Epilepsy

Perception, imagination and change

Medical History Museum, University of Melbourne

Epilepsy is one of the most common neurological conditions, affecting one in 26 people during their lifetime. Despite its prevalence, epilepsy has long been misunderstood, immersed in superstition, fear and prejudice. In the 19th century neurologists began to understand the causes of the disorder, although misleading terms such as 'the dreamy state', 'psychic seizures' and 'double consciousness' prevailed. Even today, fear and misinformation affect the daily lives of many people with this condition.

Epilepsy: Perception, imagination and change explores different cultural and historical perspectives on epilepsy, and includes accounts from scientists and physicians whose discoveries are improving the quality of lives of people with epilepsy. Both the book and the exhibition held at the University of Melbourne's Medical History Museum in 2014 embrace research undertaken by Dr Jim Chambliss in collaboration with artists who have epilepsy. Many of their works are reproduced here, accompanied by their own descriptions of their art and its place in their lives.

This book increases our understanding and awareness of epilepsy. The history affects our perceptions of the condition, the artwork shows the power of the artists' imaginations, and the innovative work of leading researchers points to the changes ahead.

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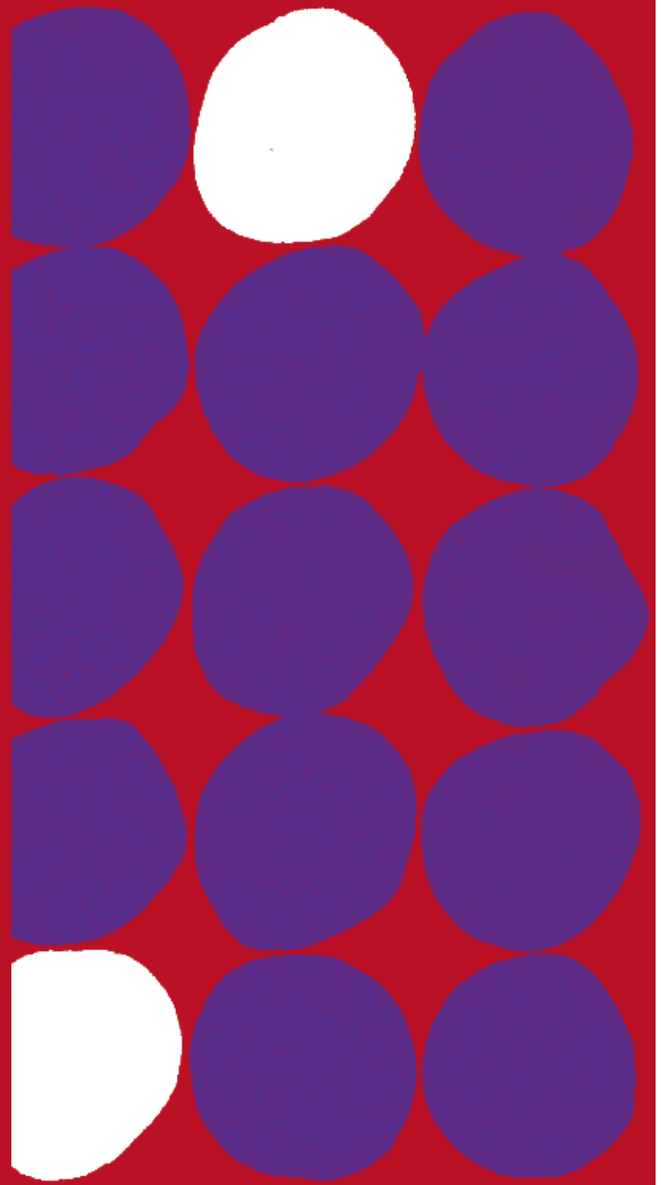

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Perception, imagination and change

Edited by
Jim Chambliss, Mark Cook and Jacqueline Healy

Medical History Museum, University of Melbourne

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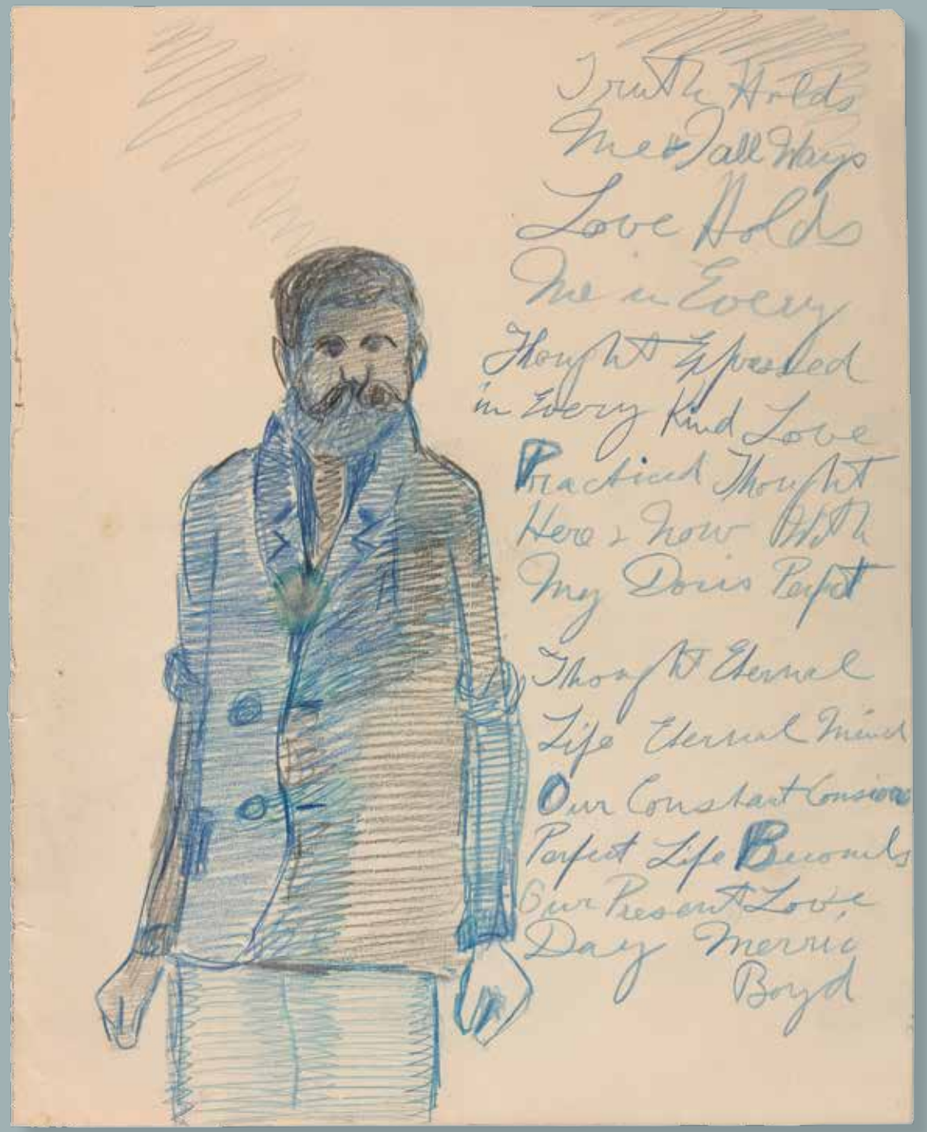
Cat. 100 **Phrenology bust**, c. 1850–1914; porcelain, ink, brass, wood; 9.5 × 5.0 × 5.0 cm. Gift of Alan Attwood, Medical History Museum, MHM04392, University of Melbourne.

Contents

Cat. 62 Merric Boyd, ***Truth holds me***, pencil on paper, 31.3 × 24.5 cm. 95-0509-001-01, Bundanon Trust Collection.



Foreword

Epilepsy, called 'the sacred disease' by Hippocrates, has presented complex social and medical issues since first identified. For centuries people with this condition have been ostracised from their communities due to a lack of understanding of their condition. Today, new treatments, practices and community education have resulted in far better outcomes for patients and their families.

The University of Melbourne is an international research leader in epilepsy and this exhibition and catalogue bring together past, current and future endeavours in this field. This exhibition at the Faculty's Medical History Museum was curated in consultation with Professor Mark Cook, Sir John Eccles Chair of Medicine and Director of Neurology at St Vincent's Hospital, Melbourne, and marks the 50th anniversary of the Epilepsy Foundation in Victoria. The accompanying catalogue, *Epilepsy: Perception, imagination and change*, covers cultural and historical perspectives in Australia and Asia to establish a context for the remarkable breadth of current innovations. It presents the range of treatments and recent discoveries including medical, surgical and genetic approaches. A key aspect of this exhibition is the inclusion of works of art by artists with epilepsy. The artists, through their statements, share with us the personal challenges faced by individuals with this condition. Their creativity is inspiring.

This publication brings together the views of people with epilepsy, prominent members of the medical profession, the broader research community and important advocacy organisations such as the Epilepsy Foundation. All represent key aspects of the story of epilepsy from varied historical, medical and personal perspectives. I thank them all for their contributions.

I also sincerely thank UCB Australia Pty Ltd for proudly sponsoring this very significant exhibition and catalogue at the University of Melbourne, which celebrate the rich legacy of medical history and current innovative research in this field.

