriching the tale.

Early stereotactic strategies

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James Parkinson described the clinical features of his eponymous disease in 1817. A century later there were still only limited medical options available to treat this disease.

In this environ, surgical opportunities were explored. In 1908 Horsley and Clarke described the apparatus that made possible stereotactic operations of deeper cortical structures in experimental animals. It would take over forty years for this to be a possibility in humans. To make such operations a reality there needed to be a method to image intracranial landmarks and a means to identify and target these structures with accuracy. With the introduction of the pneumoencephalogram (PEG) and published atlases of the human brain, the stage was set for stereotactic surgery for Parkinson's disease and other movement disorders.

Nashold in 1970 estimated that over 40,000 stereotactic operations had been performed during the previous 10 years. These procedures were performed in all major centres throughout the world and Sydney, Australia was no exception. In this presentation I will report on the series from the Royal North Shore Hospital of Sydney of 413 operations performed during the period 1958-1965.

With the introduction of L-Dopa in the mid 1960's these surgical procedures lost favor; the instruments were packed away; this epoch of Parkinson management was all but forgotten.

Julius Hallervorden and the value of life

Martin Krause

Royal North Shore Hospital & University of Sydney

'I heard that they were going to do that, and so I went up to them and told them "Look here now, boys, if you are going to kill all these people, at least take the brains out so that the material could be utilized. They asked then 'how many can you examine?' I said an unlimited number – the more the better.'

These were Julius Hallervorden's words as reported by himself to Major Leo Alexander (US Military document L-170).

Julius Hallervorden – psychiatrist, neurologist and neuropathologist – was prosector and director of the histopathology department of the Kaiser Wilhelm Institute in Berlin in the late thirties and early forties of the previous century. He was the scientist after whom the Hallervorden-Spatz disease is named, he was a close friend of the German-Jewish colleagues Ludwig Pick and Max Bielschowsky whom he helped during the Third Reich, respected by his colleagues, free of envy, collegial and described as "lovable, helpful, always embraced the worries of others, young at heart, overwhelmingly joyful, open minded about the beauty of life" (H.W. Pia 1969).

He collected 697 brains of children and adolescents who were murdered as part of the Euthanasia program in Germany from 1939 to 1942.

Was he a ruthless opportunist, an abettor of murder, or a scientist "whose research was outside the realm of politics or humanitarian system of values" (v. Platten-Hallermund)?

Hystero-epilepsy: Professor Gamgee's account of his visit to the Salpêtrière 1878

Padraic Grattan-Smith

Sydney

On August 23 and 24 1878 Arthur Gamgee, Brackenbury Professor of Physiology in Owens College Manchester, visited the Salpêtrière in the company of a group of distinguished figures in medicine and science including Virchow. During the visit the features of Hystero-Epilepsy were demonstrated by Professor Charcot in person. The visitors were shown dramatic seizures that could be precipitated by gripping the skin of the breasts on both sides and stopped by ovarian compression. The effects of mesmerism were demonstrated as well as the reversal of signs such as hemianaesthesia and colour blindness by the actions of magnets and solenoids. Professor Gamgee himself easily induced a trance in one of these patients. Professor Gamgee provided a narrative of these events in the British Medical Journal of the same year. The talk will focus on this paper which illustrates the reactions of Gamgee, a physiologist but also an experienced physician, to these extraordinary phenomena.

Archival films of movement disorders

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Cinematography was developed in the late nineteenth century and rapidly embraced by neurologists to capture the various neurological signs and syndromes in the growing nosology of the field. The technique was particularly pertinent to movement disorders, because the changing and varied movements of these diagnoses could not be captured well in traditional photographic techniques. The Movement Disorder Society and other archival sources provide access to a large collection of early films that represent the scientific work of such luminaries as Marinescu, Van Gehuchten, Herz, Birkmayer and Putnam. Movement disorders, captured by creative techniques and using a variety of props and methods, included Parkinson's disease, dystonia, choreas, and tremors. Used for medical documentation and education, these films provide a rich archive of disorders in their natural state before current therapies became available and demonstrate early treatment strategies.

