The opinions expressed in the newsletter by authors and interviewees do not necessarily reflect the opinion of the Editor or the UCL Institute of Neurology and National Hospital for Neurology and Neurosurgery.

All photographs have been kindly provided by Medical Illustration, Queen Square Library and the Editors private collection.
CONTENTS

Editorial 02

News 03
News from Queen Square (January—May 2015)

Alumni Interview 10
Professor Takamichi Hattori discusses his time at Queen Square and career

Queen Square Library and Archives 13
News and events from the Queen Square Library

Contemporary India: A Challenge to the Idea of India? 15
Dr Selai reports on a recent event hosted High Commission of India

Neurology 2015 14
Report on the recent Neurology 2015 conference

The Impact of Epilepsy on Arranged Marriages in India 9
Dr Selai reports on a recent trip to India

Photos from our Archives 17
A look at a group photograph from the Queen Square Archives
In this edition of the newsletter we are fortunate to have two reports, written by the Head of the Education Unit Dr Caroline Selai, within the topic of the UCL Grand Challenge of Intercultural Interaction. Our Alumni interview is with Professor Takamichi Hattori, one of our Japanese Alumni, who shares his memories of Queen Square and his career with us. We have a piece on the Neurology 2015 conference held in March this year, and a request to help put names to faces for some group photographs from the early 1970’s. We also have an update from the Queen Square Library including details on the renaming of the Archives “pod” as the Louise Shepherd Room. Louise, who passed away in 2013, was a great supporter of the Alumnus Association. She was Librarian at Queen Square from 1993 until her early retirement in 2012. Her sense of humour and determination was a constant source of inspiration and she is still missed by all those who knew her.

I am also pleased to announce that, 7 Queen Square, where the Education Unit and student cluster room is based, will be refurbished and expanded, with work due to commence in August 2015. We intend to rename the student social learning space as the Pat Harris room, in recognition of all the work Pat did to support students and visitors at Queen Square from the 1960’s until late 1980’s. For those of you who remember when Pat worked at Queen Square, we would really love to hear from you with any recollections you may have.

Our membership continues to grow and I would, as always, appreciate your assistance in spreading the word and informing alumni not currently on my mailing list to get in touch. I would also be very happy to publicise events and news from you, and also enjoy hearing recollections from your time here at Queen Square.

I would like to thank the following people, without whom this edition of the newsletter would not have been possible; Professors Andrew Lees and Simon Shorvon for their continued support and enthusiasm; Professor Takamichi Hattori for his interview; Miss Jean Reynolds and Ms Sarah Lawson for their attention to detail in proof reading this edition; Ms Sarah Lawson and Mr George Kaim for Queen Square Library and Archive material, and the Queen Square Library for photographs used in this edition.

I hope you enjoy reading the ninth edition of the Queen Square Alumnus Association newsletter.

David Blandred
**NEWS FROM QUEEN SQUARE**

**FEBRUARY 2015**

**UCL awarded £10m to develop new dementia treatments**

Alzheimer’s Research UK announced in February a £30m Drug Discovery Alliance, launching three flagship Drug Discovery Institutes at UCL, the University of Cambridge and the University of Oxford. The Drug Discovery Institutes will see 90 new research scientists employed in state-of-the-art facilities to fast-track the development of new treatments for Alzheimer’s disease and other dementias.

Each Institute will be led by a Chief Scientific Officer working in tandem with some of the UK’s leading academic researchers based at each of the three universities and Alzheimer’s Research UK’s own in-house research leaders. New ideas and breakthroughs from academic research teams in each university, and beyond, will be driven straight into the hands of dedicated biology and chemistry teams in each Institute, expert in designing and developing potential new medicines. The UCL Drug Discovery Institute, embedded within UCL’s multi-Faculty campus in the heart of London, will unite world-class dementia researchers with drug discovery experts. The University has strong clinical partnerships with the NHS and state-of-the-art infrastructure to support this pioneering initiative.

“Although our understanding of dementia has increased considerably in the past decade, this has yet to yield commensurate benefits for patients. By bridging the vital gap between basic research and treatments for patients, the Drug Discovery Institutes should help us to change this. Harnessing the considerable expertise in dementia and drug discovery across UCL and throughout the partnership, we hope to identify promising drugs that could make a real difference to patients’ lives.”

Professor Giampietro Schiavo, UCL Institute of Neurology, is the co-lead academic scientist at UCL Drug Discovery Institute

**MARCH 2015**

**Professor Mary Reilly is elected to be the first female President of the Association of British Neurologists in 83 years**

Professor Mary Reilly has been elected ABN President from 2017-2019 and is President elect from 2015-2017.

Professor Reilly has been a consultant neurologist at Queen Square since 1998 and was promoted to Professor of Clinical Neurology at UCL in 2010. She is head of the Division of Clinical Neurology and Co-Director of the MRC Centre for Neuromuscular Diseases in the Department of Molecular Neurosciences at UCL Institute of Neurology. She is internationally recognised for her expertise in research and clinical practice related to peripheral nerve diseases.