Professor Stephen K Smith
Dean, Faculty of Medicine, Dentistry and Health Sciences
University of Melbourne

Cat. 118 Italian, **Majolica drug jar for essence of *Aconitum napellus* (monk's hood or wolf's bane)**, late 18th – 19th century, earthenware, 14.3 × 11.8 cm diameter, inscribed *ES. ACCONito NAPElli*. Gift of the estate of Graham Roseby, 2009. MHM2009.42, Medical History Museum, University of Melbourne.

Sponsor's message

UCB is proud to be a supporter of the University of Melbourne and the creation of this substantial book, *Epilepsy: Perception, imagination and change*.

Established in 1928, UCB was known for many years for its diversified pharmaceuticals, chemicals and films expertise. Since 2004, visionary leaps and agile changes have transformed UCB from a leader in the chemical sector into a leading biopharmaceutical company, featuring a development portfolio of small- and large-molecule drugs focusing on immunology and the central nervous system.

Our promise is to help tackle the serious unmet medical needs affecting hundreds of thousands of patients around the globe. This is what drives us every day to never give up on developing innovative medicines in our therapeutic areas of focus: central nervous system and immunology disorders. In recent years we have brought several new therapies to patients living with severe diseases in these areas. Each UCB colleague across the world contributes throughout the activity chain, from early research through to manufacturing and sales, to ensure we provide meaningful solutions to patients and their families.

An important part of our philosophy is to take a holistic approach to patients, and we aim to find solutions tailored to their circumstances. UCB hopes you enjoy reading this comprehensive book on the history and treatment of epilepsy.

Arnaud Lefevre
Managing Director, Australia and New Zealand
UCB Australia Pty Ltd

Cat. 30 Shea O'Keefe (Australia), ***A time for rest***, 2004, pen and pencil on monoprint, 48.5 × 48.0 cm. UCB Collection.

Preface

At the inaugural meeting of the National Council of Women in October 1903, Dr Mary Page Stone (1865–1910) gave a paper on the suffering of people with epilepsy and the need for epileptic 'colonies'. Her presentation so moved the congress that epilepsy became a major issue for the National Council of Women. The result was the establishment of the Talbot Colony for Epileptics. The 1893 student photograph of Dr Mary Page Stone in the Medical History Museum Collection is one of the few surviving images of her. It is most fortunate that in bringing together artists, medical practitioners and historians to evoke the major milestones in the story of epilepsy her story has been uncovered, enabling us to trace the involvement of Melbourne Medical School alumni as one of the earliest Australian milestones.

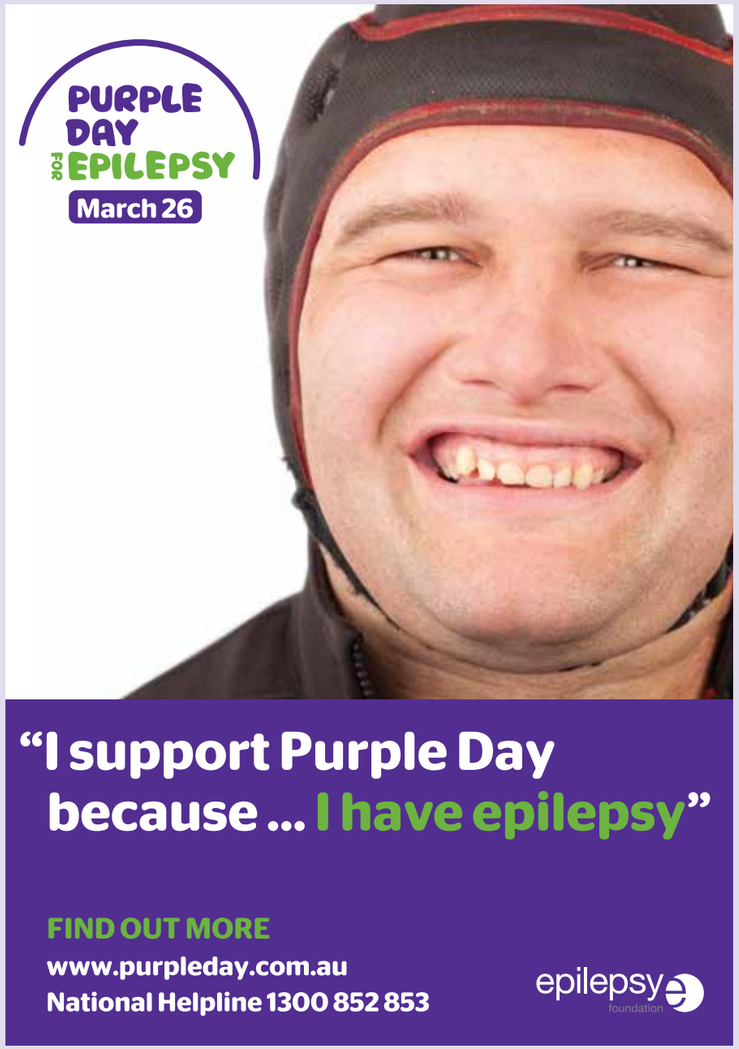
Established by a grant from the Wellcome Trust, the Medical History Museum opened in April 1967. An important collection in the university, it holds over 6000 items covering the history of the Melbourne Medical School, the Australian Medical Association and medicine generally in Australia and internationally. The Medical History Museum partners with research areas across the University of Melbourne and other research institutes to present its exhibitions and publications program. Under the leadership of Professor Mark Cook, Sir John Eccles Chair of Medicine and Director of Neurology at St Vincent's Hospital, Melbourne, curators Dr Jim Chambliss (who recently completed a PhD on art and epilepsy) and Dr Jaqueline Healy (Curator, Medical History Museum), have brought together photographs, archival material and artworks to share historical and current viewpoints.

This exhibition *Epilepsy: Perception, imagination and change* includes artworks from the artists' collections, private collections, corporate collections and the Bundanon Trust Collection. Artefacts, documents and ephemera from the Medical History Museum, Epilepsy Foundation, Oakleigh and District Historical Society, St Vincent's Hospital Archives, State Library of Victoria and the Walter and Eliza Hall Institute Archives are included. I thank all the lenders, in particular the artists, who have participated in this exhibition.

The Talbot Colony for Epileptics operated from 1907 to 1961 on the site in Clayton now occupied by Monash University. Its closure was the catalyst for the establishment of the Epilepsy Foundation, which marks its 50th anniversary this year, and continues to advocate for the rights of people with epilepsy. This catalogue and exhibition tell of many inspiring people, challenges and achievements.

Professor James D Best
Head of Melbourne Medical School
University of Melbourne

Cat. 105 **Mary Page Stone** as a student, in Talma Studios, Melbourne (photographer), ***5th year medical students 1893***, 1893, photograph and ink on card, 54.2 × 64.6 cm. MHM00511, Medical History Museum, University of Melbourne.



Epilepsy: Perception, imagination and change

Epilepsy has for centuries been a misunderstood condition immersed in superstition and prejudice. The English neurologist John Hughlings Jackson (1835–1911) termed aspects of focal epilepsy the ‘dreamy state’, ‘psychic seizures’ and ‘double consciousness’. Prior to this, people with epilepsy were viewed through the vortex of religious revelation, possession or insanity. Even today, apprehension and misinformation deeply affect the daily lives of people with this condition.

The Medical History Museum’s purpose is to encourage appreciation and understanding of the history of medicine and its role in society through direct engagement with the collections. It stimulates active learning about the history of medicine through research, teaching and dialogue among communities of students, faculty, scholars, alumni and the wider public. This exhibition and catalogue bring together neurologists, historians, key organisations and people with epilepsy to explore past, present and future understandings of epilepsy’s causes, effects and treatments.

By exploring cultural and historical perspectives on epilepsy at different times and places we can better understand deep-seated views and biases. David Castle’s essay on stigma comments on the history of epilepsy being ‘4000 years of ignorance, superstition and stigma followed by 100 years of knowledge, superstition and stigma’. Despite Hippocrates and his school in fifth-century Greece recognising that epilepsy was a physical illness, as outlined by Edward Byrne, general scientific and community understanding of this took many more centuries to achieve. Indeed, there is still progress to be made: Chong-Tin Tan argues that many of the names given to the condition in South-East Asia are weighted with stigma and need to be replaced.

Neurologist and historian Peter Bladin takes us through a sobering history of epilepsy in Victoria. Items from the Medical History Museum such as the post-mortem instruments case from the Yarra Bend Asylum remind us of the fate of many people with epilepsy in 19th-century Australia, who were interned in prisons or asylums. In 1907, circumstances improved in Victoria with the establishment of the Talbot Colony for Epileptics, where residents were educated and provided with employment opportunities. Evocative early photographs of the colony from the Epilepsy Foundation Collection are in the exhibition.

Crucial to change in this area were treatments and new discoveries. Gregory Cascino from the Mayo Clinic provides an illuminating overview of the extraordinary rate of change in this field, while Frank Vajda maps the contribution of medical therapies. Leading authorities from the University of Melbourne, Terence J O’Brien and Patrick Kwan, outline

Cat. 86 Epilepsy Foundation, ***Purple day for epilepsy, March 26***, 2014, poster, print on paper, 30.0 × 21.0 cm. Epilepsy Foundation Collection.

innovations in surgical treatments, while Sam Berkovic and Ingrid Scheffer discuss research in genetics. Mark Cook, Sir John Eccles Chair of Medicine and Director of Neurology at St Vincent's Hospital, Melbourne, believes epilepsy is one of the worst neurological conditions. His essay on future directions presents new research and techniques to predict seizures, empowering individuals to manage their condition.

