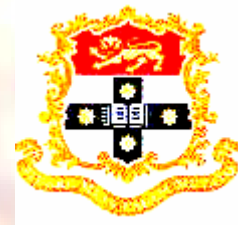
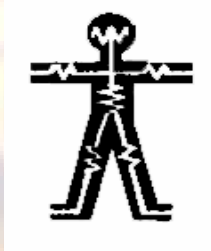


# THE NERVE RESEARCH FOUNDATION

The University of Sydney



## INSTITUTE OF CLINICAL NEUROSCIENCES

Royal Prince Alfred Hospital



## ANNUAL REPORT

2001

# ANNUAL REPORT 2001



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## **PRESIDENT & DIRECTORS REPORT**

The past year has been another very successful year for the Nerve Research Foundation, University of Sydney. Over 30 distinct research projects were undertaken. Special mention should be made of:

1. Studies by Professor Prineas in Multiple Sclerosis, which have shown that antibody and complement play an important role in disease production. This should open up further therapeutic options.
2. Professor Nicholson identified the gene for the commonest form of hereditary sensory neuropathy. This world first discovery should also lead to possible new therapies.
3. Professor Armati and Dr De Kroon showed for the first time that live human brain cells can make the lipoprotein Apo E that is associated as a risk factor for late onset Alzheimer's disease.
4. Professor Pollard's group identified the target of autoimmune neuropathy as a protein within the myelin sheath of nerves, which acts as the main adhesion molecule binding the layers together. This work was reported in the leading clinical neurology journal and attracted editorial discussion.

We are particularly grateful to those individuals who have continued their highly valued and generous donations to the work of the Foundation. Without their support the work could not continue. We also would like to acknowledge those pharmaceutical companies, Schering, Schering Plough, Boehringer-Ingelheim and Aventis who have supported young research fellows and our annual fundraising dinner.

Fundraising has continued to be a difficult task in these uncertain times. However, our many faithful supporters have yet again spent time, energy and money for the Foundation. Diane Watson orchestrated yet another excellent Annual Fundraiser with a cocktail party at the W Hotel Woolloomooloo. This was very successful and began our new initiative to purchase and equip a building, which will become the Institute of Neuroscience and Mental Health in which the brightest young research workers will be brought together with clinicians to tackle together these diseases of the brain and mind.

This is our most recent opportunity to expand the role of the Foundation to encompass over 70 neuroscience research laboratories in the University of Sydney and the Institute of Clinical Neuroscience at Royal Prince Alfred Hospital. On the agenda for this Annual General Meeting is a proposal to change the name of the foundation to the Brain and Nerve Foundation. The Brain and Nerve Foundation would thus become the fundraising arm and the public face of Sydney University Neuroscience, which is the greatest concentration of Neuroscience in the nation.

Professor J D Pollard & A/Prof P Armati  
Co Directors

Mr D Jacobs  
President

## **CHAIRMANS REPORT - THE INSTITUTE OF CLINICAL NEUROSCIENCE - RPAH**

In the Year 2001 many successes were achieved in a difficult environment. This report details the outstanding work achieved and describes some of the clinical services provided. The major areas of research have included multiple sclerosis, disorders of balance and hearing, peripheral neuropathy, alcohol and the brain, epilepsy, motor neuron disease, cognitive neurology, disorders of CSF circulation and the application of PET to neurological disease. The standing of RPA neurological research was recognised at the World Congress of Neurology in London where four of our staff were invited lecturers, more than any other Australian hospital.

We were delighted that neurological research at RPA will continue strongly following the appointment of Dr Brian Owler as neurosurgical registrar. Dr Owler has completed his PhD studies at Cambridge and will pursue his interests in the CSF circulation and neurochemical monitoring during neurosurgical procedures particularly in regard to brain injury. Members of the neurosurgical team played a major role in the highly successful World Congress of Neurology held in Sydney in September 2001. Associate Professor Michael Besser our Neuroscience Area Coordinator was recognised for his outstanding contributions to Neurosurgery in Australia by the award of the Order of Australia (AM).

In 2001 a successful Stroke Unit began at RPA. Dr Chris Derry from the National Hospital Queen Square London was an outstanding stroke fellow and was supported by Boehringer-Ingelheim. Stroke is a major cause of morbidity and mortality in the community and it is an area in which a multidisciplinary approach is essential for the successful management of affected patients. A registrar/fellow dedicated to the important task of coordinating the many contributors to the care of these patients, is clearly essential and we shall endeavor to raise the funds to continue this essential service.

These are difficult times in medicine. Our colleagues in Neurosurgery and other surgical specialties have been required to pay unreasonable and unsustainable medical indemnity fees. Many of them are facing major decisions concerning their future employment. Despite these stresses the standard of care for neuroscience patients is outstandingly high. Neurosurgical patients are mostly admitted on the day of surgery, so that they spend less time in hospital and more beds are available for others who need them. Strategies to reduce the number of patient falls in wards following surgery has resulted in a 36% reduction. The rate of patients needing to return to the theatre after cataract surgery has been reduced from 5% to 1% by the use of new surgical techniques involving smaller incisions.

The outstanding contributions of many members of the Institute are detailed in the following pages. I would like to thank all member for their dedication and hard work and wonderful achievements and their continued support throughout the year.

Professor J D Pollard  
Chairman  
Institute of Clinical Neuroscience  
Royal Prince Alfred Hospital

# **MEMBERS OF THE NERVE RESEARCH FOUNDATION**

## **COUNCIL**

Mr DL Jacobs, President  
Mr R Low, Vice President  
Prof. J Pollard, Co-Director  
A/Prof. P Armati, Co-Director  
The Hon Justice Kim Santow, Chancellor, University of Sydney  
Ms Renata Kaldor , Deputy Chancellor, University of Sydney  
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Ms R O'Neill  
Mr J Armati, AOM  
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Mr J Baker  
Dr J Milburn  
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A/Prof. P Armati  
Professor R Ouvrier  
Professor SR Leeder  
Emeritus Professor J G McLeod, AO  
Professor J Young, AO

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Ms R O'Neill  
Mr J Armati, AOM

## **HONORARY LIFE MEMBERS**

Mr DL Jacobs  
Mr M Bannigan  
Ms R O'Neill  
Dr R Kerr  
Mr J Baker  
Mr E Barnum  
Mr R Wallace  
Mr S Carroll, AO

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## **SCHOOL OF BIOLOGICAL SCIENCES – Academic Staff**

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Dr K Williams, BSc(Hons), PhD

### **Technical Staff**

Mr A Cook

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Associate Professor G Nicholson, MB BS PhD, FRACP  
Associate Professor J Watson, DPhil *Oxon*, BSc, MB BS, FRACP  
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Dr A Mohammed, MB BS, FRACP  
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Dr M Besser, Neurosurgery  
Ms R Cullen, Nurse Unit Manager, E7 North  
Ms J Gavin, Clinical Nerve Consultant, Neurosciences  
Ms L Healy, Institute of Clinical Neurosciences, Business Manager  
Sr C Hennessy, Nurse Unit Manager, E7 Intensive Care Unit  
Dr D McDowell, Neurosurgery  
Ms Kit Eu, Allied Health  
Mr M Shepherd, Nurse Unit Manager, E7 South

### **Department of Neurology**

#### **Director Of Neurology**

Professor JD Pollard, BSc (Med) MB BS PhD, FRACP, FRCP, Bushell Professor of Neurology

#### **Neurologists**

Dr L Davies, MB BS, FRACP, Staff Neurologist  
Dr J Ell, MB BS, FRACP, Visiting Neurologist  
Clinical Associate Professor MJ Fulham, MB BS, FRACP, Staff Neurologist  
Clinical Professor GM Halmagyi, MB BS, BSc (Med), FRACP, Staff Neurologist  
Professor JD Pollard, MB BS, BSc (Med), PhD, FRACP, FRCP, Bushell Professor of Neurology,  
Academic Head  
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Dr JC Walsh, MB BS, BSc (Med), FRACP, Staff Neurologist  
Assoc/Professor JDG Watson, DPhil, MB BS, BSc, FRACP, Senior Lecturer  
Dr A Mohamed, MBBS, FRACP, Staff Neurologist

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Dr J Leicester, MB BS, FRACP