The Association of British Neurologists (ABN) was established 83 years ago in 1932; Professor Reilly originally came to Queen Square to undertake research with Professor Anita Harding in 1991.
Structure of gene messenger molecules reveals key role in diseases

Messenger RNAs (mRNA) are linear molecules that contain instructions for producing the proteins that keep living cells functioning. A new study by UCL researchers has shown how the three-dimensional structures of mRNAs determine their stability and efficiency inside cells. This new knowledge could help to explain how seemingly minor mutations that alter mRNA structure might cause things to go wrong in neurodegenerative diseases like Alzheimer’s.

mRNAs carry genetic information from DNA to be translated into proteins. They are generated as long chains of molecules, but they fold up into complex structures by making connections between different sections of the chain. Despite the importance of these structures to how mRNAs function, very little was known about them until now.

The study published in Nature reports a new technique allowing scientists to identify connections that hook sections of an mRNA together.

Further investigation showed that these connections affect how mRNAs interact with other molecules inside cells, and so influence how much protein they eventually produce.

A particularly important connection is found in an mRNA that codes for a protein called X-box binding protein 1.

Genetic mutations in mRNAs can lead to faulty connections and cause the wrong amounts of protein to be produced. This suggests that a wide range of human diseases could be caused by such mutations.

Imaging shows early brain changes in FTD patients

Research using brain imaging has found changes in the brain can be identified in people with frontotemporal dementia (FTD) 5-10 years before symptoms appear.

In the study, published in The Lancet Neurology, researchers from UCL Institute of Neurology analysed data from 220 participants to identify whether neuroimaging and cognitive changes before symptom onset could be shown in FTD, as reflected in findings from studies of other genetic dementias. The study was led by Dr Jon Rohrer and Professor Martin Rossor, as part of a consortium of research centres across Europe and Canada with expertise in familial FTD called the Genetic Frontotemporal dementia Initiative (GENFI).

FTD is a highly inheritable disease and the second most common form of young-onset dementia after Alzheimer’s disease. The disease is caused when nerve cells in the frontal and/or temporal lobes of the brain are affected by abnormal changes and eventually die. In about a third of patients the disease is caused by genetic mutations usually in one of three genes: GRN, MAPT or C9orf72.

In the study participants were made up of 118 mutation carriers and 102 non-carriers (those at risk of carrying a mutation because a first-degree relative was a known symptomatic carrier). The researchers calculated time to expected onset as the difference between age at assessment and mean age at onset within the family. Participants underwent a range of tests including standardised clinical assessments, MRI scans and neuropsychological battery. The researchers used linear mixed-effects models to examine whether the association of neuropsychology and imaging measures with time to expected onset of symptoms differed between mutation carriers and non-carriers.

The team found that examining changes in brain imaging could help define biomarkers that can track disease progression which is important for future therapeutic trials. Looking at the individual genetic sub-groups the researchers found different parts of the brain become affected first in each of the groups.

There are now promising avenues for treatment of FTD however it is still not known when drugs should be started or how response to treatment should be measured. It is anticipated that eventually these markers will be utilised in future clinical trials of drugs in genetic FTD.
New test measures deadly protein in Huntington’s disease patients’ spinal fluid

A new test has been able to measure for the first time the build-up of a harmful mutant protein in the nervous system of patients during the progression of Huntington’s disease (HD). Published in the Journal of Clinical Investigation, the team behind the findings, including researchers from the UCL Institute of Neurology, hope that the new assay will enable the testing of drugs that aim to lower the production of the pathogenic mutant huntingtin protein that causes the disease, and could be useful in predicting or monitoring the progression of HD.

HD is caused by a single gene mutation that results in the production of mutant huntingtin protein. The mutated gene was identified in 1993 but until now it has not been possible to quantify the mutant protein in the nervous system of living HD patients.

The international team of scientists from University College London, IRBM Promidis, University of British Columbia, University of Iowa and CHDI Foundation developed a new ultra-sensitive test using the Singulex SMC Technology Erenna Immunoassay system that is able to detect mutant huntingtin in the cerebrospinal fluid (CSF) of HD patients, including some who carry the HD mutation but have not yet developed symptoms.

The test, called a ‘single molecule counting assay’, combines fluorescent antibodies with a laser detection chamber to count individual molecules of mutant huntingtin with a very low detection threshold. The research team’s findings were validated in CSF samples from two different groups of volunteers in London and Vancouver.

CSF is used in the diagnosis of other neurodegenerative diseases like Alzheimer’s and Parkinson’s, but until now the protein that causes HD had never been detected in CSF. As well as detecting the protein for the first time, the researchers found that the level of mutant huntingtin was higher in volunteers with more advanced disease. What’s more, the concentration of mutant huntingtin predicted the severity of movement and cognitive problems in patients.