Important milestones show the influence of individuals, innovations and organisations. For example, Colin Smith writes about the intriguing relationship between Dr JM Springthorpe and the artist Merric Boyd. Boyd's family bought him a property in Murrumbeena so he would be close to a doctor who would treat his epilepsy in a sensitive manner. Works by Boyd from the Bundanon Trust show the power of his imagination. Neuroradiologist Patricia Desmond writes about the significance of the first brain scan; such breakthroughs are illustrated by photographs of early EEG and CAT scan machines from the St Vincent's Hospital Heritage Archives. Advocacy, activism and practical support by and for people with epilepsy are important to the history of the condition in Victoria. Jeremy Maxwell explains the political and community significance of the establishment of the Epilepsy Foundation—which marks its 50th anniversary this year—in promoting the rights of people with epilepsy. The minutes of one of the Foundation's first meetings and its inaugural magazine, *Expectations*, now in the Epilepsy Foundation Collection, are included in the exhibition.

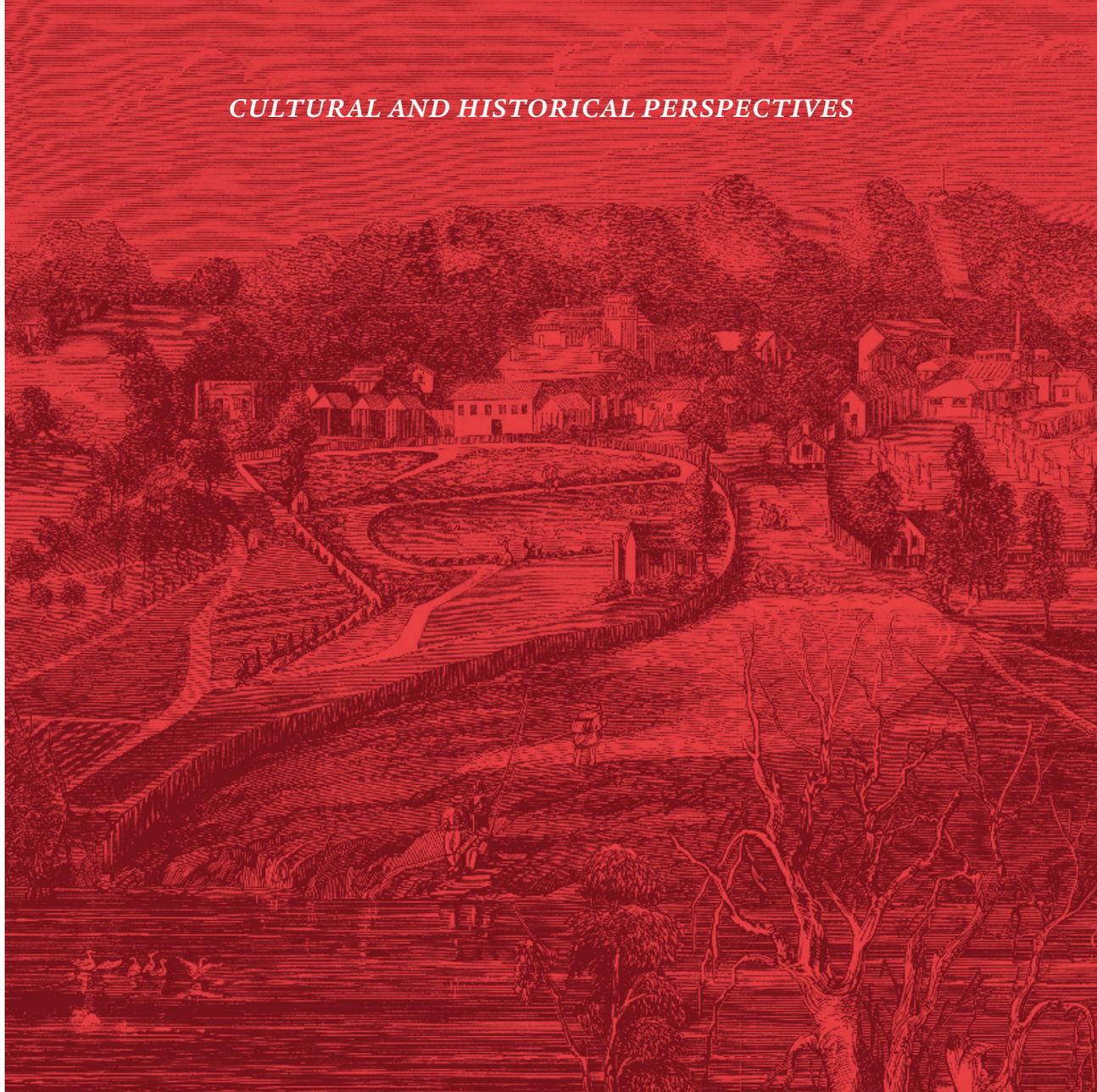
A key part of the exhibition and catalogue is research undertaken by Jim Chambliss in collaboration with artists. Jim was recently awarded what we believe is the world's first PhD in creative arts and medicine, by the University of Melbourne. The complexity of his thesis involved traversing these two disciplines with the cooperation of the University's School of Culture and Communication, the Department of Medicine at St Vincent's Hospital and the Epilepsy Foundation. Studying the work of over 100 artists with epilepsy, Jim discovered that more than 90 per cent of the artists with focal epilepsy had experienced what he terms 'intrinsic perceptions' and integrated these fascinating images and experiences into their art. Thirty-seven artists are represented in the exhibition, and the works of many are reproduced in this publication, the majority accompanied by their own descriptions of their art and its relationship to their lives.

This exhibition and catalogue increase our understanding and awareness of epilepsy. The history affects—even today—our perception of the condition, the artwork shows the power of the artists' imaginations, and the innovative work of leading researchers points to the changes ahead.

Dr Jacqueline Healy
Curator, Medical History Museum
Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne

Cat. 123 Frederick Grosse (engraver), ***The Yarra Bend Asylum for the Insane*** (detail, colour altered), wood engraving in *The Illustrated Australian News*, 23 May 1868. IAN23/05/68/12, courtesy State Library of Victoria.

CULTURAL AND HISTORICAL PERSPECTIVES



Hans Baldung (printmaker), Martin Flach the younger (printer), ***St Valentine***, c. 1511, woodcut on paper, 6.0 × 4.6 cm. British Museum. Help from the physician St Valentine was evoked in cases of epilepsy.

Stigma and epilepsy

I remember so vividly seeing, for the first time, someone having an epileptic seizure. I must have been about eight years old and we were in the change-rooms at school, getting dressed after gym. One of my classmates started to make weird laughing-like sounds. (He had a form of epilepsy in which this was part of the seizure: so-called 'gelastic' epilepsy.) Everyone fell silent and stared at him. His arms then started to flail about and he half-ran, half-fell across the floor, then collapsed. His arms and legs convulsed for about 30 seconds, then stopped and he just lay there. Soon one of the teachers arrived and my classmates and I were moved away and back to the classroom.

Why do I have such a vivid recall of this event, after so many years, having witnessed numerous seizures and even having had to manage status epilepticus? (I studied neurology, and even neurosurgery for a brief period, before moving into psychiatry training.) Well, clearly one reason is that it was all so unexpected and frightening: we simply didn't know what was going on and the whole event was traumatic for all of us. But I suppose more important, and relevant to this article, is the fact that I, for one, saw my classmate in a different way after that. I was wary of him, embarrassed and avoidant and had the thought that if I got too close to him or if his saliva touched me, I might also get epilepsy.

So, fear and ignorance drove my response and it was only after a long discussion with my mother, who happened to be a professor of neurology and who had a particular interest in gelastic epilepsy, that I started to understand it more. My mother told me some key facts, such as that epilepsy is not contagious. She also reminded me that my childhood hero, Alexander the Great, had epilepsy, as did a number of other famous historical figures (Julius Caesar, Peter the Great, Lord Byron, Vincent van Gogh). This was helpful enough, but what really affected me was when she challenged me to think about the event I have described from my classmate's point of view, to empathise and understand how he would respond to a sudden change in my behaviour towards him.

I believe this experience gave me some early insight into the whole issue of stigma and epilepsy. As a psychiatrist, I am very much aware of the stigma associated with so many mental illnesses and am conscious of the negative effects this can have on the lives of people with mental illness, as well as on their family members. It may be that it is because brain disorders and mind disorders are so poorly understood that they are more likely than many physical illnesses to carry the burden of stigma.