### **Advanced Trainees in Neurology**

Dr S Vucic  
Dr C Kipps

### **Clinical Neurophysiology Unit**

Dr L Davies, MB BS, FRACP, Staff Neurologist (Head)  
Dr JC Walsh, MB BS, BSc (Med), FRACP, Staff Neurologist  
Dr A Mohamed, MBBS, FRACP, Staff Neurologist  
Dr J Spies, MB BS, PhD, FRACP, Staff Neurologist  
Ms E O'Connell, BN, CNS  
Ms T Ottavio, BN, RN, Nursing Unit Manager  
Ms J Boserio, CNS  
Ms T Mills, RN  
Ms M Pereira, BN, RN  
Ms E Sheridan, BA, CNS  
Ms R Spittal, RN

### **Office and Clerical**

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Ms R Rattan  
Ms M Piper

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Clinical Associate Professor M Besser, MB BS, FRACS, FRCSC (C), FACS  
Ms Bernadette Loughlane, Nurse Coordinator  
Ms Louise Healy, Business Manager

### **Honorary Neuroscientists**

Professor M Bennett, BE, MSc, PhD, DSc, FAA  
Dr RJ Bandler, BA, PhD, DSc, FAA  
Associate Professor DF Davey, BSc, PhD  
Professor GAR Johnston, PhD, MSc, FRACI  
Associate Professor PJ Armati, MSc, PhD  
Dr T-L Chan Ling, M Optom, PhD, FAAO

### **Department of Neuropathology**

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Professor of Neuropathology  
Dr RS Pamphlett, MD ChB, BSc (Med), FRACP, MRCPPath  
Honorary Consultant, Neuropathologist  
Mr K Nicoll, MB BS, Registrar

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Mrs D Sheedy, BA, Research Assistant  
Dr G Dixon, BSc, PhD, Research Officer  
Ms M Sarris, BSc, Visiting Scholar  
Ms T Garrick, Research Assistant  
Dr Y Chu, MD, Senior Research Officer

**Senior Technical Officer**

Mr S Kum Jew

**Technical Officer**

Mr A Fortis

**Neuropsychology Unit****Director**

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Dr G Savage, PhD, Clinical Neuropsychologist

Dr N Breen, MSc, BSc, MAPS, Clinical Neuropsychologist

Ms S Coombes, MSc, BSc, Clinical Neuropsychologist

Ms S Day, BA, MPH, RN

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**Honorary Staff**

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**Neurosurgeons**

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Dr D McDowell, MB BS, FRACS, Visiting Neurosurgeon

Dr JW Brennan, BSc, MB BS, FRACS, Visiting Neurosurgeon

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Dr B Owler, MB BS

**Senior Technical Staff**

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**Advanced Trainees in Neurosurgery**

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Dr R Allen, MB BS

**Unaccredited Registrar in Neurosurgery**

Dr S Enger, MB BS

## NeuroOtolology Unit

### Clinical and Research

Dr S Aw, MB BS, PhD, Scientific Officer  
Ms A Burgess, PhD, NHMRC Research Assistant  
Ms S Burton-Bradley, RN  
Mr Paul Chen, NHMRC Research Assistant  
Dr PD Cremer, MB BS, BSc(Med), FRACP, Associate Neurologist  
A/Professor JG Colebatch, MB BS, PhD, FRACP, Associate Neurologist  
Professor IS Curthoys, BA, PhD, Consultant Psychologist  
Dr JJ Ell, MB BS, FRACP, Visiting Neurologist  
Dr D Gilchrist, PhD, NHMRC Research Officer  
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Ms I Hannigan, CNS  
Ms K de Lapp, BS, Physiotherapist  
Mr H Macdougall, BSc, Research Assistant  
Mr M O'Brien, BA, Psychologist  
Ms M Pereira, BN, RN  
Dr D V Pohl, MB BS, FRACS, Visiting Surgeon  
Mr C Tsang, BA, Audiologist  
Dr SR Watson, MB BS, PhD, FRACP, Associate Neurologist  
Mr C Whitfeld, BSc, Audiologist  
Ms R Yavor, Clinical Nurse Specialist  
Ms T Mills, RN  
Ms T Ottavio, RN, BN, Nursing Unit Manager

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Ms I Menezes  
Ms R McCabe

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Mr A Cartwright, Computer Programmer  
Ms T Le, BE, Network Engineer  
Mr L McGarvie, BE(Mech), MBiomedE, Biomedical Engineer  
Mr S Pratap, Technical Officer  
Mr J Bryant, Technical Officer

## Department of PET & Nuclear Medicine

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Dr J Roberts, Staff Specialist (part-time)  
Dr R Mansberg, Staff Specialist (part-time)  
Dr M Wilkinson, Registrar PET  
Dr I Drivas, Registrar Nuclear Medicine  
Dr A Mohamed, Fellow-in-PET

### **Scientific Staff**

Dr S Eberl, BE, MSc, MACPSEM Principal Scientific Officer  
Dr R Fawdry, PhD, Principal Scientific Officer  
Dr R Fulton, BAppSc, MACPSEM Principal Scientific Officer  
Mr D Henderson, BAppSc, GradDipSc, MRACICChem, Senior Scientific Officer  
Dr M Kassiou, BSc(Hons), PhD MRACICChem, Principal Scientific Officer  
Dr S Meikle, BAppSc, PhD, MACPSEM Principal Scientific Officer  
Ms J Towson, MSc, Principal Scientific Officer

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Mr E Francia, RN  
Ms B Perry, RN

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Ms J Brackenreg, BAppSci (MRT), Acting Chief Technologist  
Ms S Fotias-Alam, BAppSci(MRT), Senior Technologist  
Mr D Rainey, DipMRT, Senior Technologist  
Mr C Constable, Senior Technologist  
Ms A Smith, Technologist  
Ms R Smith, Technologist  
Ms S Meikle, Technologist  
Ms N Loughlan, Technologist  
Mr A Lawler, Technologist  
Mr A Waugh, Technologist

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Mrs Y Hillier, Secretary  
Ms L Balingit, Receptionist/Typist

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Dr M Jennings, MB BS, FRANZCP, FRACP(C), FFPMANZCA, Psychiatrist  
Dr A Aggarwal, MB BS, FRACP, FAFRM(RACP), FFPMANZCA, Rehabilitation Medicine  
Ms J Keller, RN  
Ms A Helou, Grad Dip Soc Com, MScMED(PM), Clinical Nurse Consultant Clinical Coordinator  
Ms J Cohen, M Psych (Clin) MAPS, Clinical Psychologist

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Dr M McGee-Collett, MB BS (Syd) FRACS, Neurological Surgeon  
Dr P Glare, FRACP, FFPMANZCA, Cancer Pain & Palliative Medicine  
Dr L Martin, BDS (Hons), Head, Department of Dentistry  
Dr C Senior, MB BS (Hons) FRACOG, Gynaecologist

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Dr M Jennings, MB BS, FRAZCP, FRCP(C), Psychiatrist

Dr RT White, MB BS, FRANZCP, MRCPsych, Psychiatrist

## Department of Radiology

### *Neuroradiology*

Dr Richard Waugh, Acting Director

Dr J Hallinan, MB BS, FRACR

Dr G Parker, MB BS, FRACR

Dr E Thompson, MB BS, FRACR

Dr J Soper, MB BS, FRACR

## Rehabilitation Medicine

Dr P Henke, MB BS, DPRM, FACRM, Head

Dr C Winer, LLB, MB BS, FACRM, MRCS, DRCOG, MLCOM, DPRM, Visiting Medical Officer

Mr M O'Brien, BA, DipRehabCouns, MaPsS, Psychologist

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### **Senior Medical Staff**

Dr M Mendelsohn, MB BS, FRACS, VMO, Clinical Head

Professor W P R Gibson, AM, MD, FRACS, FRCS, Professor of Otolaryngology, Academic Head

Dr G R Croxson, MB BS, FRACS, Senior VMO

Dr A Clifford, MB BS, FRACS, VMO

Dr D Pohl, MB BS, FRACS, Senior VMO

### **Senior Technical Staff**

Dr H Sanli, PhD, Scientific Officer, Cochlear Implant Unit

### **Vocational Registrars in Training**

Dr C Palme

Dr P Yeung

Dr G Lvoff

Dr S Kuo

Dr S Mackay

### **Visiting Fellows**

Dr N Mansell

Dr P Valentine

### **Audiologists**

Dr D Rockey

Dr C-S Tsang

Ms M Bray

Ms C Pearce

## Allied Health

Ms K Eu, BEc, BSoc Admin, Allied Health Director  
Ms B Vale, BAppSc(OT), Occupational Therapist  
Ms R Ray, BAppSc(OT), Occupational Therapist  
Ms K Williams, BAppSc(Phys), Physiotherapist  
Ms M Lam, BAppSc(Phys), Physiotherapist  
Ms J Young, BAppSc(Phys), Physiotherapist  
Ms K Garvey, BAppSc(SpPath), Speech Pathologist  
Ms R Manusu, BAppSc(SpPath), Speech Pathologist  
Ms M Doctor, BSW, MSW, Social Worker  
Ms C Robinson, BSW, Social Worker  
Ms E Frigg, BSc, MND, Dietitian