2015 will see the start of the first human clinical trial of a gene silencing or huntingtin-lowering drug, which specifically aims to reduce production of mutant huntingtin in the brains of HD patients. Being able to detect and measure the amount of mutant huntingtin present in the nervous system will be a valuable way of seeing whether the gene-silencing drug is hitting its target and has the intended effect, lowering the amount of disease-causing mHTT protein. Meanwhile, this new technique will be an invaluable tool to help researchers study the effects of this devastating disease in the living nervous system.

Behaviour changes common in early stage familial Alzheimer’s

Behavioural changes such as irritability, sleep changes and depression are common characteristics in the early stages of familial Alzheimer’s disease, according to research published in Brain. Researchers, including Professor Martin Rossor, UCL Institute of Neurology, sought to characterise early behavioural features in carriers of autosomal dominant Alzheimer’s disease mutations.

In the study 155 people with or at-risk from autosomal dominant Alzheimer’s disease (97 asymptomatic, 25 mildly symptomatic and 33 overtly affected carriers) were evaluated with formal questionnaires: the neuropsychiatric inventory-questionnaire, the 15-item geriatric depression scale and the clinical dementia rating scale. The participants were compared to 106 non-carriers. Depression, apathy, disinhibition, irritability, sleep changes and agitation were more common and the degree of self-rated depression more severe in mildly symptomatic mutation carriers compared to non-carriers.

Scientists have already determined patients who carry the gene mutation will develop familial Alzheimer’s disease. As a result they are able to obtain a detailed understanding of the natural history of the disease using molecular markers in cerebrospinal fluid, imaging and cognitive changes. Previous smaller studies of patients with familial Alzheimer’s disease and population studies has suggested psychiatric symptoms, particularly depression, are found with early disease and may precede the development of cognitive impairment.

The study emerged from the Dominantly Inherited Alzheimer’s disease (DIAN) network, an international collaboration of leading scientists set up to characterise early clinical and biomarker changes occurring in patients with Autosomal Dominant Alzheimer’s disease.
Deep brain stimulation for Tourette syndrome

In the largest trial ever performed, and recruiting the most severely affected patients with Tourette syndrome, researchers led by Dr Tom Foltynie, UCL Institute of Neurology, have shown that deep brain stimulation of an area of the brain called the globus pallidus (GPI) can lead to significant reduction in tic severity when stimulation is switched ON compared with when switched OFF.

In the study, published in The Lancet Neurology, when researchers compared patients’ tic severity before surgery to severity at their latest follow up, the mean improvement was 40%, accompanied by similar improvements in quality of life.

Most people affected by Tourette syndrome develop minor involuntary movements or noises (known as motor or phonic tics) during childhood. These tend to disappear as they reach adulthood. In a minority of individuals however they persist into adulthood and can be severe. In the most extremely affected people, tics can occur relentlessly preventing normal conversation, walking, or day to day function. Some patients have developed spinal injuries because of the violent nature of their tics. Drug treatments for Tourette syndrome are only partially helpful, and frequently have sedating side effects. New treatment options are therefore necessary.

The researchers are now working hard to use the findings of the trial to try and persuade NHS England to commission a small deep brain stimulation service for similarly affected patients at the National Hospital for Neurology and Neurosurgery (NHNN).

Professor Ray Dolan elected as a Member of the European Academy of Sciences and Arts

The European Academy of Sciences and Arts was founded in 1990 and focuses on interdisciplinary discussion across specialist areas, ideologies and scientific cultures as well as promoting transnational dialogue and visionary developments of new scientific knowledge and academic thinking. It now brings together over 1700 scientists and researchers, philosophers and artists from Europe, Asia and the USA, including 32 Nobel Prize winners.

Teaching Awards 2015

Clinical Teaching Awards

For the fourth year voting was held for the Djamshidian-Chinthapalli award for outstanding contribution to teaching (for bedside teaching for MSc/Diploma students) and the winner of the award was Dr Duncan Austin with an honorable mention for Dr Niccolo Mencacci, Dr Ross Nortley, Dr Alex Horga, Dr Mark Nowell, Dr Catherine Slattery and Dr Niamh Cawley.

The winner for Clinical Tutor of the year went to Dr James Gratwicke (eligibility was based on clinical sessions, Master Classes, and Case Presentation sessions), with an honorable mention to Dr Kenji Yamamoto, Dr Wallace Brownlee, Dr Fion Bremner and Prof Niall Quinn.

Clinical Neuroscience Dr Tony Pullen Lecturer of the Year Award

Dr Katerina Fotopoulou has been awarded the “Clinical Neuroscience Dr Tony Pullen Lecturer of the Year Award” for 2014-15. Students cited Dr Fotopoulou for a very interactive and enjoyable lecture.

Second place goes to Dr Ed Wild, with third place being shared between Professor Nick Fox and Professor John Rothwell.