Stigma refers to 'mark': being marked out from others and being perceived as different. Erving Goffman is often quoted in this regard; he suggested that stigma is driven by persons being perceived as 'not quite human',[1] and thus, as Feist *et al*. have it, 'are therefore, in the minds of others, fair targets for stigma and prejudice'.[2] The consequence is an ostracising of the individual as a social misfit, such that certain attributes of the

individual 'serve to spoil one's own social identity'.[3] Jacoby suggests that the basis for stigma lies in an underlying biological need to live in effective groups: people who do not conform become targets of attack as they are seen as not conforming to societal norms.[4]

In reference to epilepsy, Bandstra and colleagues quote Kale on the history of epilepsy being '4000 years of ignorance, superstition and stigma followed by 100 years of knowledge, superstition and stigma'.[5] In a similar vein, they quote the Epilepsy Foundation: 'Take one cup of ignorance and blend in a heaping teaspoon of fear. Let the ingredients stand for about 5000 years and then wait for discrimination to rise'.[6]

Stigma regarding epilepsy does indeed have a very long history, reaching across human cultures. For this overview, I have relied heavily on a paper entitled 'The history and stigma of epilepsy', published in 2003.[7] In his celebrated book *The falling sickness* Owsei Temkin outlined how the ancient Mesopotamians equated seizures with the god of the Moon: an often-repeated suggestion of a linkage between maladies of the brain or mind and lunar cycles.[8] The Code of Hammurabi (1780 BCE) outlawed marriage for people with epilepsy and disallowed them from testifying in court.[9]

In Ancient Greece, epilepsy was considered somehow divine: the Oracle at Delphi entered a trance by breathing the smoke of certain burnt plants, and often convulsed or at least collapsed in the process, after uttering the prophecy. Hippocrates in around 400 BCE remonstrated against this belief, stating: 'The alleged divine character is only a shelter for ignorance and fraudulent character'. He recognised epilepsy as a brain disease and believed that it could and should be treated 'not by magic, but by diet and drugs'.[10]

Biblical references to epilepsy are also to be found. St Mark's Gospel describes a child having a seizure and reasons that it was the manifestation of 'a spirit of dumbness'. This led to the general view in Christian circles that epilepsy was caused by an 'unclean dumb and deaf spirit' and that 'epileptics were demoniacs'.[11] People with epilepsy were segregated from the rest of the congregation in church, in the belief that their saliva or even breath could infect others: a response redolent of early concerns about people with HIV/AIDS, who were in some jurisdictions precluded from drinking from the same communion cup as other worshippers. One 15th-century writer warned: '… neither talk nor bath with [the person with epilepsy] since by their mere breath they infect people'.[12]

It was at the time of the Enlightenment in the late 17th century that people started seriously to question prevailing negative views about epilepsy. This period of scientific scrutiny and reductive reasoning also saw, alongside a general increase in humanitarian attitudes, advances in care for people with mental illness. Growing knowledge and understanding led to a less prejudiced and pejorative view of people with epilepsy.

But clearly the story does not end there, even in terms of legislated discrimination against people with epilepsy. Indeed, in the United States a number of states passed legislation preventing people with epilepsy from marrying, with such a law remaining on the statutes in one state until as recently as 1980; in the United Kingdom a similar law was repealed only in 1970. Again in the United States, laws allowing certain dining and recreational establishments to deny entry to people with epilepsy were revoked only in the 1970s.[13]

Gains have been made in terms of a reduction of legislative discrimination against people with epilepsy. The International League Against Epilepsy quotes a 1987 ruling by the United States Supreme Court: 'a review of the history of epilepsy provides a salient example that fear, rather than handicap itself, is the major impetus for discrimination against people with handicaps'.[14]

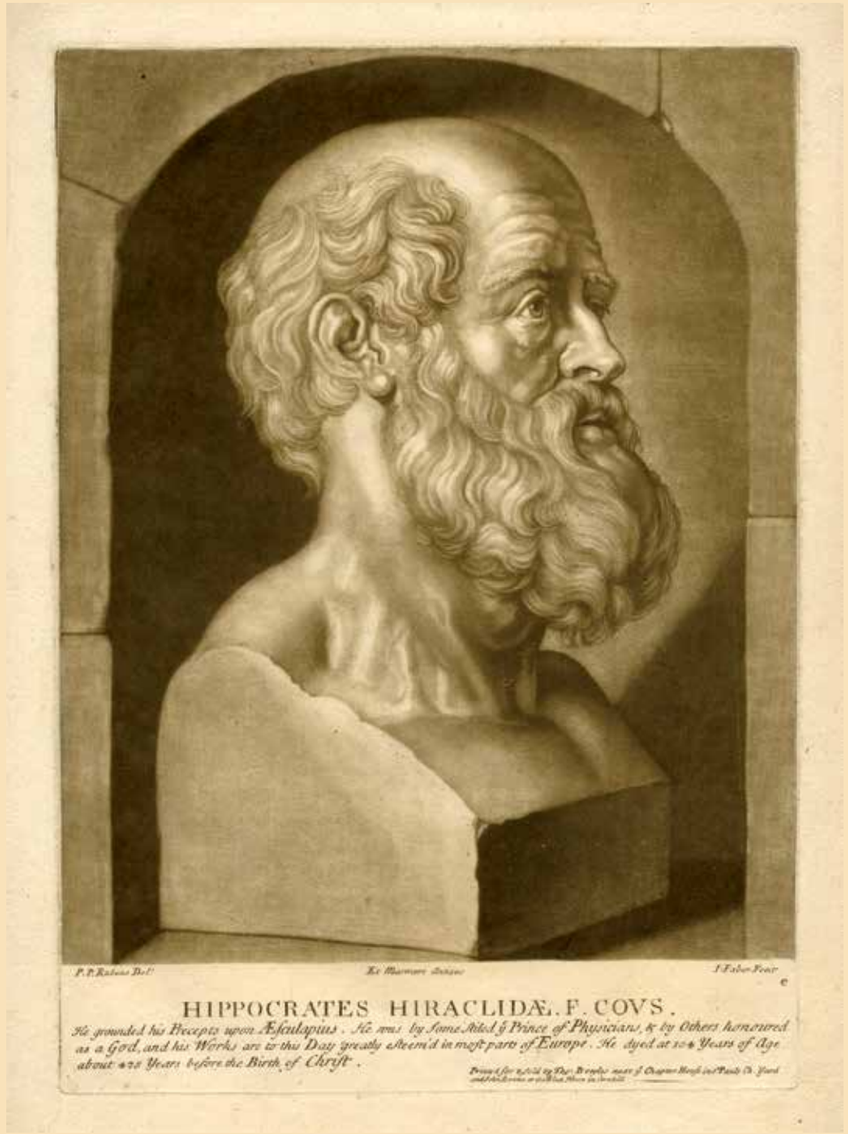
Yet stigma still exists, with many people who have epilepsy perceiving themselves as stigmatised and many people in the general population still believing, for example, in an 'epileptic personality', associated with aggression, hyper-religiosity and hypersexuality.[15]

What can we do to continue to battle stigma against people with epilepsy? How can we foster understanding and acceptance in people such as me as an eight-year-old, witnessing my classmate's seizure? The Institute of Medicine suggests that a major plank in such an endeavour is educating the public about epilepsy; it also supports media-watch schemes under which pejorative statements and images relating to epilepsy are monitored and pressure is brought to bear on media and others not to publish such material.[16] The International League Against Epilepsy suggests encouraging well-known people with epilepsy to come forward and become role models: similar campaigns have helped destigmatise depression, for example.[17]

Fear and lack of knowledge are powerful drivers in all human beings, and tend to cause us to react in a way that at some basic level makes us feel safe. Stigma against certain groups can result. We all need to be aware of these potential responses in ourselves and others and to endeavour, through word and deed, to ameliorate them.

Professor David Castle

1 E Goffman, *Stigma: Notes on the management of spoiled identity*, Englewood Cliffs, NJ: Prentice-Hall, 1963.
2 KM Fiest *et al*., 'Stigma in epilepsy', *Current Neurology and Neuroscience Reports*, vol. 14, no. 5, May 2014, pp. 444–9.
3 Goffman, *Stigma*.
4 A Jacoby, 'Epilepsy and stigma: An update and critical review', *Current Neurology and Neurosicence Reports*, vol. 8, issue 4, July 2008, pp. 339–44.
5 NF Bandstra *et al*., 'Stigma of epilepsy', *Canadian Journal of Neurological Sciences*, vol. 35, issue 4, September 2008, pp. 436–40, quoting R Kale, 'Bringing epilepsy out of the shadows: Wide treatment gap needs to be reduced', *British Medical Journal*, vol. 315, issue 7099, 5 July 1997, pp. 2–3.
6 Epilepsy Foundation, 2007, quoted in Bandstra *et al*., 'Stigma of epilepsy'.
7 International League Against Epilepsy, 'The history and stigma of epilepsy', *Epilepsia*, vol. 44, supplement 6, 2003, pp. 12–14.
8 O Temkin, T*he falling sickness: A history of epilepsy from the Greeks to the beginnings of modern neurology*, Baltimore: Johns Hopkins Press, 1945.
9 International League Against Epilepsy, 'The history and stigma of epilepsy'.
10 *Ibid*.
11 *Ibid*.
12 Quoted in Temkin, *The falling sickness*.
13 Fiest *et al*., 'Stigma in epilepsy'.
14 International League Against Epilepsy, 'The history and stigma of epilepsy'.
15 A Jacoby *et al*., 'Epilepsy and social identity: The stigma of a chronic neurological disorder', *Lancet Neurology*, vol. 4, no. 3, March 2005, pp. 171–8.
16 Institute of Medicine, *Epilepsy across the spectrum: Promoting health and understanding*, Washington, DC: The National Academies Press, 2012.
17 International League Against Epilepsy, 'The history and stigma of epilepsy'.