## Department of Nursing E7

Ms B Loughnane, RN, CM, NNC, BHSc(N), GDNM, MHSM, Clinical Manager, Neurosciences  
Ms V Markovska, RN, MN, Clinical Nurse Consultant, Neurosciences  
Sr C Hennessey, RN, BA, Nursing Unit Manager, Neurosciences Intensive Care Unit  
Mr M Shepherd, BHS, RN, Dip App Sciences, Grad Cert Clinical Management, Nursing Unit Manager  
Ms T Ottavio, RN, BN, Nursing Unit Manager, Neurophysiology  
Ms A Cottee, RN, Acting Nursing Unit Manager  
Mr M Laxton, RN, Nursing Unit Manager

## Molecular Medicine

### Director

Associate Professor G Nicholson, MB BS, PhD, FRACP

### Hospital Scientists

Dr M Kennerson, BSc, PhD  
Mr P Lorentzos  
Ms D Radavanovic, BSc  
Mr A Hooper, BSc  
Ms P Cordoba  
Ms M Lagleva

### Research Scientists

Ms J Dawkins, BSc  
Dr D Zhu, BSc, PhD  
Mrs S Brahmbhatt, BSc

### Genetic Counsellor

Sr M Jenkins, RN

## **HIGHER DEGREE STUDENTS**

### **DOCTOR OF PHILOSOPHY – Department of Medicine & School of Biological Sciences**

S Andrias-Kauba  
W Yan  
P Kashi  
E Mathey  
S Mandadi  
N Walters  
K Podzebenko  
P Spring  
Y Wanigasekara-Mohiti

### **MASTER OF SCIENCE – Department of Medicine**

M Barnett  
T Garrick  
D Henderson  
P Kench  
M Thurtell  
T Lin  
E Krupka

### **HONOURS STUDENTS – School of Biological Sciences**

B Roediger – 1<sup>st</sup> Class and the Dakin Prize  
A Arthur – 1<sup>st</sup> Class

### **DEGREES AWARDED**

PhD – Robert De Kroon  
PhD – Jude Taylor  
PhD – S Eberl  
PhD – R Fulton

## **AWARDS**

### **PRIZES AWARDED**

In recognition of his lifelong contribution to MS research, Professor Prineas received the 2001 National MS Society/ American Academy of Neurology John Jay Dystel Prize in Neurology.

Associate Professor Michael Besser awarded the Order of Australia (AM) for contributions to neurosurgery as clinician, teacher and administrator.

Ben Roediger was awarded the Dakin Prize for the best Honours thesis in the School of Biological Sciences.

## GUEST LECTURERS AND VISITING SCIENTISTS

Professor Alastair Compston, Professor of Neurology and Chairman of Department. Cambridge University, Guest Lecturer, 6<sup>th</sup> Annual R. O'Neill Lecture Series

Dr Patrick Emond, Laboratoire de Biophysique Médicale et Pharmaceutique, Université Tours, France

Dr Christiane Franzius, Nuclear Medicine Clinic, University of Munster, Munster, Germany

Prof Alex Gektin, Alkali Halide Crystal Department, Institute for Single Crystals, Kharkov, Ukraine

Dr Jean-Marc Grognet, Direction de la Recherche Technologique, Commissariat à l'Énergie Atomique, Fontenay-Aux-Roses, France

Dr Christian Loc'h, Service Hospitalier Frédéric Joliot, Commissariat à l'Énergie Atomique, Orsay, France

Dr Richard Loiacono, Department of Pharmacology, Monash University, Melbourne

Dr Alex Pitman, Centre for PET, Peter MacCallum Cancer Institute, Melbourne

Associate Professor Aditya Gupta, Neurosciences Centre, All India Institute of Medical Sciences, Ansari Nagar, New Delhi

Professor S.C. Bantwal, Head - Department of Neurosurgery, Fr. Mullers Medical College, Mangalore, India

Professor Mitch Berger, Chairman - Department of Neurosurgery, University of California, San Francisco, USA

Professor Peter Blumbergs, Dr. Jillian Kril, Dr. Catriona McLean, Dr. Michael Rodriguez, Professor Anthony Tannenberg - Lecturers: Australian postgraduate course in neuropathology

Professor Luca Durelli, Professor of Neurology, Dept. of Neurosciences of Torino University Medical School, Torino, Italy

## RESEARCH

### School of Biological Sciences - Neuroscience Unit

#### Alzheimer's Disease

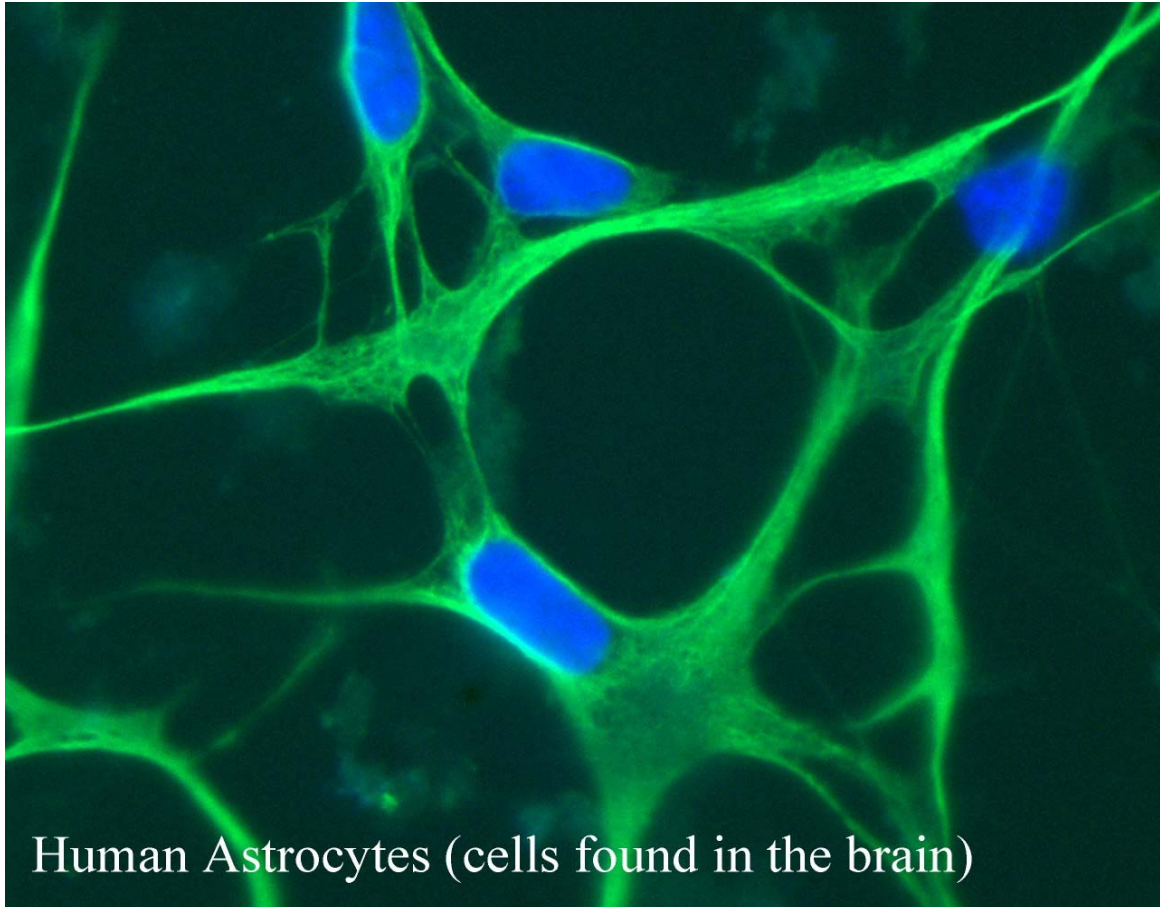
#### Work performed by B. Roediger and Associate Professor P J Armati: Examining tissue culture model of relevance to Alzheimer's disease

The linkage of the *APOE4* gene to late onset disease has led to questions concerning the role of apoE in late-onset Alzheimer's disease pathogenesis. Recently, a role for apoE receptors in the disease process has been implicated, and may be involved in nerve cell function. Although the cause of Alzheimer's disease remains unknown, there is growing evidence for a role for oxidative stress as a major underlying mechanism of the disease.