Murder on Macquarie Street, revisited

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At the 2010 meeting of this Society I presented a paper entitled “Slight case of murder in Macquarie Street”. Now that our Society is holding its annual meeting here on Macquarie Street, I am taking the opportunity to revisit the scene of the crime.

The original headline appeared as a leading article in the Medical Journal of Australia on the 29th April 1985. The victim was the Kanematsu Institute which once stood in the grounds of Sydney Hospital; the perpetrator, the NSW State Government. The real loss had occurred many years before with the departure of an extraordinary team of neuroscientists who had formed a scientific collaboration in the 1930s. The collaboration had included two future Nobel Prize winners, Sir John Eccles and Sir Bernard Katz, and Stephen Kuffler, later one of America’s foremost research neurophysiologists. This heyday of neuroscience in Sydney is now all but forgotten and a distant memory to most.

Some of these memories will be revived.

“Temps perdu”: is it a “foreign country”? Fifty years since JC Eccles won the Nobel prize

John Carmody

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Australia is a pragmatic country which does not really value intellectual achievement, finding its heroes, rather, in sporting events. So the 10 Nobel Australian Prizes make barely a ripple on the national consciousness: the 50th anniversary of the award to the neuroscientist, John Eccles, has passed unnoticed.

After graduating in medicine from the University of Melbourne, he went to Oxford with a Rhodes Scholarship to work with Charles Sherrington – “the one man in the world whom I wished to have as my master” (1) -- driven to understand the relation of mind and brain. In some respects he was destined to remain more honoured outside his native country than within it. Yet his *scientific* influence in Australia has been profound.

Going abroad for post-graduate study was the Australian norm in those inter-war years. Even when he returned in the late 1930s to become the Director of the Kanematsu Institute in Sydney, he and his colleagues were decidedly less respected than the clinicians there. His posts during what he called his “Scientific Odyssey” – in Oxford, Sydney, Dunedin, Canberra, Chicago and Buffalo – demonstrate his internationalist outlook, something which was of enormous significance during his years as foundation Professor of Physiology at the Australian National University (1951- 1966). During that time he made the John Curtin School an international centre of excellence: overwhelmingly, his PhD students returned to become major neuroscientific influences in their home countries; that pattern continued during his years as Distinguished Professor in Buffalo (1968-1975).

Though he left Australia in 1966, his enduring legacy remains the strength of neuroscience in this country. It is that scientific inheritance – as well as the extraordinary number and calibre of his own scientific attainments – which his native country should celebrate in 2013.

1. Cited by Curtis DR and P Andersen (2001) John Carew Eccles 1903-1997. *Historical Records of Australian Science* 13: 439-473

How did Hodgkin and Huxley do their calculations?

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The classical paper by Hodgkin and Huxley (*J. Physiology* (1952) 117: 500-544) is epoch-making from a couple of points of view. Firstly, their relatively simple dynamical system modelling of the flow of ions when axons are depolarized is able to predict in extraordinary detail all the main features of that depolarization, including the propagation of disturbances along the axon in time and space. Dynamical systems are one of the hot areas in mathematics, and their differential equations are an early example of a low-dimensional system with non-trivial behaviour. Predicting this behavior must have involved considerable numerical work in solving the four simultaneous differential equations, which they did using the algorithm described by Hartree (1), with a reference to an obscure publication. However, the authors do not discuss how the calculations were done, whether by hand or using the EDSAC computer at Cambridge, which ran its first program in 1949. A clue can be found in "An informal history of the Cambridge computer laboratory" (2), where Professor Hartree, Professor of Mathematical Physics at Cambridge, is named as an early user (1948-49). Perhaps they received advice from Hartree or even used his codes. We will never know, as Hartree died in 1958. Hartree was an interesting fellow in his own right, and built his own differential analyzer from Meccano in 1932 while Professor of Applied Mathematics at the University of Manchester (3).