Salute to the Hippocratic school of Kos

The Greek physician Hippocrates is one of the seminal figures in the history of our understanding of epilepsy. He founded a school on the Aegean island of Kos that was in many ways a forerunner of modern medicine. His key contribution to advancing understanding in this area was the suggestion that epilepsy is a physical condition akin to other ailments, and that it originates in the brain.

Hippocrates lived from around 460 BCE to around 377 BCE, but we know little about him with certainty. He was probably born on Kos, and the school of medicine that he founded there revolutionised approaches to disease. The key advance developed by this school was a belief that careful observation using all the senses could reveal the causes of diseases that were rooted in hereditary or environmental exposures. This opened the way to the beginnings of a scientific approach and real advances in diagnosis and treatment in many areas. The school had some knowledge of movement of fluid in veins, and of the role of the lungs in introducing air into the body. From post-mortem dissections its members understood gross morbid anatomy.

Beyond that, however, major advances were more than a millennium away. Harvey's discovery of the circulation of the blood and the story of modern neuroscience were far in the future. Hippocrates' school developed the theory of the four humours, both to explain illness and to underpin a basic approach to treatment. In the end, this held back the evolution of the discipline of physiology. Nevertheless, the school's work was a massive conceptual advance at the time.

Hippocrates moved to Athens at some stage and it is important to place his work in that context. One of the greatest periods in the advancement of knowledge was already underway in that city, starting with the pre-Socratic philosophers and culminating in the work of three of the greatest minds of the age: Socrates, Plato and Aristotle. It is likely that Hippocrates overlapped with the greatest of ancient philosophers, Socrates, and may have been influenced by him in learning to base theory on observation and to always ask the key question: 'Why?'. Hippocrates may have been present during at least one of the great dialogues. We have very little knowledge of what Hippocrates himself actually wrote, but a great deal remains that can be attributed to his school; it is common practice to attribute these writings to his intellectual influence.

The school's most spectacular example of firmly placing a disorder in the physical realm is in the case of epilepsy. This is set out with clarity and precision in the monograph *On the sacred disease*. Epilepsy had been recognised and some of its features described in Arcadian times. The abrupt onset, and the striking and sometimes violent physical accompaniments affecting a previously healthy individual, had led to the view that this

John Faber the elder, after Peter Paul Rubens, ***Hippocrates Hiraclidae F. Cous***, published by John Bowles and Thomas Bowles I, 1707–21, mezzotint on paper, 35.0 × 24.9 cm. British Museum.

illness came from the gods, hence the term 'the sacred illness'. This view was probably more widely held than beliefs in epilepsy being caused by demonic possession, which were largely a later, medieval, phenomenon.

Hippocrates and his school recognised that epilepsy was very much a physical illness. They postulated a genetic and an environmental component, using the terminology of their time. They made the point clearly that epilepsy should be regarded as having a basis in understandable things, with no need to invoke the mystical. In the process, they suggested much that has stood the test of time. The surmising of a likely genetic component was based (perhaps misleadingly in part) on physical similarities between affected parents and children. They hinted at different phenotypes (clinical manifestations) and clearly described many of the clinical characteristics of seizures on the basis of meticulous observation.

To realise the significance of these insights, we must place ourselves in Hippocrates' age. This was a society with a deep-seated belief in the mystical, magic and gods. The rudiments of science were just beginning. Everyday events were routinely ascribed to divine intervention by a bewildering array of gods and demigods. For this reason, Hippocrates' seminal observations about epilepsy were important not only because they cast epilepsy in a new and more rational light, but also because they were a step forward in the development of human thought generally. It is doubtful that these advances could have occurred without the new thinking that was emerging from Athens and sweeping across the Greek world.

What the school of Kos got wrong about epilepsy is much less important than what it got right. These early scholars defined the deeper causation in the only way they could, that is, in terms of a flawed conceptual paradigm that would be redressed only by advances in science centuries later. Imbalance of humours, with a tendency to the choleric, and a build-up of phlegm blocking the circulation of fluids and air were postulated. Hippocrates did, however, recognise correctly that the physical basis was likely to be in the brain, following an earlier recognition by the Hellenes that the brain was the site of the mind.

How influential were these observations in the centuries that followed? They spread with Hellenic culture across the Mediterranean basin with the Roman expansion and are likely to have been available to the great Arab scholars. They were temporarily lost in the West after the fall of the Western empire. Undoubtedly they influenced a primitive therapeutic approach and a mindset that improved the lot of many people with epilepsy before modern times. It is legitimate for modern epileptologists to salute the school of Kos!

Professor Edward Byrne

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PEOPLE THINK THAT EPILEPSY IS DIVINE SIMPLY BECAUSE THEY DON'T HAVE ANY IDEA WHAT CAUSES EPILEPSY.

BUT I BELIEVE THAT SOMEDAY WE WILL UNDERSTAND WHAT CAUSES EPILEPSY, AND AT THAT MOMENT, WE WILL CEASE TO BELIEVE THAT IT'S DIVINE.

AND SO IT IS WITH EVERYTHING IN THE UNIVERSE.

HIPPOCRATES

What difference does a name make? Epilepsy in Asia

Introduction

People with epilepsy are burdened by a multitude of social, psychological and economic consequences of stigmatisation, which leads to poor quality of life.[1] Western culture offers a mostly naturalistic explanation for illness, and places a strong emphasis on basic human rights. This probably minimises the misconceptions and stigma surrounding epilepsy. By way of contrast, in many parts of Asia, non-scientific explanations are still prevalent. Epilepsy may be viewed as spiritual, contagious or a form of insanity. This probably contributes to poorer attitudes towards epilepsy.[2] Names of epilepsy in Asia often reflect the misperceptions held by the speakers of the local language, and may contribute to stigma. There are moves in some countries to change these stigmatising names to reflect the modern scientific understanding of epilepsy.

Names of diseases

Misconceptions about epilepsy may have been reflected in the names chosen for epilepsy in different languages. Diseases are often named based on concepts, main symptoms and attributed causations. For example, for diabetes mellitus, *diabetes* originated from the Greek for 'siphon', indicating emaciation caused by fluids flowing out of the body. *Mellitus* is from the Latin term for 'honey', referring to the sweet urine of people who have diabetes.

In the Western world, although there is now a biological explanation for epilepsy, historically it was perceived as a spiritual or mental condition, and this was reflected in names such as *caducus* (the falling sickness), as well as *demoniacus* and *lunaticus*.[3] The term 'epilepsy', first used by the Persian physician Avicenna, was coined from the Latin word meaning 'being possessed by an outside force'.[4] With the development of modern medicine, the word epilepsy was given new meanings based on scientific concepts, and the illness was no longer perceived as spiritual or mental.

Epilepsy in Chinese: the original meaning

In the various regions of north-east Asia, the naming of epilepsy has been influenced by ancient Chinese medical concepts, as shown in the table on p.14.[5] Epilepsy was described in *The medical classic of the Yellow Emperor*, published more than 2000 years, ago as *dianji* (癲疾) and *xian* (癎).[6] The word *dian* was subsequently associated with madness. The terminology was further described in a Ming Dynasty dictionary (正字通) as a disorder of the internal organs, secondary to wind and associated with different kinds of animals, such as the goat and the pig.

Zhou Rongqi (Ming period, 1368–1644), **Crow**, from *Bencao tupu* (Illustrated herbal), 1630, paint on silk, 23.3 × 20.4 cm. Library of Zhongguo zhongyi yanjiu yuan (China Academy for Traditional Chinese Medicine). Wellcome Images.

Because of the influence of traditional Chinese medicine (TCM) in Korea and Japan, and the sharing of ideographic scripts, names for epilepsy in Korean and Japanese are similar to the name for epilepsy in Chinese. That is, *gan* in Korean is the same as *xian* (癎) in Chinese, and *tenkan* in Japanese kanji is the same as *dian xian* (癲癎) in Chinese, though the pronunciation and the phonetic-based scripts (tankan, ganjil) are different. Thus the association with insanity is also present in Korean and Japanese. Furthermore, the terminology for epilepsy in these regions continues to be used to describe insanity. In China, the word *dian* remains a term to describe a crazy or mad person. In Japan, *ten*, from the word *tenkan*, is also frequently used in psychiatry to indicate madness, for example, an insane person is called *fu-ten-nin*.[7] This may partly contribute to the persistence of misconceptions about epilepsy in Asian countries, with 24 to 57 per cent of the population regarding epilepsy as a mental illness.[8] The name for epilepsy in Mongolian, *unalt-tatalt*, also has a connotation of madness.[9]

Names for epilepsy in other Asian languages

In some south-east Asian languages, such as Burmese, Khmer (Cambodia), Lao, Malay and Thai, the names for epilepsy are associated with madness and animals, as shown in the table on p.14. This is probably due to the influence of TCM concepts. In Malay, the term *gila babi* means pig madness. As the majority of Malays are Muslim, the term also results in religious stigmatisation, because the pig is perceived as religiously not 'halal', or unclean. In the Philippines, most of the names used in various dialects refer to convulsion. However, in the Cebuano dialect spoken in some parts of Visayas (central Philippines) and Mindanao (southern Philippines), *baboyon* refers to a pig that has gone mad.[10] In East Timor, where the most widely spoken language is Tetum, the usual term is *bibi maten*, which means dead goat.[11] This term developed either because a person can sound like a goat when having a seizure or because eating goat meat was thought to cause epilepsy, as goats convulse when they are slaughtered. *Manu maten*, which means dead chicken, refers to people who have cerebral palsy and epilepsy. The official term for epilepsy is *epilepsi* but *bibi maten* continues to be used.[12] It is interesting that the Tetum terms retain some TCM references to animals, but 'madness' as a term has been dropped, to be replaced by 'dead', which is probably also stigmatising.