We examined the expression of two apoE receptors, human brain cells in tissue culture using hydrogen peroxide induced oxidative stress. We also studied the expression of these receptors, and the novel antioxidant, PrxV in response to this oxidative stress and during recovery.

Hydrogen peroxide induced cytoskeletal changes in the human neurons and disrupted the transport system. We showed that nerve cells can recover from the damage that oxidative stress caused to their transport system and also that PrxV in the human brain cells is upregulated after oxidative stress.

*Funding: Calcifer JESK Postgraduate Scholarship & Nerve Research Foundation*



Human Astrocytes (cells found in the brain)

### Work performed by P. Kashi and Associate Prof P J Armati

The main focus of this research was to study those parts of the brain particularly targeted in Alzheimer's disease, the hippocampus and amygdala. Petra Kashi and Associate Professor Armati continued their study into whether the nerve cells can take up apolipoprotein E (apo E) . This was of interest as one form of the molecule ,apoE4 is well defined as a risk factor for developing late onset Alzheimer's disease. We wanted to find out if the nerve cells could take up this molecule and then to define differences between its three major forms which are E2, E3 and E4.

We studied the molecules in mice which normally only have one form of the molecule - the apoE3 form. In these mice their gene for apoE was inactivated and instead, the human genes for these molecules were inserted into the mice. Such animals are called transgenic. These transgenic mice therefore have humanised forms of apoE As it is not possible to study human brain experimentally these animals provide a very important model for examining how the E4 form of the molecule might be such a risk factor in the disease process. We grew these brain regions in long term tissue cultures.

We found differences in the way each type of these transgenic nerve cells took up and internalised each of the three forms of apoE. The E4 form was taken up faster than E2 or E3 forms of apoE. The E3 and E2 genotypes had a more stable level of apo E uptake than E 4 genotype. This could have relevance to stabilising the internal ie cytoskeleton of the nerve cells and be important in nerve cells survival.

Nerve cells need apoE to transport cholesterol to their processes which change during learning so that neurons with more access to apo E and consequently to cholesterol may survive better. These findings are consistent with our previous studies in cultures of human nerve cells from unspecified regions of the brain.

*Funding: Calcifer JESK Postgraduate Scholarship & Glaxo Smith Kline*

### Work performed by R. DeKroon and Associate Prof P J Armati

Robert DeKroon and APA completed the investigations into the endocytosis of apolipoprotein E (apoE) in primary human brain cultures. We linked the gene for each of the three major forms of this molecule to a gene that produces a green fluorescence protein. The hybrid molecule (apoE-EGFP) fluoresces under ultraviolet light. Thus we can put these two genes which are linked together into cells and examine them with a microscope when we shine ultraviolet light onto the cells. This technique also means we can examine living cells in real time and video them. Some of the videos are on our website – look up Robert De Kroon to see them. The study has particular relevance to Alzheimer's disease as the apoE4 form of the apoE molecule is a genetic risk factor for late-onset Alzheimer's disease. We found bi-directional movement of apoE-EGFP in human brain nerve cells as their associated cells, the astrocytes Thus, active apoE recycling in cells suggests that apoE can act as a signaling molecule in nerve cells.

We also investigated the effects of oxidative stress which is thought to be important in the development of Alzheimer's disease. We looked at the way apoE-EGFP, ie the fluorescent hybrid molecule, is transported in a human cell lines to try to model the ageing process. When these cells were stressed the apoE vesicles and their speed of movement within the cells increased and their velocity were up-regulated. We think that this response is due to cell injury and has implications for the nerve cell death so characteristic of those areas of the human brain associated with memory and learning and which are targeted in late-onset Alzheimer's disease. These experiments also culminated in the completion of Robert PhD. He now holds a postdoctoral position with Dr Warren Strittmatter at Duke University Medical School in the USA.

*Funding: Calcifer JESK Postgraduate Scholarship & Glaxo Smith Kline*

## Multiple sclerosis

### Work performed by E. Mathey and Associate Professor P J Armati

There is increasing evidence that antibodies play a pathogenic role in demyelination in both MS and EAE. While the specificity of antibodies present in the CSF and serum of MS patients and EAE animals has been studied extensively, scant is known about the role of antibody in the initiation, exacerbation or resolution of disease. To date no patient isolate has been shown to actively cause demyelination in vivo. Without actually demonstrating a capacity for demyelination, anti-myelin antibodies isolated from patient samples remain merely circumstantial evidence of humorally mediated pathogenesis.

In this study serum samples from both relapsing remitting MS patients and normal controls were directly injected into the dorsal columns of rat spinal cords to determine whether there is any demyelinating activity. The sera from the 5 patients caused a localised, demyelinated lesion in the rat spinal cord while sera from 5 normal controls did not. IgG was then isolated from the RRMS and normal serum and again injected into the spinal cords of Lewis rats. Analysis of this experiment is currently ongoing.

*Funding: Calcifer JESK Postgraduate Scholarship & Australian Post Graduate Award*

### Work performed by A. Arthur and Associate Professor P J Armati

Multiple Sclerosis (MS) and Guillain-Barre syndrome (GBS) are inflammatory demyelinating disorders of the central (CNS) and peripheral (PNS) nervous system, respectively. Macrophages and microglial cells play a central role in the pathogenesis of MS and GBS due to their involvement in myelin destruction and phagocytosis.

These diseases are still not completely understood. For many years, patients suffering from antibody deficiencies have been treated with intravenous injections of immunoglobulin (IVIg). Human IVIg is pooled from approximately 10,000 donors and consists primarily of immunoglobulin G (IgG). IVIg has more recently been used to treat some autoimmune disorders. It has been found to successfully reduce inflammation in some of these patients.

Ariel Arthur and APA studied the effects of IVIg treatment on an animal model of inflammatory demyelinating disorders, experimental allergic neuritis (EAN) is an animal model that shares clinical, pathological and electrophysiological features with PNS autoimmune diseases such as Guillain-Barre syndrome.

We found clinical improvement and weight gain following administration of IVIg. Interestingly the electrophysiology of the treated and untreated EAN rats did not show any significant differences but improved during the recovery period. The immunohistochemical study showed clear evidence of demyelination with extensive patches of demyelinated nerve fibres.

Such clinical improvement is interesting as similar observations occur in patients again in the absence of electrophysiological improvement. This study indicates the need for further investigation into events at the cellular level such as regulation and distribution of Na<sup>+</sup> channels and the effects of early remyelination on clinical recovery.

*Funding: Calcifer JESK Postgraduate Scholarship*

## Neuropathic pain

### Work performed by S. Mandadi and Professor B. Roufogalis: Synthetic derivatives and plant extracts of capsaicin and gingerols as analgesics acting at the vanilloid receptor

By studying molecules similar to capsaicin – the ‘hot’ component of chilli peppers, and gingerol, the ‘spicy’ taste in ginger we hope to provide a novel approach to development of new therapeutic agents for the treatment of debilitating chronic pain in humans. These molecules act on the receptors present on peripheral nerve cells. If the receptor, known as VR1, can be desensitised this could provide an analgesic.

We have previously defined about 30 synthetic derivative molecules similar to those of gingerol and capsaicin as well as a plant extract. These novel and potentially important molecules show strong activation of the VR1 pain receptor with low pungency compared to that of capsaicin. These compounds activated the VR1 receptor in a subset of peripheral nerve cells. The other subsets of nerve cells do not have this receptor. The study has therefore concentrated on those nerve cells which do express VR1 and these cells have been grown in tissue culture and their responses to capsaicin and these novel analogues have been found to affect the calcium fluxes in the cells. Thus we think that gingerol and capsaicin analogues also act on this receptor in the same way as capsaicin. Because our group has found that these new molecules have low pungency they may be suitable candidates for analgesic development. Their mechanism of action in regulating pain pathways is still under investigation.