Honourable mentions to Prof Adolfo Bronstein, Professor Nick Wood, Dr Mark Edwards, Dr Helene Plun-Favreau, Prof Jack Price, Dr Fion Bremner, Prof James Fawcett, Prof Joanna Zakrzewska, Dr Joern Diedrichsen, Dr Jonathan Rohrer, Prof Mary Reilly and Prof Matthew Walker who all polled votes.
NEWS FROM QUEEN SQUARE

MAY 2015

Professor John Hardy elected member of EMBO

Professor John Hardy, UCL Institute of Neurology, has been elected to EMBO membership, alongside other outstanding researchers in the life sciences.

The latest scientists to join EMBO come from 19 different countries and include 18 female scientists recognized for their contributions to life science research. The EMBO Membership currently comprises more than 1700 life scientists. EMBO Members serve on selection committees for EMBO programmes, mentor young scientists, and provide suggestions and feedback on the activities of EMBO.

New EMBO Members and Associate Members are invited to present their research at the EMBO Members' Meeting from 28-30 October 2015 in Heidelberg.

Professor Alan Thompson elected to the Fellowship of the Academy of Medical Sciences

Five researchers from across SLMS, including Professor Alan Thompson, Dean of the UCL Faculty of Brain Sciences, have been recognised for their contribution to the advancement of medical science by election to the Fellowship of the Academy of Medical Sciences. Professors Peter Brocklehurst, Frances Brodsky, Diana Kuh, Catherine Law and Alan Thompson joined the existing Fellows of the Academy to bring the total membership to 1134.

The independent Academy of Medical Sciences promotes advances in medical science and campaigns to ensure these are translated into benefits for patients. Academy Fellows are elected for excellence in medical research, for innovative application of scientific knowledge or for their conspicuous service to healthcare.

UCL Institute of Neurology researchers awarded MRC fellowships

Fellowships were recently awarded to researchers supported by the BRC, the National Institute for Health Research and the NIHR Queen Square Dementia Biomedical Research Unit.

Ten awards were granted nationally from the Medical Research Council, with four going to Dr Pietro Fratta, Dr Jonathan Rohrer, Dr Ed Wild and Dr Gavin Winston, all from the UCL Institute of Neurology.

Dr Fratta’s fellowship will focus on amyotrophic lateral sclerosis (ALS), a rapidly progressive neurological disease that attacks the nerve cells. Dr Fratta will explore RNA metabolism in ALS, testing whether RNA molecules, which allow the information contained in the DNA to be carried out in cells, are crucial for motor neurons to survive and what role this plays in motor neuron disease. In the past five years a series of genetic discoveries have made it apparent that a number of crucial genes are involved in various steps of RNA metabolism.

Dr Rohrer will use his fellowship to identify new biomarkers in frontotemporal dementia (FTD), the second most common form of young-onset dementia after Alzheimer’s disease, to look at MRI imaging, PET imaging, cerebrospinal fluid and serum markers. Dr Rohrer will measure when they first become abnormal and how they change over time to know when the best time is to give disease-modifying therapy. He and his team wish to see how early change can be seen, particularly in FTD groups who have a genetic mutation from birth but only develop symptoms in their fifties or sixties.

Dr Wild aims to develop further our understanding of the neuropathobiology of Huntington’s Disease by studying patients’ cerebrospinal fluid and how the brain responds to things that go wrong.

Dr Winston’s research focuses on pre-surgical evaluation in refractory focal epilepsy, a type of epilepsy that means medicines don’t work well, or at all, to control seizures. At the moment, a range of different techniques are used to study refractory focal epilepsy such as imaging (MRI, PET and Single-Photon Emission Computed Tomography (SPECT)), EEG, and neuropsychology. Currently, these techniques are expensive and time consuming and, by using a combination of these techniques, they do not always find where seizures are coming from.
A new genetic switch uncovered in the long genes expressed in our brain

A new mechanism for splicing-based gene regulation has been discovered in vertebrates by a team of researchers at UCL Institute of Neurology and UCL Genetics Institute, showing that sometimes cells select a piece of a gene known as an exon, but then later discard this piece in the process called ‘recursive splicing’. This process was observed in some of the longest genes that are expressed in the brain, which is of clinical significance, since these genes are often implicated in autism or other neurodevelopmental disorders.

The study published in Nature reports newly discovered recursive sites that are highly conserved in mammals. The genes containing recursive splice sites are among the longest genes in vertebrate genomes, because they contain extremely long introns. The exons of a gene are combined together during a process called splicing in order to create the messenger RNA (mRNA). These mRNA molecules contain the instructions for producing the proteins that keep our cells functioning. Moreover, in our genes, different shots can be selected and combined into different variants of the motion picture via alternative splicing.

This mechanism increases the complexity of mRNAs and proteins that are made from the limited number of ~20,000 genes present in human genome. This is particularly important for the cells in the brain, allowing them to perform complex brain functions that enable us to think and learn. Long introns contain literally hundreds of cryptic sequences that could be used to direct the splicing process, and thus the cellular machinery faces great challenges in distinguishing true exons from those that appear very similar to exons, but are not supposed to be used.