1. Hartree DR (1932-3). A practical method for the numerical solution of differential equations. *Memoirs of the Manchester Literary and Philosophical Society* 77: 91-107.
2. <http://www.cl.cam.ac.uk/events/EDSAC99/history.html>.
3. http://en.wikipedia.org/wiki/Douglas_Hartree.

God and Magog: William Richard Gowers 1845-1915: the recruitment and career of 'one of the greatest clinicians and teachers of clinical medicine in the nineteenth century'

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Pioneer English neurologists, John Hughlings Jackson (1835-1911) and William Richard Gowers (1845-1915) were described by Macdonald Critchley as the “benevolent Gog and Magog of Queen Square”. Jackson (Gog), the thinker and speculator, contrasted with Gowers (Magog), the diagnostician and teacher, in both approach and temperament. Yet each profoundly admired the other. The National Hospital for the Paralysed and Epileptic, Queen Square (established in 1860), became a magnet not only for outstanding neurologists but, particularly through its association with “that Godless institution”, London University, a magnet also for Nonconformists. Both Hughlings Jackson and Gowers came from Dissenting backgrounds yet, in the age of Darwin, Jackson lost his faith, while Gowers, the son of a Hackney bootmaker, never did. Gowers, virtually orphaned at the age of 11, was recruited to medicine and mentored for his London University matriculation examinations through the intervention of successive Congregationalist ministers. Through his career he accommodated Thomas Arnold's “muscular Christianity” as he joined the burgeoning Victorian professional class, accepted a knighthood, and enthusiastically supported the expanding Empire. This paper explores these issues, drawing on archival research undertaken for the author's recent biography (with Professors Mervyn Eadie and Andrew Lees: *Sir William Richard Gowers 1845-1915: Exploring the Victorian Brain* (Oxford University Press, 2012). Sources of special interest include Gowers' shorthand diaries kept when a medical apprentice in 1862-3, drawings and unpublished lectures from the Queen Square archives, diaries he kept for his children, letters from Rudyard Kipling, and Gowers' own letters to G E 'Peking' Morrison (held in the Mitchell Library, Sydney).

The thalidomide story: History, neurology, and radiology

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Thalidomide emerged during the search for wonder drugs after 1945.

As a sedative, it caused an epidemic of sensory peripheral neuropathy of axonal degenerative type. Reduction of fascicular diameter with loss of large and proliferation of small calibre axons was found in the longest nerves.

As an anti-emetic in pregnancy it caused two epidemics of birth defects, with world-wide repercussions to be described.

The radiology of these birth defects includes congenital Charcot's joints, and the absence of bands of sensory nerve supply from the limb skeleton.

The neuropathology underlying these birth defects is reduction in fascicular diameter, loss of large and proliferation of small calibre axons.

This is consistent with sensory peripheral neuropathy occurring in the embryo, damaging neurotrophism and reducing limb growth.

Two later epidemics of sensory neuropathy confirm its sensory neurotoxicity.

Phylogenetic theories of conversion hysteria during the Great War

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Stafford Read in 1920 conceded that the phylogenetic aspect of shell shock was not considered as much as it should have been. Instinct theory, popularised by William McDougall, encouraged the military to train soldiers to unleash their pugnacity. However the troublesome instinct for soldiers was fear, and to the military it was that of their self-preservation. Suppression of fear was considered a main cause of shell shock and the works of Pavlov and Cannon were becoming accessible stimulating consideration of the psychophysiological factors involved. Bonhoeffer stated "shell shock was the triumph of instinct over ideas". For the British, W.H.R. Rivers in his early work at Maghull Hospital considered that fear could lead to a regression to the behaviours associated with self-preservation, and that this accounted for conversion symptoms (but not neurasthenia which was caused by repressive mechanisms). Psychotherapy was ineffective for this condition, hence his move to Craiglockhart Hospital and treating neurasthenic officers. Ernst Kretschmer evolved similar concepts in Germany about the 'danger-instincts', as did the French. This form of war neurosis was 'simple' though because of the primitive level of functioning treatment was difficult. Maintenance of these symptoms was reinforced by secondary factors. However once the symptoms were initiated, and of survival value, conversion symptoms proved very difficult to reverse. It is proposed that the lack of therapeutic response to the myriad of treatments provided was a factor discouraging subsequent phyletic research in conversion disorders.