*Funding: International Postgraduate Research Scholarship & ARC-Discovery grant*

## Stroke

### Work performed by Y. Wanigasekara-Mohiti, Professor B. Roufogalis and Associate Professor P J Armati: An animal model of stroke

Glutamate is a molecule which commonly excites nerve cells in the brain. It is important in learning and memory and in the developing brain. However high levels of this neurotransmitter can kill nerve cells. Such a toxic effect has been implicated in epilepsy, Alzheimer’s disease, Multiple Sclerosis and brain trauma such as is induced by strokes. Excessive glutamate also changes the Calcium flow within these cells. This study examined the regulation of calcium fluxes within nerve cells to better understand how this occurs and how protective therapies could be developed. The study examined rat and human brain cells and showed that there are important differences in the response of rat and human nerve cells to a glutamate insult. We found that only a specific subset of nerve cells were affected. We also found for the first time that particular groups of human neurons produce molecules which could prove to be protective. Future studies will examine the mechanism of this protective effect.

## Department of Medicine – University of Sydney

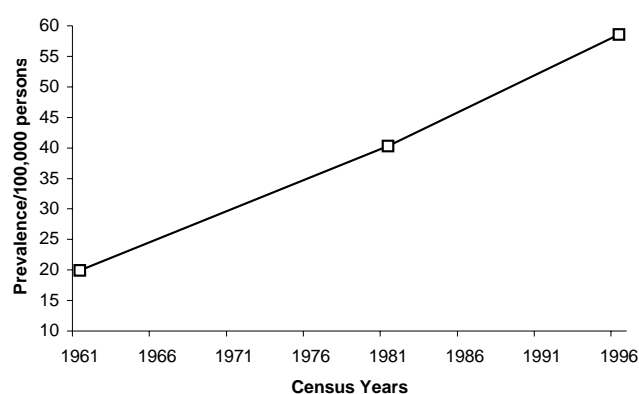
### Multiple Sclerosis

Multiple sclerosis is the most common cause of chronic disability neurologic disability afflicting young adults. The symptoms are varied, but attacks may cause double or blurred vision, imbalance, weakness, loss of sensation, or loss of control of bladder and bowel.

Patients may suffer repeated attacks from which recovery may be incomplete. Later in the disease course, some patients progressively deteriorate between acute attacks of disability.

Studies of MS in Europe and Britain have suggested that the frequency of multiple sclerosis in the Northern hemisphere is increasing. Professor J McLeod from the Department of Medicine has recently completed a comprehensive new study of the disease in Newcastle, Australia. The study, conducted in conjunction with the University of Newcastle, sought to determine whether the frequency of MS had increased since the previous epidemiological survey of the region in 1981. The current study showed a significant further increase in MS prevalence (Figure 1), and this was attributable to an increase in the number of new cases diagnosed per year, increased survival of patients, and an increase in the ratio of females to males with the condition. Ongoing work is examining the reasons for these changes in the epidemiological profile of MS.

Figure 1. Age standardised prevalence rates (all persons) Newcastle 1961-1996



In addition to epidemiological work, researchers at RPA have focused on the role of the immune system in causing the symptoms and signs of MS. For the first time, Professor John Prineas and colleagues, in collaboration with researchers in the United States, demonstrated ongoing immune-mediated destruction of myelin – the fatty sheath around nerves in the brain which bears the brunt of damage in MS - at the edges of chronic MS plaques from patients with the secondary progressive form of the disease.

Current research in Professor Prineas' laboratory focuses on the evolution of the earliest acute MS plaques, and promising early work has provided new insights into the pathogenesis of relapsing-remitting disease. In recognition of his lifelong contribution to MS research, Professor Prineas received the 2001 National MS Society/American Academy of Neurology John Jay Dystel Prize in Neurology.

Ongoing experimental work in the department is examining the role of circulating antibodies in multiple sclerosis, and the mechanisms by which these antibodies gain access to the brain during attacks of MS. RPA researchers have already found that immune cells (T-cells) may open the normally impermeable barrier between the blood and brain, allowing antibodies and other circulating proteins access to the brain.

This model is currently being used to help identify a subset of MS patients whose blood contains such antibodies. It is hoped that such an approach will lead to more specific therapy in this patient group.

The Department of Neurology has participated in many large international trials of new therapies in MS and continued with this important patient-based research in 2000-2001.

Treatment with several forms of beta-interferon, a cytokine which regulates the immune system, has been shown in previous studies at RPA to modestly reduce the frequency of attacks (by approximately 30%) in patients with the relapsing and remitting form of MS. Current clinical research focuses on the role of these medications in patients with secondary progressive MS, and follow-up of the patients is expected to continue for several years.

Ongoing collaborative work with the Department of Immunology and the Centenary Institute of Cancer Medicine and Cell Biology is aimed at developing newer therapies which stem from basic science research into MS. Members of the Department of Neurology have also forged collaborations with several overseas groups in the US and UK.

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## The Peripheral Nervous System

### Peripheral Neuropathies

Neuropathies are disorders of nervous tissue excluding the brain and spinal cord. This peripheral nervous system includes the nerves found in our limbs, which activate our muscles and provide information about our environment (sensation). It also includes nerves of the autonomic nervous system, which automatically control our blood pressure, respiratory effort and sexual function. Thus, neuropathies cause paralysis, loss of sensation and they may also impair the control of blood pressure or cause impotence in males.

Nerve fibres either motor or sensory arise from cells, which reside in or near the spinal cord. Those fibres, which pass down arms or legs to the fingers or toes, are very long and in an average man may measure well over a meter in length despite the fact that the cell from which they grow is microscopic in size. If this nerve cell were magnified to the size of a golf ball then the nerve fibre would be equivalent in size to a piece of electric cable six miles long.

Hence, nerve cells of this type have an enormous metabolic load to maintain their long extension and produce the many proteins and other chemicals they contain. It is therefore not surprising that peripheral neuropathy may result whenever nerve cells are stressed and this may occur in almost all systemic diseases such as diabetes, vitamin deficiency, cancer and many toxins. Hence, neuropathies are very common in the community.

### Inflammatory Neuropathies

These are mostly autoimmune disorders resulting from a misdirected attack by the body's immune system. An acute inflammatory neuropathy known as the Guillain Barre syndrome (GBS) is a rapidly progressive disorder causing often total paralysis within a few days. Since the muscles of respiration may also be paralysed, patients need to be admitted to an intensive care ward so that they may be artificially respired until recovery occurs.

A chronic inflammatory neuropathy known as CIDP, is a slowly progressive condition or may follow a relapsing and remitting course similar to multiple sclerosis. CIDP if untreated results in severe paralysis and incapacity.

Researchers in the Neurology Department at Royal Prince Alfred Hospital have been studying GBS and CIDP in an attempt to better understand the cause of these conditions so that more effective therapy may be developed. These disorders affect patients of all ages.

Since GBS causes total paralysis requiring respiratory support in an intensive care unit it is an extremely frightening condition since the brain is not affected and the patients awareness is unimpaired. Moreover, the cost of intensive care therapy is very high, approximately \$2,000 per day. Hence effective treatment would not only spare the patient much suffering but shortening the length of hospital stay would save the community considerable expense.

### Causes and Treatment

Advances in the understanding and management of the conditions have recently followed from the observations that each disorder can be divided into clinical subgroups within which patients show similar clinical features. In these homogenous subgroups distinctive pathogenic mechanisms are beginning to emerge.

For instance in one subgroup of GBS, patients suffer paralysis of the muscles which move the eyes, many have paralysis of the muscles involved in swallowing and they are also very unsteady on walking. This group of patients has been shown to have a circulating antibody, which acts, at the nerve muscle junction to cause paralysis similarly to black widow spider venom.

Researchers within the Neurology department have followed the largest group of patients with the chronic disease CIDP recorded in the literature. They have identified a group of patients with this disorder in whom antibodies can be demonstrated which react with the insulating material around nerve fibres – the myelin sheath. These patients usually can be successfully treated if the antibody is removed by a process called plasma exchange or if it is neutralized by the administration of antibodies harvested from normal subjects (intravenous immunoglobulin). The research workers have shown that the disease causing antibody reacts with a specific protein within the myelin sheath which functions to bind together the many layers of this insulating structure. As a consequence the layers fall apart and the nerve fibres no longer function to faithfully conduct nerve impulses. This finding is very important and the protein in question the P zero (P0) protein is the first proven autoantigen (target of the immune attack) in this group of diseases. This work was recently published in the major neurology journal, *Annals of Neurology*, where it was editorialised.