The present study used high throughput DNA sequencing to identify many previously un-observed splicing events within long introns, and then examined the mechanisms that distinguish bona fide splice sites from cryptic splice sites. The study finds that recursive sites are initially defined by an exon, which is later removed, and therefore it remains ‘invisible’.

Mutations in two novel genes cause primary dystonia

Researchers at the UCL Institute of Neurology have identified mutations in hippocalcin (HPCA) and potassium channel tetramerization domain containing 17 (KCTD17) as novel causes of primary dystonia, a disorder which afflicts over 70,000 people in the UK alone. The studies, done by Dr Gavin Charlesworth and Dr Nicolo Mencacci under the supervision of Professor Nick Wood and Professor Kailash Bhatia, are published as separate papers in the American Journal of Human Genetics.

In the first study, recessive mutations in the gene HPCA were identified as the cause of childhood onset isolated dystonia in three siblings from a consanguineous family of Middle Eastern origin. Subsequently, mutations in the same gene were also detected in a second family exhibiting young-onset dystonia, confirming HPCA as the responsible gene. In the second study, a missense mutation in KCTD17 was identified as the cause in a very large British family with dominantly inherited myoclonus-dystonia – a condition in which dystonia is present together with non-epileptic myoclonic jerks. This same mutation was then detected in a separate, unrelated family originating from Germany who exhibited the same condition.

Both studies used the next-generation exome sequencing platform at Institute of Neurology in combination with more traditional genetic linkage studies. At present, little is known about the cellular functions of HPCA and KCTD17 and their mutation leads to the development of dystonia. Nonetheless, both genes were shown to be highly expressed in the brain, and, in particular, the basal ganglia (an area intimately connected with movement disorders). In the case of both genes, experiments in cell models demonstrated significant abnormalities in intra-cellular calcium signaling, adding weight to previous data suggesting that perturbed calcium homeostasis may play a central role in the pathogenesis of this condition.

To gain further preliminary insight into the biology of KCTD17 and HPCA, weighted gene co-expression network analysis (WGCNA) was used. WGCNA uses brain regional mRNA expression data to generate modules of genes that are highly co-expressed and co-regulated and therefore likely to be functionally related. The analysis was performed based on the dataset generated by the UK Brain Expression Consortium (UKBEC) using samples from more than a hundred neuropathologically normal brains. This analysis showed that, in the basal ganglia KCTD17 and HPCA form part of the same gene network and that this network is significantly enriched for genes involved in postsynaptic regulation of dopaminergic transmission.
Professor John Duncan, Clinical Director of the National Hospital for Neurology and Neurosurgery and I were pleased to be invited, by Professor Gagandeep Singh, Professor and Head, department of Neurology, Dayanand Medical College, Ludhiana, India, to be Co-Principal Investigators (Co-PIs) on a research project entitled: ‘The Impact of Epilepsy on Arranged Marriages in India: a Descriptive Study and Formulation of Recommendations’.

Arranged marriages continue to account for an overwhelming majority of marriages in the Indian subcontinent. In India, as in many societies, marriage is one of the most important social institutions and a means to establish relations between two families. The marriage contract may be withdrawn by either party or mutually. The financial aspects include the settling of property rights.

When a potential bride or groom is being sized up, what qualities are important? Typically the height, physical appearance, occupation, qualifications and religion or caste group are all discussed as well as a wealth of other family information. But what if the bride or groom has epilepsy? Should this be declared? If so, when? At the very start of any marriage negotiations or sometime later?

It is questions such as these, posed by many young people with epilepsy who attend his clinic, that prompted Professor Gagandeep Singh to apply for funds for this research project.

Neurologists and other clinicians are of course expert at answering questions about the causes, treatment and management of epilepsy, but questions about marriage and whether (or not) to disclose a health condition such as epilepsy go beyond the purely clinical and enter the psycho-social or even the moral realm! Many clinicians may feel these questions go beyond their remit and expertise.

**The project: phase 1:** With a small grant from UCL Grand Challenge of Global Health, Professor Duncan and I attended a one-day round-table meeting in Ludhiana on 19th April 2015, convened by Professor Singh.

The meeting was attended by a large number of clinicians, social scientists, researchers, students and others. The goal was to discuss this important topic, to pool our collective experiences and to start to formulate recommendations.

**Research methods:** There are many methodological challenges in designing a research project such as this. What are the precise research questions and what is the most appropriate study design? We decided to collect both quantitative and qualitative data. At the meeting, data presented was in the form of clinical case-reports, focus groups and the preliminary results of a survey of people with epilepsy, their carers and the general population.

**Recommendations arising:** The report and recommendations are still being drafted. However, one example is the suggestion to discuss the impact of epilepsy on marriage with patients at a much earlier stage i.e. as they approach their teenage years and early adulthood. Certainly long before marriage negotiations commence and earlier than just a few weeks before the marriage ceremony!