To include or not to include: the formation of a neurological society in New Zealand

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The formation of the Neurological Association of New Zealand (NANZ) brought together a disparate group of clinicians and scientists interested in the nervous system. A neurosurgeon, Richard Robinson and a physiologist, Archie McIntyre, invited clinicians and scientists with an interest in neurological diseases to attend a meeting in Dunedin in 1957. The meeting was planned to coincide with a visit of Denny-Brown. Robinson proposed to use the meeting to establish a neurological society. The only dissenter was New Zealand's senior neurologist, Dr I. M. Allen, who speculated on the different effects of an inclusive neurological society that included everyone interested in neurological diseases and an exclusive society limited to those who had been fully trained in neurology or neurosurgery. Allen believed that a policy of inclusion "would lead to dilution and depreciation of the standard of training to be accepted, failure to develop the best standard of work, and the eventual provision of work at a lower instead of a higher level". An exclusive policy of admission would mean there would not be enough members to form an association. Allen did not attend, but several clinicians and neuroscientists attended the meeting. Denny-Brown delivered two lectures and nine shorter papers were presented. The scientific meeting was followed by a business meeting at which it was agreed to form a NANZ. A formal constitution was not proposed, but it was decided that membership would be open to anyone interested in joining. The NANZ has continued to meet annually. It operated without a formal constitution for the first 17 years. At times the association has had difficulty combining its role as a forum for scientific presentations with its function as a professional body for neurologists and neurosurgeons. Over time radiologists, non-clinical scientists and neurosurgeons have tended to withdraw from the association's activities.

Huntington's disease in Australia – “out of the darkness”

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Huntington's Disease (HD) is a highly complex neurogenetic condition, with a perplexing array of symptoms, which has a profound influence on the families it affects. Those carrying the mutated gene will eventually develop movement disorders, varying degrees of cognitive impairment and sometimes psychiatric conditions. Death usually occurs 15-20 years after the initial diagnosis. As an autosomal dominant condition, each child of an affected parent has a 50/50 chance of inheriting the mutated gene.

Most of the features of HD were outlined 150 years ago in the US. A young family physician, George Huntington, was able to describe the eponymous disease due to his local knowledge – his father and grandfather, both physicians, had lived amongst and treated HD families.

Little is known about the unfolding of this challenging disease outside the US and UK. In this paper, I will discuss aspects of the history of HD in Australia, including the earliest cases of the disease and HD in indigenous communities. Two Tasmanian doctors were responsible for the first Australian medical paper on HD in 1902 and the first extensive survey in 1949 - bearing a striking similarity to the US where local knowledge allowed early identification of the disease.

The secrecy and stigma surrounding HD for much of the twentieth century has been long-acknowledged but little-explored. Geneticist Peter Harper in the UK and historian Alice Wexler in the US have drawn attention to the role of the eugenics movement in fostering this stigmatization. Although not as successful in Australia, I will argue that exposure to eugenic ideas encouraged HD families to hide the presence of the disease.

Finally, the presentation will end with the 1970s. By this time things had begun to change for many HD families – patient organisations were formed to provide basic facts about the disease and to bring isolated families together.

Prefrontal lobotomy in a Sydney teaching hospital, circa 1950

Richard White^a and **Martin McGee-Collett**^b

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b Royal Prince Alfred Hospital.

In many minds there is a persisting misconception that psychosurgery was an unorthodox treatment linked with disgraced physicians such as Dr Walter Freeman in the USA and Dr Harry Bailey in Australia. In fact, during the 1940s and early 1950s, many leaders of the medical profession fostered psychosurgery as a legitimate medical therapy.