How do names for epilepsy contribute to stigma?

We believe these names convey concepts that are not only erroneous, but also contribute to stigma. Stigma (from the Latin term *stigmata*, meaning marks or blemishes) means being regarded negatively because one is different from the norm. All societies develop norms, and people who are perceived as differing from them are stigmatised, or rejected as abnormal, and therefore marginalised. Fear of the unknown arouses negative feelings, which we try to reduce by rejecting the cause of the fear: in this case, the person who has epilepsy.[13] Names for epilepsy that convey images of insanity and religious uncleanliness evoke responses from the public that people with epilepsy are abnormal, as well as feelings of insecurity caused by the unknown, thus contributing to the stigmatisation of epilepsy patients and their isolation from society.

Should names for epilepsy be changed? Responses from Malaysia, Hong Kong, Korea, Japan and China

Fernandes and others studied a group of high school students who received one of two versions of the 'Stigma scale of epilepsy'.[14] The versions differed only in the term used for epilepsy: 'People with epilepsy' (PWE) group, and 'Epileptic' group. Members of the 'Epileptic' group have more difficulty finding employment and at school. The authors concluded that the words we use can influence our perceptions and have consequences in stigma. Thus, names for epilepsy that contribute to stigma should be changed to a non-stigmatising name.

As mentioned above, in Malay the term *gila babi* means pig madness, which has negative social and religious connotations. The name for epilepsy has therefore been changed to *penyakit sawan* (seizure disorder) or *epilepsi*. *Penyakit sawan* has been well accepted and is currently widely used in Malaysia.

In 2010 the Hospital Authority of Hong Kong replaced the old name for epilepsy, *dian xian* (癲癎), with *nao xian zheng* (腦癎症, epilepsy), which is more consistent with modern scientific concepts, has no connections to animals, and less obvious reference to psychiatric disorders.[15] In Korea the name for epilepsy, *gan-zil* (간질, 癎疾, meaning mad sickness), was replaced by *noi-jeon-jeung* (뇌전증, 腦電症, cerebro-electric disorder) in 2011.[16] The new term was subsequently approved by the Korean National Parliament and will be promoted through campaigns and public education.

In Japan a 1980 survey of the attitudes of 349 Japanese epileptologists found that 35.2 per cent thought that it was necessary to change the term *tenkan*, and 32.7 per cent agreed to use the term epilepsy instead. However, many of the respondents thought that a change in terminology without a corresponding change in public attitudes would be useless, and 50.1 per cent did not think changing the term *tenkan* was necessary.[17] *Tenkan* continues to be used in Japan today.

In mainland China, the China Association Against Epilepsy (CAAE) has made efforts in the past few years to change the terminology *dian xian* (癲痫). A nationwide public campaign was carried out in 2009 to choose a new name. A few possible terms were selected, but because of differences in opinion between members of the CAAE board, and the complicated procedures for approving changes to medical terminology in China, a definitive decision is yet to be reached.

Names for 'seizure' in various Asian languages[18]

Language (country)	**Name**	**Meaning**	**Stigmatising**	**Change**
Chinese (China)	癲痫（癲癇） (*dian xian*) or 羊癲 风 (*yang dian feng*)	madness or goat madness	++	
Chinese (Taiwan)	癲癇 (*dian xian*)	madness	++	
Chinese (Hong Kong)	癲癇 (*dian xian*)	madness	++	脑症 (*nao xian zhen*)
Japanese	*tenkan* (てんかん; 癲癇)	madness	++	
Korean	*gan-zil* (간질;癎疾)	mad sickness	++	*noi-jeon-jeung* (뇌전증; 腦電症; cerebro-electric disorder)
Mongolian	*unalt-tatalt*	madness; convulsion	++	
Malay (Malaysia/Indonesia)	*gila babi*	mad pig; mad pig disease	++++	*penyakit sawan* or *epilepsi*
Lao	*sak pa moo*	sickness mad pig; mad pig disease	++	
Thai	*sok lom bai*	sickness mad pig; mad pig disease	++	
Burmese (Myanmar)	*wet you pyan yawga*	mad pig disease	++	
Khmer (Cambodia)	*chkourt chrouk*	mad pig disease	+++	
Tagalog (Philippines)	*kumbulsyon*	convulsion	0	
Cebuano (Philippines)	*baboyon*	pig that has gone mad	++	
Tetum (Timor Leste)	*bibi maten*	dead goat	++	
Tamil	*valippu*	tremor; jerk	0	

How should names be changed?

How should a new name for epilepsy be chosen? Guidelines for new names used by the Korean epilepsy community list the following general principles: the new terminology must be neutral, scientific, easily differentiated from terms with some apparent similarity in meaning (such as convulsion, fits or spasm), easy to use as both a noun and an adjective, and likely to be acceptable to the international epilepsy community.

Professor Chong-Tin Tan

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16 BI Lee *et al.*, 'New epilepsy name in Korea', 2011, www.caoa-epilepsy.org/img/pdf/Epilepsy%20Renaming%20in%20Korea.pdf, accessed 24 May 2012.
17 Hosokawa *et al.*, 'Awareness and attitudes of Japanese epileptologists'.
18 Reproduced with permission from Lim *et al.*, 'Name of epilepsy, does it matter?'.

The history of epilepsy in Victoria

Early days: the Port Phillip Settlement

The Port Phillip Settlement started life in 1835 as an agricultural and grazing venture, serviced by a small village. Originally a southern outpost of New South Wales, it gained status as a separate colony in 1851. Population increase was initially gradual and unremarkable, reaching 11 000 by 1841, including 33 doctors, who pursued a mixed lifestyle of medicine and grazing. But less than two months after Victoria's separation from New South Wales, the discovery of large deposits of gold triggered a gold rush—with a population influx that threatened to overwhelm the new colony's basic facilities. Within a year the population had risen to 170 000; in two years it had ballooned to 280 000—some 40 000 more than New South Wales. The public health problems that this produced would dog Victoria for decades to come.

In the mid-19th century, epilepsy sufferers in the public health systems of most countries were housed in asylums for the insane. The fledgling colony of Victoria, however, had no asylums, so local gaols were used instead.[1] These institutions contained not only felons but also disturbed citizens, whose unsuitable behavior was due to a variety of causes such as alcohol, head injury and infection, as well as various mental disorders. By such means the authorities intended to keep the streets secure and free of danger. Since epilepsy was believed to be an indicator of later insanity, it seemed reasonable to house 'the epileptic' in the prison system as well.

Judging by the statistics in official reports to the Colonial Office in London, epileptic seizures (labelled 'epilepsy') were by no means uncommon. Even from early times, Victoria had a considerable number of prisons scattered across both rural and metropolitan areas, and in some the documented epilepsy rate was considerable. For example, in 1877, seven per cent of the population of the Melbourne Gaol and 13 per cent of the Geelong Gaol was in this category, and the occasional death from seizure was recorded.

By good fortune, Victoria in its earlier years had an extremely conscientious chief gaoler, Mr Wintle, and government colonial surgeon, Dr Cussen, who monitored the health status of the inmates. Problems were kept to a minimum—only one death from epilepsy between 1836 and 1841—despite the fact that prisons were notorious places at the time, especially for infectious epidemics.

In 1868 Victoria's chief medical officer, Dr McCreery, noted in his 'Return of diseases for Victoria' that in the Melbourne Gaol diseases of the nervous system occupied a prominent place 'in consequence of a number of lunatics in the gaol',[2] implying that these people had no place there. By this time the move to asylums had begun.

Cat. 101a **Post-mortem instruments set, in case**, c. 1880; wood, brass, metal; 27.4 × 16.4 × 4.6 cm; inscribed on brass plate *Yarra Bend Asylum*. MHM04523, gift of Alan Kilgour, 2005, Medical History Museum, University of Melbourne.

In 1871, a death from seizures in Melbourne Gaol attracted considerable comment; the jury at the inquest noted: 'the neglect of authorities in not keeping the prisoners liable to fits in an associated prison ward is highly reprehensible'.[3] Similar comment was made in the *Australian Medical Journal*, a timely warning that the practice of imprisoning such individuals must cease.[4]

Youthful offenders were housed in 'industrial schools'—the reformatory system of that era. These were another hazardous situation needing attention, being notorious pools of rapidly spreading infection. Epidemics sweeping through the populations there were prone to cause fever-induced convulsions, sometimes with lethal consequences.[5] These were no place for young sufferers of epilepsy, as discussed below in regard to Dr Jamieson's presentation to the Medical Society of Victoria in 1878.