**Next steps:** We are planning a larger study. We will give an update in a forthcoming newsletter. Watch this space!

**Do you have an experience to share with us?**

Do any of your patients ask questions about the impact of their health condition on arranged marriages? Are they people with epilepsy and/or other health conditions? Is this an issue in other parts of the world as well as in India? Please email me at c.selai@ucl.ac.uk if you have an experience to share. We look forward to hearing from you.
I worked at the Department of Neurology, Matsuda City Hospital. From April 1978 to March 2008 I held several positions at Chiba University, first as Lecturer, then Assistant Professor, Associate Professor of Neurology, before becoming Professor and Director of Neurology. Since April 2008 I have been Professor Emiritus of Chiba University and Chief Director of Dowakai Medical Corporation, Hunabashi, Chiba.

I was interested in neurology since I was a medical student, but in those days neurology was in an infantile stage in Japan and there were very few independent Neurology Departments. In the Neuropsychiatric department I learned about neurology. My instructors recommended that I study at Queen Square. Studying abroad was not easy in those days, because of mainly economical reasons. I finally realised my ambition and became a postgraduate student of Queen Square. The teaching there was excellent and far more than I expected. I enjoyed the outpatient teaching, clinical demonstrations, neuroradiology demonstrations, ward rounds, clinico-pathological conference and others. I learned a lot by the splendid array of excellent teachers during my two years. In those days there were many doctors from USA who has just finished neurology or neurosurgery residency programmes. I was impressed by the way they discussed cases, and I wanted to do a residency in Neurology in America. After passing the ECFMG examination, I applied for several training programmes there, but I was not accepted. The reason was later made clear, because I applied only for the more prestigious programmes. Dr W.G.P. Mair, consultant neuropathologist in Queen Square helped me, and I was eventually accepted by Professor Bernard Smith in Buffalo, New York who had been a good friend of Dr Mair since their student days in Aberdeen, Scotland.
2. What were your impressions of Queen Square? Can you elaborate about your experiences here?

In those days there was no CT or MRI. To find a diagnosis I was dependent on taking a meticulous history together with a full neurological examination, techniques which I learned from the excellent teachers at Queen Square. I then understood deeply what neurology was, and how I should strive to be a good clinical neurologist.

3. Who are the people you remember most from your time at Queen Square?

There were many excellent clinicians such as Dr Macdonald Critchley, Dr William Gooddy, Dr Ralph Ross-Russell, Dr Denis Williams, Dr M.J. McArdle, Dr Michael Kremer, Professor Roger Gilliatt, Professor John Marshall, Dr Reginald Kelly, Dr. Peter Gautier Smith, Dr John Morgan-Hughes, Dr. Roger Bannister, Dr Ian McDonald, Dr Roman Kocen, Dr Kevin Zilkha, Dr Joseph Blau, Dr Newson Davis and Dr Michael Harrison. Even now, I can remember well the above doctors. I liked the ward rounds and clinical demonstrations given by the senior consultants. The teaching of Professor Marshall was clear and impressive. I was also impressed by the teaching I received from Dr Macdonald Critchley, one of the most excellent speakers I ever met, especially his knowledge about the history of Queen Square and the famous doctors connected with it such as Gordon Holmes, Kinnier Willson, William Gowers and Hughlings Jackson. I was taught in neuropathology by Professor W.B. Blackwood, Dr W.G.P. Mair and Dr A.D. Dayan. Professor Blackwood even gave me a desk and chair in his department. I was taught neuroradiology from Dr James Bull, Dr George du Boulay and Dr Brian Kendall; neurophysiology by Dr A.M. Halliday and Dr William Cobb; neurosurgery by Professor V. Logue and Mr Lindsay Symon; neuro-ophthalmology by Mr Michael Sanders, and neuro-Otology by Dr M.R. Dix. I also enjoyed the teaching of clinico-anatomy as given by Dr Marion Smith and Dr Peter Nathan.

4. How did the practice of neurology in the UK compare with Japan?

I was a postgraduate student and as such had very good clinical experiences in Queen Square. Neurology in Japan was in its infancy at that time.

5. If you were starting your career now, what research area would you choose to work in?

I do not know. I think it is not difficult to find a research theme in the field of neurology so long as we have a mind that is research orientated. During my time as a professor our group published over 300 scientific papers in English including 36 doctoral dissertations.

6. What advice would you give to a young researcher?

Three steps: (not only for a researcher but also for a clinician)

One: Find a good teacher.

Two: Have a good human relationship with the teacher.

Three: Work hard and obtain a good evaluation from the teacher.
7. Can you tell me something of the links and collaborations you forged with researchers at Queen Square whilst at Chiba University?

Since I had become a Professor and Director of Neurology in Chiba University, two of my staff members studied at Queen Square. Dr Rjuji Sakakibara within the neuro-urology department with Professor Clare Fowler and Dr Masato Asahina in the autonomic department with Professor Christopher Mathias. They had very good collaborations and published many scientific papers. When I was a president of the Japan Neurogenic Bladder Society and Japan Autonomic Society I invited both professors to visit Japan on different occasions. Professor Fowler invited me to her country house when I visited England, and I and my wife had a very good time with her and her husband. Dr Satoshi Kuwabara (my successor and associate editor of JNNP) had a good relationship with Professor Hugh Bostock and published many scientific papers.