Prefrontal lobotomy was introduced as a treatment for major mental disorders in Portugal in 1936. Its popularity in the United States and in Europe was greatest at the end of the 1940s and the commencement of the 1950s, but then dramatically waned, partially superseded by the introduction of the first modern antipsychotic and antidepressant drugs.

In the USA and in the UK, tens of thousands of psychosurgical procedures were performed before 1950, but contemporaneous clinical reports tended to be scanty. Whereas psychosurgery was certainly performed in Australia during the 1950s, published Australian case reports before the 1970s are rare.

The current presentation relies particularly on a presentation to the Royal Prince Alfred Hospital Medical Officer's Association in 1951 by Dr Rex Money, the Head of Neurosurgery, in which he described a series of thirteen cases that he treated by prefrontal lobotomy. Other materials written by, and about, Dr Money, assist the construction of a snapshot of prefrontal lobotomy at the Royal Prince Alfred Hospital prior to 1951.

The presentation is not intended as advocacy for psychosurgery, but as an attempt to supplement, and contextualise, our data regarding the prefrontal lobotomy era.

Neuropathology from tropical Australia to Antarctica: John Burton Cleland

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John Cleland had a distinguished career as a bacteriologist and pathologist, investigating bubonic plague in Perth, Western Australia, early in his career and the trypanosomal disease known as *Surra* in camels at tropical Port Hedland.

As microbiologists at the New South Wales Department of Public Health, Cleland and Alfred Campbell (an acclaimed neurologist) painstakingly investigated epidemics of acute encephalomyelitis, initially dubbed 'the mysterious disease', or Australian X-disease, and later renamed Murray Valley encephalitis by Nobel Prize-winner, Macfarlane Burnet. These initial epidemics occurred in the late summer months of 1917 and 1918 in rural areas of western New South Wales and northern Victoria, far from major cities. Among 134 patients, almost half were aged less than 5 years; there were more than twice as many males as females; and mortality was 70 per cent. The illness usually lasted 4-6 days and, for survivors, convalescence was rapid. The likelihood that the epidemics were caused by a viral infection was confirmed by experimentally injecting brain and spinal cord material from patients who died into macaque monkeys, sheep and occasionally other animals, leading to transmitted disease. In later epidemics, it was shown that Murray Valley encephalitis was a mosquito-borne viral disease carried by birds from tropical Australia and infecting other birds and animals.

Much later in his life, Cleland and Ronald Southcott (1969) sought to explain the illnesses of Douglas Mawson and Xavier Mertz during their ill-fated expedition in Antarctica in 1912-1913. They suggested controversially that the various symptoms were caused by excessive intake of vitamin A from eating dog or seal liver.

Influenced by books his father had given him as a boy, Cleland became interested in botanical studies of fungi, publishing two volumes on South Australian fungi. As a naturalist, he was also fascinated by ornithology and various anthropological surveys. Cleland Wildlife Park within Cleland Conservation Park in the Adelaide Hills commemorates this extraordinarily gifted sage.

Sir Grafton Elliot Smith: a neglected polymath

Ian Steele-Russell

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Grafton Elliot Smith (1871-1937), professor of anatomy, Egyptologist and anthropologist, was born in 1871 in Grafton, New South Wales. He graduated in 1893 from the University of Sydney with an M.B., Ch.M. In 1895 he obtained his M.D. with a gold medal for his thesis on the anatomy and histology of the neuroanatomy of non-placental mammals. His discovery that the brains of monotremes and marsupials lack a corpus callosum was a major contribution to comparative anatomy.

Elliot Smith went to England in 1896 as an advanced student at St John's College, Cambridge (B.A. 1898; M.A. 1903), becoming a fellow of his College in 1899 in recognition for his neuroanatomical research.

In 1900 he was appointed first professor of anatomy in the Egyptian Government School of Medicine, Cairo. Here Elliot Smith organized a virtually new department, providing most of the anatomy teaching himself. At the same time, he developed his long lasting interests in Egyptology and anthropology. In 1909 Smith was appointed to the chair of anatomy at Manchester University, where he began a revolution in the teaching of the subject in Britain. Lectures were no longer the principal method of instruction, creating time for the introduction of radiological anatomy and the inclusion of histology. His main emphasis was on the functional implications of structure. He launched ideas that took twenty-five years to gain general currency in British medical teaching.