When disease mechanisms are unraveled in this fashion appropriate treatment strategies can usually then be developed. In this instance where antibodies can be shown and their target identified strategies to remove or neutralize the antibody have been shown to provide effective treatment. However in those groups of patients, which share similar clinical features but have different pathogenic mechanisms, alternate therapies need to be developed.

### Painful Neuropathies

Peripheral nerves each consist of several thousand individual nerve fibres. These vary in size and function. Large fibres convey certain sensations such as vibration or joint position sense and others are motor fibres. The sensation of pain is conveyed in small nerve fibres. Some neuropathies are characterised not by paralysis but rather by pain and it has been presumed that in these conditions the small nerve fibres are at fault. These neuropathies have been very difficult to diagnose since objective clinical findings are minimal and the usual diagnostic test nerve conduction studies, are often normal since these tests examine only the function of the larger nerve fibres.

Researchers within the Department of Neurology have shown that abnormalities of small nerve fibre function can be diagnosed by performing tests of autonomic nerve function (for example, blood pressure changes in the standing and lying position) and by examining the skin.

A small piece of skin is removed by punch biopsy and following appropriate staining methods, the small nerve fibres within the skin can be demonstrated. RPAH research has shown that in many patients with small fibre painful neuropathy there is a highly significant loss of the small nerve fibres within skin compared to normal subjects. This finding has been confirmed even in patients with a normal examination and routine nerve conduction studies. This demonstration of small fibre involvement is the first step in the process of improving the management of this common and disabling condition. In current studies the group is examining the efficacy of new therapeutic agents to relieve the symptoms of painful neuropathy.

**Supported by NH&MRC, The Philip Bushell Foundation**

### [The Concord Hospital Molecular Medicine Laboratory and ANZAC Medical Research Institute and Molecular Neurobiology Laboratory](#)

In 2001 year we studied a number of hereditary neuropathies. We found the gene mutation causing hereditary sensory neuropathy and mapped the chromosomal location of another neuropathy "Intermediate CMT". Hereditary sensory neuropathy (HSN) is the commonest genetic disorder of human sensory nerves. After studying hereditary sensory neuropathy for some years, we mapped the causative gene mutation to chromosome 9 in 1996. This discovery is a world first and was confirmed by our collaborators in Boston in a large American family originating from Germany.

We found a common mutation in an Australian convict family transported from southern England and in three southern English families. Two other English families also had the same mutation. We found a different mutation in an Austrian family. Interestingly, this family comes from a region close to the Austrian-German border and may be distantly related to the American family. Another mutation produces a slightly different phenotype. As the mutations are found in an enzyme, we are creating a mouse model in order to determine whether drug treatments can be devised which can alter the function of the enzyme in a direction which would diminish the effect of the disease. We also mapped the gene locus for a large Intermediate Charcot-Marie-Tooth neuropathy family.

The results of our studies have already translated into practical results. Affected individuals with HSN1 and Intermediate CMT can be accurately diagnosed and preventative treatments can be commenced to avoid complications of these disorders. Affected families can now be offered the possibility of having non-affected children through our genetic diagnostic and counseling clinic.

### [Institute of Clinical Neurosciences - RPAH](#)

#### [Department of Neurophysiology - RPAH](#)

During the year Dr John Walsh, past head of the department, retired. His central interest, the investigation, management and surgical therapy of epilepsy, has been continued in the department with the appointment of Dr Armin Mohammed as a staff specialist. During the year the department has acquired a new system for recording electrical activity in the brain with an ambulatory device that can be worn during normal activities.

This enables us to investigate patients with epilepsy out of hospital as they go about their lives. This system will also enable us to monitor brain wave activity in premature babies in the neonatal intensive care.

The department is active in measuring muscle and nerve parameters in patients with motor neurone disease, neuropathies and myopathies. These measurements enable the standardization of treatments and the investigation of new treatments. In the last year the introduction of Mycophenolate, a drug used for treatment of organ rejection in kidney and heart transplants, has provided significant benefit to a number of patients with chronic inflammatory neuropathies. A muscle paralyzing agent, Botulinum Toxin, has proven very useful in the management of a number of neurological conditions with excess movements such as spasticity and torticollis. This year we have begun using this agent to treat patients with disorders of sweating with considerable success.

During the year Dr Leo Davies and Dr Roger Pamphlett traveled to East Timor at the request of the World Health Organization to investigate a cluster of neurological illnesses amongst prisoners being held in Dili. It was feared that they were being poisoned as an act of revenge but it transpired that they had a variety of neurological illnesses with no common link. This helped to defuse a difficult local situation.

### Epilepsy

Epilepsy is a debilitating neurological condition affecting up to 1% of the population. Epilepsy is the predisposition to seizures and is a chronic medical condition produced by temporary changes in the electrical function of the brain. During seizures there is an interference with the various functions performed by the brain involving movement, awareness, cognition or sensation. There are deep social ramifications associated with epilepsy in terms of driving and employment that define this disease well beyond its medical implications. The majority of patients that have epilepsy that is difficult to manage have seizures originating from the temporal lobes of the brain. There are now many medical and surgical treatments available for epilepsy that allow a majority of patients to live normal productive lives. Neurosurgery performed for the cure of epilepsy is an important treatment in patients with seizures resistant to medications.

Identification of the region of abnormal brain where seizures originate is a difficult but crucial part of the neurosurgical treatment of epilepsy. To this end many tests are carried out some of which are associated with significant risk to the patient.

Positron Emission Tomography (PET) and Single Photon Emission Computed Tomography (SPECT) are methods which image the brain (similar to a CT scan) that use radiation released from compounds injected in the patient. Unlike a CT scan that tells you what the brain looks like, PET or SPECT can tell you what chemicals are in the brain, where they may be found and how these chemicals behave. This would allow us to study a specific chemical in the brain in patients with epilepsy or any other neurological condition. The Departments of Neurology and PET & Nuclear Medicine have been involved in a number of projects to rationalize and develop new methods of imaging the brain in epilepsy.

A review of patients who have been investigated in Royal Prince Alfred Hospital in the last 7 years showed that FDG-PET (looking at energy metabolism of the brain) is extremely useful in patients with epilepsy arising from the temporal lobes. A significant problem in patients with temporal lobe epilepsy is the presence of abnormalities in both the right and left temporal lobes. PET was able to frequently identify the more abnormal temporal lobe without the need for more invasive tests. In addition in many patients whose origin of epilepsy was unknown in the brain, PET was able to identify abnormalities where other tests failed. This would allow treatment for patients with difficult to control epilepsy that would otherwise be left untreated.

Using a group of 65 normal subjects from the age of 22 to 90 as a benchmark, an automated computer method of comparison was used to identify abnormalities in PET scans of epileptic patients. Using this method, abnormal areas of the brain of the epileptic patient would be highlighted by comparing them to a large number of normal subjects.

This has the advantage that an experienced observer is not needed to interpret the scans and the abnormalities identified are statistically tested. Using this method, additional abnormalities were found in patients with epilepsy outside the temporal lobes of the brain. These patients frequently are difficult to treat, as the origin of seizures is not known.

The most exciting project has been the use of a new radioactive ligand in patients with epilepsy. This substance reflects levels of a particular chemical receptor in the brain – the muscarinic acetylcholine receptor. The study continues but in the 16 patients so far tested it has demonstrated that this type of imaging is extremely accurate in predicting the site of seizure origin in the brain. This particular receptor is also important in memory functions and its correlation to cognitive impairment in epileptics will be useful. We have also found the test useful in patients with bilateral temporal epilepsy where it accurately predicts the side of seizure origin. This has been confirmed with intracranial electroencephalographic recordings.

### Department of Neuropathology

Professor Harper was awarded an R24 grant from the National Institute of Alcohol Abuse and Alcoholism which is one of the units of the National Institutes of Health in the USA. This grant will assist with the development and expansion of the NSW Tissue Resource Centre and will lead to new collaborative studies with a number of groups in the USA. The 3 year grant (approximately \$1.2m) will be used to appoint four new staff members and new technical equipment will be purchased for the quality control and study of brain tissue.

### Neurosurgery Department

Neurosurgery changed almost entirely to day-of-surgery with an admission rate of over 75%. This allowed more efficient use of our bed capacity and has improved patient care. Dr. Brian Oowler Neurosurgical Registrar set up a laboratory research program at the University of Sydney to study CSF circulation disorders as well as the role of neuro-chemical monitoring particularly in brain injury.