8. What do you think is the most important piece of work you accomplished in your career?

I was interested in neurogenic bladder, and had published many papers especially since Dr Rjuji Sakakibara joined us. Why I was interested in neurogenic bladder is probably related to the teaching I received from Dr Peter Nathan when I was at Queen Square. Professor Fowler told me that Dr Nathan was the first Uro-Neurologist in the world. (I remember that Dr Nathan was kind to Japanese people which was rather unusual at that time, not many years after the end of World War II.)

9. What does Queen Square mean to you?

Everything, as I am a neurologist. Now I administer a three hundred bed hospital and four out-patient clinics together with two nursing homes. I have never forgotten the neurological mind-set which I obtained during my stay at Queen Square.

Finally, I would like to express my sincere gratitude to the many excellent teachers I met at Queen Square and to the good secretary Miss Pat Harris.
Queen Square Archives exhibition: Queen Square: a journey through its buildings & institutions

This exhibition, which will run in Queen Square Library until August 2015, features photographs, objects, plans and maps from Queen Square Archives, reflecting the Square’s history from the early 18th century to the present day. A compilation of selected images is available online. Please see the exhibition handout for further information

http://www.queensquare.org.uk/archives/visiting/exhibitions

A Full online gallery of works from our recent Letter in Mind art exhibition is still available. All proceeds go directly to the National Brain Appeal. The National Brain Appeal will be re-running this event in the Autumn.


You can visit to see our displays in person: Monday - Friday 9am-7pm (last entry 6pm). Please see our opening hours page for details of our opening over the coming weeks.

http://www.ucl.ac.uk/ion/library/lib-info

Queen Square Library evening reception: Monday 22nd June, 4-6pm

We are holding an early evening reception in Queen Square Library marking the renaming of the Archives “pod” as the Louise Shepherd Room and the fifth anniversary of the re-opening of Queen Square Library.

Highlight of the evening will be the unveiling of a new commemorative plaque on the Archives “pod” in Louise’s memory.

We would be delighted if Queen Square Alumni were able to join us to celebrate. Light refreshments will be provided.

Please RSVP to: sarah.lawson@ucl.ac.uk
NEUROLOGY 2015:
Leading edge Neurology for the practising clinician

The 2nd course, which we hope will become an annual highlight of the British Neurology calendar, was held in late March 2015. The course is aimed at consultants and senior trainees in neurology and other neuroscience specialties, and seeks to provide a comprehensive update on the practical hospital management of neurological diseases. The emphasis of this year’s course was on modern techniques and therapies in a clinical setting, and the clinical practice of neurology. Lectures were given on 6 plenary topics: therapy in acute neurology, neuromuscular diseases, headache and Parkinson’s disease, difficult therapy areas, neuropsychiatry and dementia, and stroke. There was also a video session on eye movement disorders by Dr Gordon Plant, a CPC session led by Dr Michael Lunn, a ‘Town Hall’ session, and a Nobel lecture; by Professor Jim Rothman, winner of the 2013 Nobel Prize for Physiology or Medicine, who is a Research Professor at UCL Institute of Neurology, Queen Square.

There were just under 400 participants, which was double the number who attended the course in 2014. Each participant received a course booklet produced by the Education Unit (http://www.ucl.ac.uk/education/neurology2015booklet.pdf) which included speaker biographies and abstracts, along with a scientific paper relevant to their talk. The book was also interspersed with photographs from the Queen Square Archives. Mr Daniel Cotfas was the chief editor for the booklet and he uncovered some wonderful photographs from the Queen Square Library, one of which I have included on the contents page.

The feedback received was generally very positive, and we will try to make sure that Neurology 2016 (to be held on the 31st March – 1st April 2016) is as good as, if not better than Neurology 2015. The consensus view seemed to be that the CPC session led by Dr Michael Lunn was the stand-out lecture, with Dr Rees excelling as the discussant (most importantly reaching the correct diagnosis).

It was really nice to see so many Alumni at this conference and to be able to speak with most of you. We plan to run a special Alumni event either just before or just after Neurology 2016, and we welcome your thoughts on how best to arrange this.
UCL, in partnership with the High Commission of India, London, hosted an event on “Contemporary India – A Challenge to the Idea of India?”, on the 22nd of May at India House. The event was co-hosted by Dr Virander Paul, Deputy High Commissioner of India, and chaired by Dr Caroline Selai, Senior Lecturer, UCL Institute of Neurology. The panellists were Dr S.Y. Quraishi, former Chief Election Commissioner of India, Dr Mukulika Banerjee, Associate Professor, Dept of Anthropology, LSE and Inaugural Director of LSE’s South Asia Centre, Mr Richard Heald, CEO of the UK India Business Council, and Dr Sunil Khilnani, Avantha Professor and Director of King’s India Institute.