In wartime England, he diversified his interests and served the war effort with studies of the neurological problems of shell-shock. He was a member of the General Medical Council from 1913 to 1919. A fellow of the Royal College of Physicians from 1915, Smith was appointed to the chair of anatomy at University College, London, in 1919, where he remained loyal to the Bentham legacy.

In this presentation his legacy to three widely different disciplines (anatomy, anthropology and Egyptology) on three separate continents (Africa, Australia, and Europe) will be evaluated.

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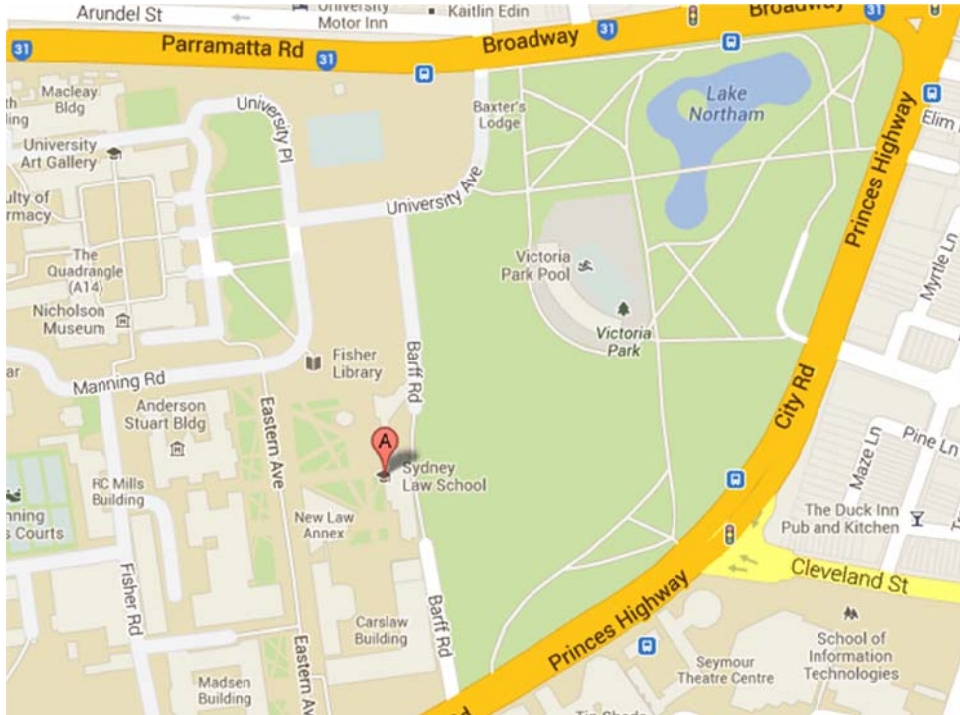
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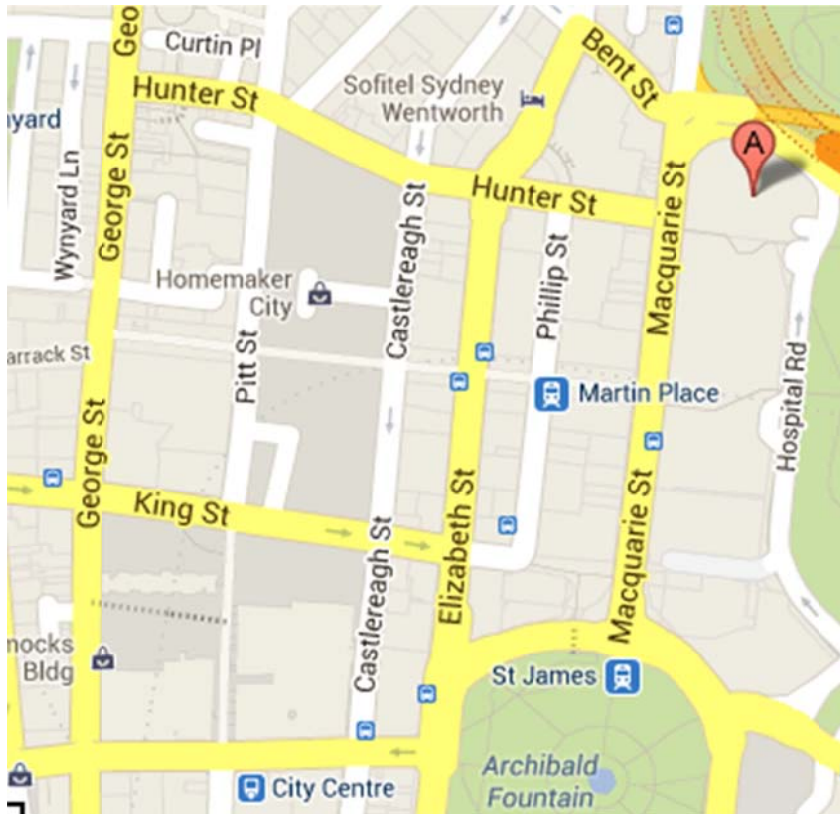
VENUES