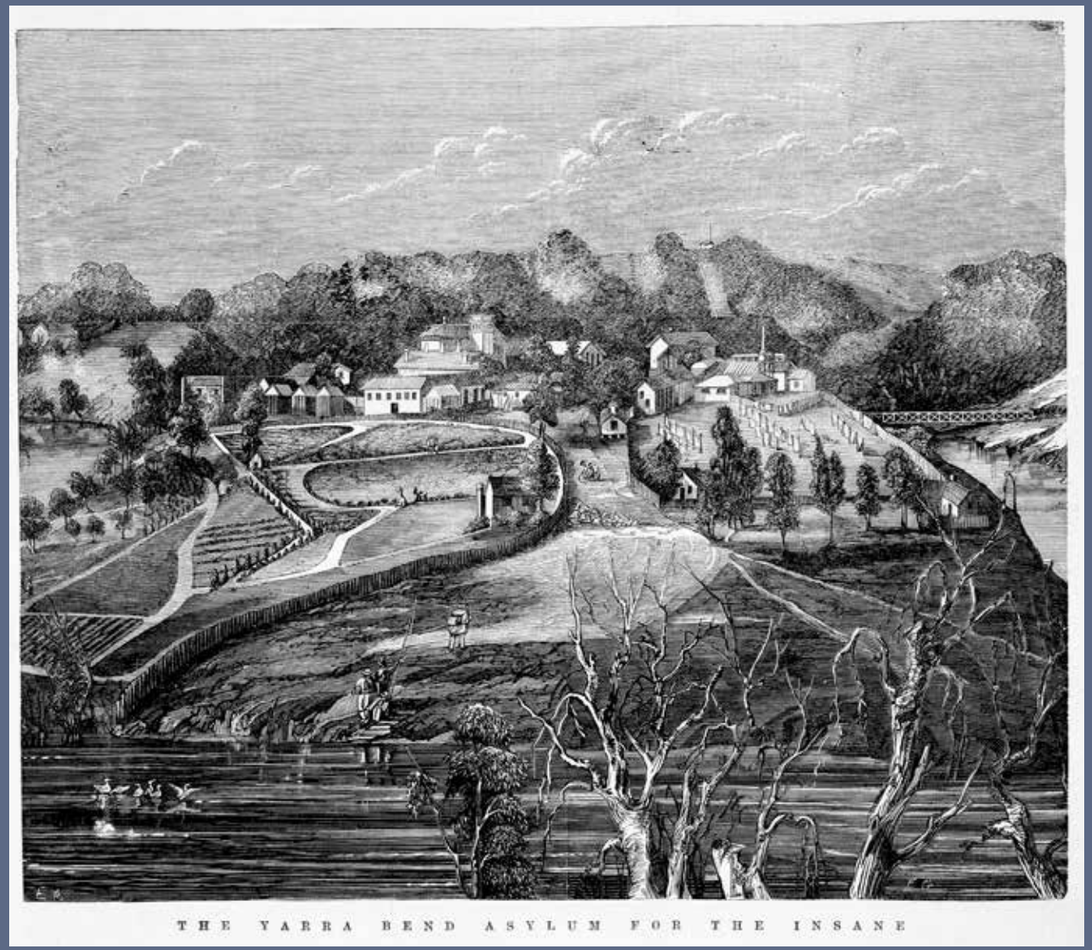
Epilepsy in Victoria's colonial asylum system

The move from prisons to asylums

It had become abundantly clear that it was essential to move people with epilepsy or mental disorders out of the prison system,[6] and the 1848 completion of the Yarra Bend Lunatic Asylum, just outside Melbourne, marked the beginning of this shift.[7] At its height Yarra Bend accommodated over 1100 inmates. It was finally closed in 1925; part of it became a venereal diseases clinic until 1956. The only relic of it to be seen now is an old bluestone pillar bearing a historic plaque, near the site of the recently demolished Fairlea Women's Prison. In its time, Yarra Bend Asylum was said to be the fourth-largest asylum in the world, although there were some in the United States that held over 6000 inmates.

The asylum system in 19th-century Victoria was no place for the management of epilepsy.[8] Not only had asylums traditionally fostered hostile attitudes towards inmates with epilepsy, but the strict attention needed to manage epilepsy properly was not to be found there. Nevertheless, until 1898, asylums were the repository for 'epileptics'. The tale of two Elizabeths of Yarra Bend Asylum vividly illustrates the two extremes of epilepsy in gold rush Victoria.

The first was Elizabeth Marks, aged 12 years, admitted to Yarra Bend Asylum in 1852 as 'an epileptic'. She had come from Canvas Town on Yarra Southside, a vast array of tents in which new immigrants lived until they found better accommodation. In fact the only problem was that Elizabeth's epilepsy could not be managed in such living conditions. The admitting doctor wrote in his notes that although her 'form of mental disease' was 'slight mental instability', when further comment was made in her record there seemed to be no problem other than occasional seizures. His opinion:



Cat. 123 Frederick Grosse (engraver), ***The Yarra Bend Asylum for the Insane***, wood engraving in *The Illustrated Australian News*, 23 May 1868. IAN23/05/68/12, courtesy State Library of Victoria.

> This girl is fitter for being an inmate of a benevolent asylum or a hospital than this asylum. The mental imbecility being almost imperceptible except immediately after a fit. She's improved very much in health and the fits are much less frequent. She answers questions readily and accurately.[9]

Nowadays we would need a good description of her seizure pattern to make the diagnosis accurately, but it would seem that this lass had a primary generalised epilepsy and that the medication epilim would have suited her nicely. However, that was 120 years away. It is hard for us to contemplate why this young girl even needed to go into an asylum, but that was the response to epilepsy in those days.

The second Elizabeth was a 'typical case' to the mind of the community of her time. Eliza Richardson, aged 31, was admission number 76 to Yarra Bend Asylum, on 3 January 1853. She was of no fixed abode, and more importantly she had a history of being in the New Norfolk Asylum in Van Diemen's Land some time before coming to Victoria. Originally she came from Richmond in Yorkshire.

Eliza's diagnosis was 'epilepsy and profligacy'. She was shockingly dirty and ragged, and was described on her admission as a 'dissolute, idle, disorderly person. Admitted from Melbourne Gaol. A mother of four children, the youngest nine months of age'.[10] In short, she was the complete picture of 'epilepsy' as the community conceived it in the mid-19th century.

While today we can recognise that these two cases were vastly different and had different prognoses, in the 1850s all epilepsy was one in the minds of professional and lay communities alike.

Remark must be made on the very real problem of alienists' antagonistic ideas about 'the epileptic'. Not only was epilepsy seen as the gateway to subsequent insanity, but 'the epileptic' was perceived as a real problem in his or her disturbance of the smooth running of the asylum, due to the characteristics of 'the epileptic personality'. This was a widely held view at the time. As late as 1902 Dr Barker, trained in England, opined:

> epileptics are the only patients in asylums who possess any inclination or evince a disposition to combine; they are observed in constant companionship, sometimes in conspiracy, and ever-ready to render assistance to one another during their fits.[11]

Unfortunately, this concept of 'the epileptic personality', with its implications of antisocial tendencies, persisted until the mid-20th century. Evidence of this can be seen in the presentation of Dr Manning, inspector-general of the insane in New South Wales, in his address to the British Medical Association in 1900,[12] and in a lecture given at the University of Melbourne by visiting American criminologist and psychologist Dr Muhl in 1939.[13]

The Zox Commission, 1884–86

The Victorian asylum system was renowned for producing magnificent asylum buildings, built with the wealth of the gold rush, but asylum management was another matter. The system itself was deeply flawed and produced a huge number of official inquiries into its running, the first barely four years after the opening of the Yarra Bend Asylum. The most significant and thorough of these was the royal commission presided over by Ephraim Zox, the 14th enquiry of the Victorian lunacy system to that date, which covered virtually its entire field.[14] For the first time the role in the asylum system of the person with epilepsy was canvassed. Despite this and significant improvements in the asylum system in general, 'expert' evidence by Dr Springthorpe and colleagues seemed uncertain at best, and there was little productive outcome for epilepsy management.

The move from asylums to special facilities

The perceptive Dr Alexander Robertson, acting inspector of lunatic asylums in Victoria, had in 1873 produced an insightful report which included a recommendation that inmates suffering from epilepsy, if reasonably well stabilised, should be boarded out, and not retained in the asylum system. But not until the 1898 report of Dr McCreery was it announced that 'steps have now been taken to provide separate accommodation by the erection of new buildings at Ballarat' and that 'Metropolitan Asylums will be relieved of a large number of patients'.[15] Eventually a cohort of 100 female patients was transferred from mental asylums throughout Victoria to quarters renovated for their use at the Ballarat Mental Hospital—still in the mental asylum system. Despite this move, and the supposedly special nursing facilities, follow-up of these patients over the years 1900–17 showed that one-third of them died in status epilepticus.[16]

Landmarks in epilepsy research in Victoria

Dr Jamieson's analysis, 1878

Dr James Jamieson was a most important identity in the early medical history of Victoria, the University of Melbourne, and the public health system—inefficient though this was. His deep interest in public health statistics supplied him with evidence of the importance of his subject for his presentation on childhood convulsions, their outcome and causes. This was a serious matter, for in Victoria in the year 1876 some 365 children aged five years or younger had died in convulsions—a significant public health matter by any reckoning. Jamieson's statistics were taken from death certificates.

In 1878 Jamieson made a presentation to the Medical Society of Victoria on the chief causes of convulsions in children.[17] Jamieson's concept of the pathophysiology of epilepsy adhered to the pre-Jacksonian major role for the brain stem, but he felt that the major factor underlying these convulsions was some 'eccentric' cause, situated outside the brain: probably 'gastric irritation' due to improper food intake. He ran through the usual list of

traditionally held causes: teething, intestinal worms and the like, contrary to the general opinion voiced in the post-presentation discussion.