Clinical research at Royal Prince Alfred Hospital focused on aneurysmal subarachnoid haemorrhage, particularly the biochemical changes within the CSF and the use of doppler monitoring for vasospasm and venous sinus stenting.

A major focus of the Neurosurgery Department was the World Congress of Neurosurgery held in Sydney in September 2001. A number of papers were presented and the Department was actively involved in the Scientific Program.

Planning continues for the opening of a second neurosurgical operating room within the RTP program. Tenders were written for new equipment, including a new operating microscope, and the establishment of Australia's first intraoperative MRI scanner. All patients with aneurysmal subarachnoid haemorrhage in CSAHS will now be treated at RPA H under an agreement reached with Concord Hospital.

Associate Professor Michael Besser continued as an examiner in neurosurgery for the Royal Australasian College of Surgeons. He was appointed to the Editorial Board of the Journal of Clinical Neurosciences and was awarded the Order of Australia (AM).

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# **RESEARCH PUBLICATIONS**

## **PUBLISHED CONFERENCE PROCEEDINGS & ABSTRACTS**

Bell WP, Zavitsanou K, Sarris M, Huang XF. Alterations of membrane phospholipid composition in the anterior cingulate cortex of schizophrenia. Proc. Aust. Neuroscience Soc. 2001,12:135.

Cai W, Feng D, Fulton R. A knowledge-based image smoothing technique for dynamic PET studies. In: Proceedings of IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 18/114-117

Chan-Ling T, Chu Y, Harper C, Hughes S. Absence of astrocyte precursor cells (APCs) and reduced proliferative potential of astrocytes in adult rat and human retina. Society for Neuroscience, San Diego.

Chu Y, Hughes S, Harper C, Chan-Ling T. In vivo characterisation of a lineage restricted astrocyte precursor cell in an oligodendrocyte permissive environment. Society for Neuroscience, San Diego.

Dixon G , Harper CG. Ratio of local circuit neurons in the human anterior thalamus is not altered in schizophrenia. Proceedings of the Australian Neuroscience Society, 2001, 12: 136.

Dixon G, Sarris M, Garrick T, Harper C. The New South Wales tissue resource centre. Journal of Neuropathology and Experimental, Neurology, 2001, 60(5): 532. Presented at the 77<sup>th</sup> Annual Meeting of the American Association of Neuropathologists, Chicago

Fulton RR, Meikle SR, Eberl S, Pfeiffer J, Fulham MJ. Correction for head movements in positron emission tomography using an optical motion tracking system. In: Conference Record of the IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 17/58-62

Garrick TM, Sarris M, Harper CG, Kril J, Pamphlett R. Keeping a 'breast' of schizophrenia. Proceedings of the Australian Neuroscience Society, 2001, 12: 135.

Garrick TM, Sarris M. Harper CG, Kril J, Pamphlett R. Mamillary bodies in alcoholism and schizophrenia.. Alcoholism: Clinical and Experimental Research, Supplement 2001, 25 (5): 452. Presented at Research Society of Alcoholism, Montreal

Hutton BF, Kyme A, Lau YH, Skerrett DW, Fulton RR. A hybrid 3D reconstruction/registration algorithm for correction of head motion in emission tomography. In: Proceedings of IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 15/36-40

Jelinek HF, Davis WS, Harper C. Fractal analysis: A useful and rapid tool in neuropathology. Proceedings of the Australian Neuroscience Society, 2001, 12: 128.

Lerch MLF, Rosenfeld AB, Simmonds PE, Taylor GN, Meikle SR, Perevertailo VL. Spectral characterisation of a blue-enhanced silicon photodiode. In: Conference Record of the IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 7/52-56

Meikle SR, Fulton RR, Eberl S, Dahlbom M, Wong K-P, Fulham MJ. An investigation of coded aperture imaging for small animal SPECT. In: Conference Record of the IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 21/81-85

Sarris M, Arianayagam M, Garrick T Harper C. Regional brain volumes in schizophrenia-a post mortem study. Proceedings of the Australian Neuroscience Society, 2001. 12: 233.

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Sarris M, Konopka M, Lee CS. Differential nm23 protein immunoreactivity in the progression of oesophageal adenocarcinoma. Pathol Internat 2000; 50:A4.

Takacs GJ, Rosenfeld AB, Lerch MLF, Taylor GN, Meikle SR, Eberl S, Simmonds PE, Perevertailo VL, Allen BJ. Design and simulation of scintillator-pixel photodetector with the ability to measure position of interaction. In: Conference Record of the IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 20/20-24

Wong KP, Feng D, Meikle SR, Fulham MJ. Non-invasive extraction of physiological parameters in quantitative PET studies using simultaneous estimation and cluster analysis. In: Conference Record of the IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 18/141-145

Wong KP, Feng D, Meikle SR, Fulham MJ. Segmentation of dynamic PET images using cluster analysis. In: Conference Record of the IEEE Nuclear Science Symposium and Medical Imaging Conference, Lyon: IEEE, pp 18/126-130

## **RESEARCH PUBLICATIONS**

### **UNPUBLISHED ABSTRACTS – INVITED LECTURES AND SEMINARS**

Armati PJ, Schwann cells, cytokines and antigen presentation, Seminar Series, School of Anatomy, University of NSW, Sydney

Armati PJ, Trapezoids and octopuses - how to be very flat, coiled and important: the truth about myelin forming cells". Myelinators Meetings, University of Sydney, (Invited speaker and Chair)

Armati PJ, Apolipoprotein E and Alzheimer's Disease, GlaxoWellcome Investigators Meeting, Melbourne

Armati PJ, How can confocal microscopy help Alzheimer's Disease?, Key Centre for Microscopy and Microanalysis, Sydney

Besser M., How I Do it - Carotid Endarterectomy, World Congress, Sydney

Besser M., Brainstem Tumours, World Congress, Sydney

Besser M., Parasagittal Meningioma, World Congress, Sydney

Dawkins JL, Hulme DJ, Brahmabhatt SB and Nicholson GA. Positional cloning of the hereditary sensory neuropathy type I (HSN1) gene. 22<sup>nd</sup> Lorne Genome Conference, Lorne, Victoria.

Dixon G, Sarris M, Harper C. Better brain banking – issues of the tissues. 2<sup>nd</sup> Asian Pacific Forum for human brain banking. Fukushima, Japan

Garrick T, Sarris M, Harper C, Kril J, Pamphlett R. Mamillary Bodies in Schizophrenia. Australian Society for Psychiatric Research Annual Scientific Meeting, Melbourne

Harper C. The effects of alcohol and nutritional defecits on the brain. Dept. of Psychiatry and Behavioural Sciences and Neuroscience Program, Stanford University School of Medicine

Harper C. Are we damaging our brains with alcohol? -Neurology Grand Rounds, University of Rochester Medical Center

Harper, C. Is Mad Cow Disease in Australia? Prion diseases. 19<sup>th</sup> Review and Recent Advances in Pathology, Royal Prince Alfred Hospital

Harper, C. An update on the neuropathology of Creutzfeldt-Jacob disease, Southern Neurology, St. George Hospital

Harper C. Convenor - Australian Postgraduate Neuropathology Course, Sydney, January.  
Sarris M. Schizophrenia and the brain, Kogarah High School

Kennerson ML, Rizos H, Nassif NT and Nicholson GA. Cellular localisation of C170RF1A: a primate specific gene associated with the disease causing CMT1A-REP binary repeat. 22<sup>nd</sup> Lorne Genome Conference, Lorne, Victoria.

Kennerson ML, Zhu D, Garner RJM, Storey E, Merory J, Robertson SP and Nicholson GA Linkage Analysis in dominant intermediate Charcot-Marie-Tooth neuropathy (DI-CMT). American Society of Human Genetics, San Diego, California.