Dr Virander Paul, DHCI, introduced the event, and talked about the cultural, linguistic and religious diversity and complexity of India as well as its demographic might. He also spoke about how this diversity sometimes makes India difficult to understand, and that events such as these aim to promote a richer understanding of the sub-continent.

Dr Caroline Selai, Senior Lecturer in Clinical Neuroscience, UCL Institute of Neurology and Co-director of the UCL Cultural Consultation Service (CCS), spoke about the motivation for this event in light of questions of national identity and citizenship surfacing in many different countries across the world. In particular, India holds special importance for UCL, given the increasing numbers of staff and students of Indian origin at UCL as well as increasing research collaborations with Indian institutions. She also spoke about the work of the UCL Cultural Consultation Service, which she co-directs. Many of the presenting issues concern cultural identity, and the cultural construction of self.

The first speaker, Dr S.Y. Quraishi, former Chief Election Commissioner of India and currently FICCI Fellow at King’s College, is working on democracy and electoral challenges in India. He spoke about the general elections in 2014 in India, which were the biggest elections in world history with 834 million voters. He spoke about the challenges of delivering a good election in India particularly in light of the geographical, linguistic, climatic, and regional diversity of the country. However, he also referred to how a good election by itself does not imply a good democracy, and spoke about the many limitations in India’s democratic polity. While it might be relatively easy to introduce democracy, especially in a newly independent nation, it is far more difficult to sustain, which India has managed to do where several other nations have failed.

This was followed by Dr Mukulika Banerjee, Inaugural Director of LSE’s South Asia Centre and the author of Why India Votes? Her work brings an anthropologist’s perspective to what motivates Indians to vote in such large numbers. She shared the results of her research which showed that people from lower socio-economic strata are more likely to vote than rich or educated voters, and that lack of education does not stand in the way of political participation. Aside from obvious reasons such as voting for patronage or for a particular party or candidate, she found additionally that a strong motivating factor for people to vote was that everyone is treated equally at the polling booth, and that the ink mark actually created a sense of peer pressure since it acted as a visible evidence of having voted. During the 2014 elections which had a record 66.38% turnout, there were additional factors like the successful branding and marketing of the winning party as well as widespread advertising by local organisations to encourage people to vote.
Mr Richard Heald, UKIBC spoke about the work of their organisation to promote trade between UK and India, and his remarks focused on the recently launched Make in India programme of the NDA government. The program was formally launched in September 2014, in line with Prime Minister Narendra Modi’s agenda to eliminate poverty through creating a domestic manufacturing base and encouraging foreign businesses to invest and create jobs in India. However, even though there have been pledges of inward foreign investment, they haven’t translated yet because of issues such as red tape and retrospective taxation that create an uncertain business environment. One of the major achievements has been ushering in co-operative and competitive federalism through the abolition of the Planning Commission and the recommendations of the Fourteenth Finance Commission which mean that state governments can increasingly compete to attract investments. However, the government has been unable to pass legislation on the proposed Goods and Services Tax, or the contentious Land Acquisition Act. Finally, the UK government itself needs to engage more substantively with the Indian government in order for bilateral trade to improve.

The fourth panellist, Dr Sunil Khilnani, King’s College, reiterated the importance of India for the world and also spoke about its complexity and how it provides a laboratory for every interesting problem in the world today. He spoke about how the nation defined itself soon after Independence in a way that was distinct from other neighbouring countries: not on linguistic grounds, not on religious grounds, through a federal structure, and not on the basis of a military authority. This identity gave it a unique place in the international polity. However, the ‘idea of India’ faces several challenges in today’s context: the fact that it is a young nation also means that it lacks historical memory, the success of its democracy itself has brought new and varied claims, economic growth has been uneven with some regions doing better than others and the resulting inequality acts as a challenge to the union, and finally, the neighbourhood – India is the only open democratic society in the region.

This was followed by a lively question and answer session, with questions varying from the demographic dividend, to questions about higher education and innovation, as well as state capacity to carry out policy reform.

A vote of thanks was given by Dr Amit Batla, Clinical Teaching Fellow, Institute of Neurology. The evening concluded with a reception.
PHOTOS FROM OUR ARCHIVES

Do you recognise any of the people in these photos? Please help to complete the Queen Square Archives.

Queen Square Archives has an ever growing collection of group photographs which have been digitised. We have already managed to identify many of the people in several photos and are appealing to Queen Square Alumni members for help to identify the remaining people in these photographs. If you can recognise anybody, please contact Sarah Lawson with details (forename, initials, surname, and country of origin – if possible) specifying the photo title and the relevant letter on the photo. If you think that we have misidentified someone or have any queries please also contact:

Sarah Lawson sarah.lawson@ucl.ac.uk - Librarian, Queen Square Library, Archive & Museum

Post Graduate Group
Maida Vale Hospital May 1972
PHOTOS FROM OUR ARCHIVES

Gilliatt Group June 1972

ION Med Illustration
Neg No P385 0001 (June 1972)