Lecture Theatre 101, New Law School Building, University of Sydney (Tuesday, Wednesday, Friday main sessions)

Fisher Library, Level 2 – Exhibition Space (Tuesday evening reception)



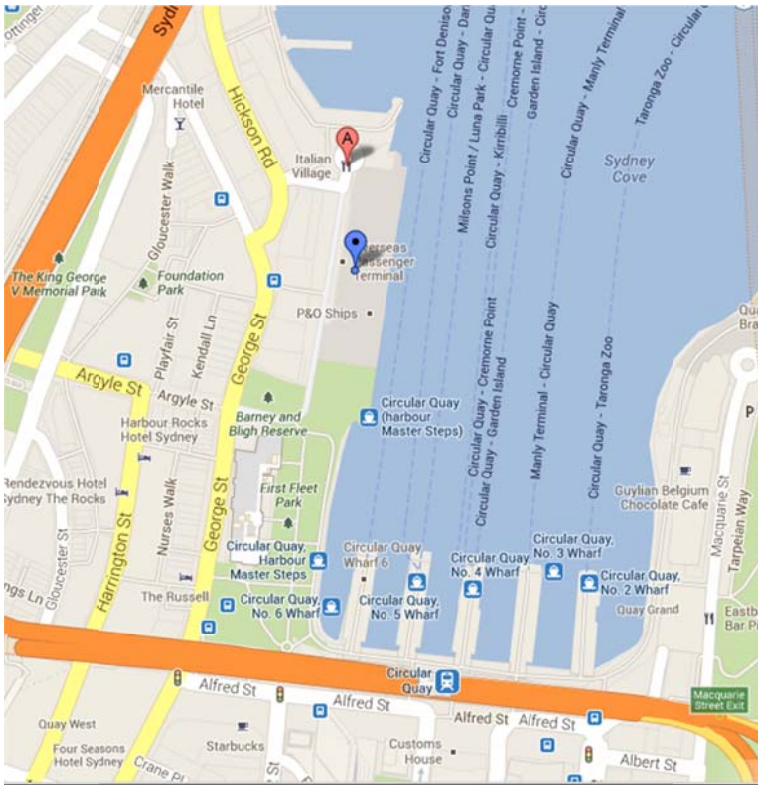
Friends Room, State Library of NSW – enter Bent Street (around corner from Macquarie Street)
(Wednesday evening event)



Old Quarantine Station, North Head (Thursday sessions)



Imperial Peking at the Rocks – 15 Circular Quay W, The Rocks NSW 2000 (Friday evening conference dinner)



Coffy Lecture Theatre, Level 1, Centre Block, Sydney Hospital, Macquarie Street (Saturday sessions)