McLeod JD, Update on cerebrovascular disease. 20<sup>th</sup> International Congress of Life, Disability and Health Assurance Medicine, Sydney

Pollard JD Management of MS and the Management of Demyelinating Disease ASNA meeting, Kuala Lumpur, Malaysia, Association of South East Asian Neurological Associations

Pollard JD New Ideas about peripheral neuropathies, Royal Australian College of Physicians, Darling Harbour, Sydney

Pollard JD Current Research and New Treatments for MS, MS Information Forum, Sydney

Pollard JD Diagnosis and Management of Chronic Inflammatory Demyelinating Polyradiculopathy, World Congress of Neurology, London, UK

Pollard JD Sudden Paraplegia, World Congress of Neurology, London, UK

Sarris M , Arianayagam M , Garrick T, Harper C. Sub-regional quantitative analysis of brain volumes in schizophrenia. Australian Society for Psychiatric Research Annual Scientific Meeting, Melbourne

Sarris M, Boyes, M. Garrick T, Harper, C. The NSW Tissue Resource Centre – “Just the first steps in a marathon”. 2<sup>nd</sup> Asian Pacific Forum for human brain banking. Fukushima, Japan

## **RESEARCH PUBLICATIONS**

### **BOOK CHAPTERS**

Davies L. and Clouston P., Neuropathies in Malignant Disease Chapter 10 (pp182-196) Cros D. (ed) Peripheral Neuropathies Springer Verlag NY

Harper C, Duckett S. Malnutrition and Alcoholism in the Aging Population. In: The Pathology of the Aging Nervous System. 2<sup>nd</sup> Edition Eds. Duckett S and de la Torre J. Oxford University Press.

Harper C, Butterworth RF. Nutritional and metabolic disorders. In: Greenfield's Neuropathology. 7th edition, . Lantos P, Graham D (Eds) Edward Arnold, Cambridge.

Harper CG, Scolyer, R. Alcoholism and dementia. In: The neuropathology of dementia. 2<sup>nd</sup>. edition. Esiri M , Morris H, Trojanowski J (Eds) Cambridge University Press, Cambridge.

Pollard JD, Spies JM. Immune mediated neuropathies. In: Diseases of the nervous system 3<sup>rd</sup> Ed. Cambridge University Press 2001. Asbury AK, McKhann G, McDonald W, Goadsby P, McArthur J Eds

Wong K-P, Feng D, Meikle SR, Fulham MJ. Validation of noninvasive quantification technique for neurologic FDG-PET studies. In: Molecular and pharmacological brain imaging with positron emission tomography Gjedde A, Hansen SB, Knudsen GM et al. eds, Academic Press:121-125

## RESEARCH GRANTS

<u>Title</u>	<u>Granting Body</u>	<u>\$2001</u>
The role of antibody in inflammatory demyelinating neuropathy JD Pollard, JM Spies	NH&MRC	\$71,181
Myelin repair by neural stem cells genetically modified to secrete growth factors TGF-B & IGF-1 J Pollard, R Taylor	NH&MRC	\$64,651
Role of kynurenine pathway metabolites in the pathogenesis of the AIDS dementia complex B Brew, P Armati	NHMRC	\$87,942
Forward transport of Herpes simplex virus in human nerve cells is mediated by interactions with motor proteins. A Cunningham, P Armati, R Dieffenbach	NHMRC	\$79,253
Finding the gene for Hereditary Sensory Neuropathy: a new cause of sensory neurone degeneration G Nicholson, M Kennerson	Rebecca L. Cooper Foundation	\$16,000
Functional studies on the gene causing hereditary sensory neuropathy G Nicholson, M Kennerson	Ramaciotti Foundation	\$12,000
Gene mutation screening in Parkinson's Disease G Nicholson	The Australian Brain Foundation	\$10,000
Genetic bases for Charcot-Marie-Tooth and Hereditary Sensory type I Neuropathies G Nicholson, M Kennerson	NH&MRC	\$615,000 (2001-2003)

## RESEARCH GRANTS

<u>Title</u>	<u>Granting Body</u>	<u>\$2001</u>
Phosphoimager screens and cassettes for triated binding studies C Harper	Blackburn Precinct Grant	\$6,700
Schizophrenia & Alcoholism C Harper	National Institute of Alcohol Abuse and Alcoholism (USA), Brain Tissue Resource Center for Alcohol Research, 2000-2002	US\$690,110
A Mohamed	Brain Foundation	\$10,000
A Mohamed	Glaxo Smith Kline research grant	\$10,000
Antibody & Complement M Barnett	Schering	\$35,000
Neurophysiological Studies Of the Vestibular System M Halmagyi	Schering	\$35,000

## EQUIPMENT GRANTS

<u>Title</u>	<u>Granting Body</u>	<u>\$2001</u>
Centrifuge J Pollard	Ramaciotti Foundation	\$12,000

## **BENEFACTORS**

### **INDIVIDUALS**

Mr J Armati OAM  
Mr J Anderson  
Mr & Mrs E & R Barnum  
Mr & Mrs J & P Bradwell  
Bradstreet Family  
Mr E Cigna  
Mr G Clarke  
Mr B Dalley  
Mrs E Davis  
Mr & Mrs L & P Dekroon  
Lady Mary Fairfax  
Mrs R Forge  
Mr M Gazal  
Mr B Hicks  
Mr & Mrs A & S Jansen  
Dr R Kerr

Dr V Khurana  
Mr C Knoblanche  
Ms G La Pointe  
Mr & Mrs I & C Langsford  
Mr J L'Estrange  
Mrs R McLeod  
Mr & Mrs B & K McFadyen  
Mr & Mrs R Melick  
Ms R Morden  
Nicholson Family  
Mr & Mrs R & B Pfeiffer  
Mrs M Shackman  
Mr & Mrs I & C Singleton  
Ms K Steffas  
Dr J Walsh  
Mr & Mrs A & A Yeomans

### **CORPORATIONS & ASSOCIATIONS**

Aventis Pharma  
Baker & Mackenzie Solicitors  
Biogen  
Cellarmasters  
Guillain Barre Association  
Macquarie Hockey Club  
Macquarie Portfolio Investments P/L  
Mazzaro Restaurant  
Observatory Hotel

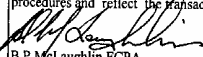
Park Hyatt Sydney  
Peppers Retreats and Resorts  
Perpetual Trustees  
Pilates Moves  
Schering P/L  
Shaw & Smith  
Sydney City Lexus  
Sydney Tower Restaurants

The University of Sydney  
Nerve Research Foundation

Statement of Income and Expenditure  
for the year ended 31 December 2001

	31 December 2001 \$	31 December 2000 \$
<b>INCOME</b>		
Grants and HECS	98,195	57,981
Scholarships/Donations/Bequests	302,160	70,500
Business & Investment Income	74,081	63,867
Fees & Charges	177,639	101,545
Other Income	9,847	288,492
<b>Total Income</b>	<b>661,922</b>	<b>582,385</b>
<b>EXPENDITURE</b>		
Staff payroll	332,931	214,578
Consumables	4,001	7,177
Equipment & Repairs/Maintenance	15,166	10,680
Services/Utilities	(24,095)	965
Travel/Conferences	16,384	8,979
Staff training	65	310
Other expenses	134,215	218,331
<b>Total Expenditure</b>	<b>478,667</b>	<b>461,020</b>
<b>SURPLUS FOR THE YEAR</b>	<b>183,255</b>	<b>121,365</b>
Total Accumulated Funds as at 1 January	955,507	834,142
<b>TOTAL ACCUMULATED FUNDS as at 31 December</b>	<b>1,138,762</b>	<b>955,507</b>

I certify that the accounts have been prepared in accordance with the University's accounting practices and procedures and reflect the transactions as recorded in the University's general ledger.

  
B P McLaughlin FCPA  
Manager (Finance & Resources)  
College of Health Sciences  
March 20, 2002

The University of Sydney  
Nerve Research Foundation

Balance Sheet  
as at 31 December 2001

	31 December 2001 \$	31 December 2000 \$
<b>ASSETS</b>		
<b>Current Assets</b>		
Prepayments	16,914	500
Investment-Cash Balance	1,121,848	755,007
<b>Total Current Assets</b>	<b>1,138,762</b>	<b>755,507</b>
<b>Fixed Assets</b>		
Growth Fund Investment Pool	200,000	200,000
<b>Total Fixed Assets</b>	<b>200,000</b>	<b>200,000</b>
<b>Total Assets</b>	<b>1,338,762</b>	<b>955,507</b>
<b>NET ASSETS</b>	<b>1,338,762</b>	<b>955,507</b>
<b>EQUITY</b>		
Accumulated Funds	1,138,762	955,507
<b>TOTAL EQUITY</b>	<b>1,138,762</b>	<b>955,507</b>

Notes to the Financial Statements  
for the reporting period ended 31 December 2001

1. Statement of Significant Accounting Policies

- (a) These accounts have been prepared on cash basis and amounts are stated at historical cost.
- (b) Income tax is not applicable to activities of the Foundation.