Strangely, Jamieson dismissed fever as a cause of this large number of convulsions with lethal outcome. This conclusion was highly significant because of the huge epidemic of scarlet fever that had ravaged the community in 1876. Jamieson felt that the increase in number of deaths was simply a factor of the rapid increase in population of the colony. But a surgeon, Mr Hewlett, immediately took issue with this, correctly pointing out that any evidence taken from death certificates runs the risk of the doctor signing the certificate simply stating the basic aetiological diagnosis and not the symptoms produced, and in fact Hewlett, again correctly, opined that in cases of childhood scarlatina, convulsions were very common and severe.

As well as his defence of the traditional conditions thought to provoke paediatric convulsions, Jamieson's therapeutic regimen for treatment of such convulsions was also of interest: clearance of bowel-irritating contents and use of chloroform anaesthetic to dampen seizure activity. He dismissed all other traditional treatments as of little or no use.

The importance of Jamieson's presentation lies largely in his use of the colony's public health statistics to highlight a significant health problem resulting from the woeful inadequacy of public facilities. The failure to install adequate infrastructure, due to the rapidity of the colony's post-gold rush development, was a major influence on Victorian medical practice well into the next century. Jamieson's paper is also important because his discussion of the treatment of convulsions gives some idea of traditional therapies used at that time, and his method of presentation—statistical tabulation—was novel to the Medical Society of Victoria.

The Springthorpe epilepsy trilogy

The return home of Dr William Springthorpe MD MRCP after completing professional training in England meant that Victoria had acquired its first neurologically trained specialist physician, for he had spent several years working at the Hospital for the Epileptic and Paralysed in Queen Square, London—the cradle of world neurology.[18] Springthorpe quickly moved into the area of clinical neurosciences, and among his principal areas of interest was epilepsy. He began collecting data on all his patients presenting with epilepsy and in 1886 presented the results to the Medical Society of Victoria.[19] Before presenting his data in the form of detailed tabulation, Springthorpe briefly described current concepts of the pathophysiology, including Hughlings Jackson's new theory of the role of the cerebral cortex.

Springthorpe's data show a wide spread of features that he thought to be of significance concerning aetiology, age of onset and seizure characteristics found in his patients, plus any other factors thought significant. The table contains a plethora of

Alice Mills (photographer), ***Dr James Jamieson***, 1886, sepia photograph and ink, 38.0 × 30.0 cm (image). MHM03901, loan from Australian Medical Association Victoria, 1994; donated 2011, Medical History Museum, University of Melbourne.

detail that, by 21st-century standards, seems irrelevant. The inclusion of such features as sunstroke, alcohol, fright, worms and fevers shows the retention of traditional views alongside the adoption of newer Jacksonian concepts.

The clinical seizure features listed by Springthorpe are unhelpful in determining the types of epilepsy syndromes encountered, although focal seizures seem to predominate. Seizure frequency would indicate that control of the epilepsy was uncertain, despite the availability of bromide anticonvulsant. But as Springthorpe's audience was quick to point out, three years as a follow-up period gave no really reliable information as to the efficacy of treatment. His reply was that he did not intend to discuss the treatment of epilepsy, but since he had been asked, he set out his therapeutic approach:

- removal of peripheral irritants
- potassium bromide, 30 grains at night … tailoring the dose to suit
- failure of bromide: zinc oxide, belladonna, cannabis indica, digitalis
- focal seizures: ligature around limb as counter-irritant
- for 'petit mal': caffeine and nitroglycerine
- finally, according to Dr Wilks of London, 'A seton set in the nape of the neck should never be forgotten'.

This is a mix of very old and reasonably new treatments; but the idea of using a seton—a skein of silk sutures threaded through the loose skin at the back of the neck to create chronic weeping suppuration—is something out of the Middle Ages!

Cat. 107 English, **Pharmacy bottle for chloral hydrate**, c. 1900, moulded painted glass and printed paper, 17.5 × 6.5 cm diameter, labelled *Chloral Hydrat*. MHM01164, Medical History Museum, University of Melbourne.

Cat. 109 English, **Pharmacy bottle for zinc and valerian**, c. 1900, painted glass, 14.0 × 5.0 cm diameter, labelled *Zinci Valerian*. MHM01189.15, Medical History Museum, University of Melbourne. Zinc and valerian were used either singly or in combination to treat epilepsy.

Cat. 112 English, **Pharmacy bottle for chloroform**, c. 1890, painted glass, 25.3 × 10.3 cm diameter, labelled *Lin Chlorof*. MHM01582.7, Medical History Museum, University of Melbourne. Chloroform was used in status epilepticus.

Cat. 110 English, **Topical medication applicator and container**, c. 1900; glass, paper (printed), wood, metal and bristle; 6.5 × 3.6 cm diameter (jar and stopper); 8.0 × 4.5 cm diameter (container), labelled *Liquor vesicatorius*. MHM01213, Medical History Museum, University of Melbourne. Formerly used in blistering a limb to treat focal epilepsy seizures.

Cat. 108 English, **Pharmacy bottle for opium**, c. 1900, moulded glass and printed paper, 18.3 × 55.8 cm diameter, labelled *LIQ.OPII SED*. MHM01166, Medical History Museum, University of Melbourne. Formerly used in some cases of epilepsy.

Cat. 106 English, **Pharmacy bottle for digitalis**, c. 1900, painted glass, 20.0 × 7.8 cm diameter, labelled *TR:DIGITAL:*. MHM01155, Medical History Museum, University of Melbourne.

Overall, Springthorpe's paper paints a good picture of the clinical concepts in this transition period from old to modern epileptology. But his most important message was the importance of considering this clinical and social problem as a subject for detailed discussion by all concerned with the management of people suffering from epilepsy.

In 1887 Springthorpe delivered the second of his trilogy of papers on epilepsy, on treatment by the removal of peripheric irritants.[20] He reiterated his concept of the pathophysiology of epilepsy: 'over-excitablity of the cells in the brain cortex; the trigger message to the brain from a peripheral irritant'. Bromides were used to dampen down the brain's excitability, but it was also necessary to eliminate the source of peripheral irritation if possible. Springthorpe divided 'irritants' into four categories:

- ovarian and uterine irritants
- intestinal worms in children
- 'gastric irritation': dyspepsia, hepatic derangement
- centric irritants: anxiety, overwork, worry.

These were age-old traditional concepts, revealing the transitional nature of epilptology in this phase of its evolution.

A year later Springthorpe presented the third paper in this trilogy: 'Notes on fifty cases of epilepsy'.[21] As this marked five years since the beginning of his observations, Springthorpe believed he had enough follow-up data to remedy the defect for which he had been criticised in his first paper. Now he could present data on 50 patients.

Of particular interest are Springthorpe's observations that well over half his patients had experienced early-onset seizures in youth, that inheritance played an important role in nearly half his patients, and that a considerable number of patients had what was then called 'petit mal' (absence seizures). He gave no details of seizure content by which we can determine the true nature of these latter seizures; the discovery of the temporal lobe seizure was still some years in the future.

Overall, Springthorpe's trilogy of presentations on the subject of epilepsy constituted an important canvassing of a prominent problem in neurological medicine, one that had huge medical and social management implications. It also gives us a complete idea of the neurological theory underpinning this important area, which at that time was undergoing a transition to a completely new conceptual framework.[22]

Current therapy for epilepsy: world view

In view of the fact that the documented evidence of the nature of anti-seizure treatment recommended by some of the early doctors in Victoria was so primitive and ineffectual—folk medicine in fact—one is driven to despair at the primitive state of medicine in the colony at that time. There one finds a strange mix of agents and procedures, for example:

Cat. 103a **Dr JW Springthorpe**, c. 1900, photograph, 12.0 × 9.8 cm. MHM00674, courtesy Dr Guy Springthorpe, Medical History Museum, University of Melbourne.

- bleeding of 5 fluid ounces from superficial temporal artery
- dose with: chloride of silver grains 5
- Jacob's Powder, grains 3
- aromatic powder, grains 2
- one quarter of the mixture every six hours

—Dr Knaggs, 1856

Other examples include:

- head shaved
- leeches to the temples
- blister to the nape of the neck
- bleeding
- if still fitting: chloroform, four to five times over next two days.

—Dr Tracy, 1858

To us today this seems medieval, indicating an abysmal deficiency of medical knowledge. But comparison with treatment methods found in international medical journals of the period show that Victoria was not behind other parts of the world in this field. The bulk of doctors then practising in Victoria were immigrants, while many young men born in Victoria and wanting to practise medicine chose to graduate from overseas medical schools, notably Edinburgh where they would have been taught at international standards. It is therefore no surprise to find the treatments for epilepsy in Victoria were very similar to those current overseas. Dr William Springthorpe had benefited from several years' medical experience at the home of world neurology—the Hospital for the Epileptic and the Paralysed, Queen Square, London—and would have been well acquainted with all the latest treatments. Primitive though it was, the treatment of epilepsy in Victoria in the 19th century was in line with that used in most countries.

The Talbot Colony for Epileptics

The Talbot Colony, which opened in 1907, marked a very significant step in the liberation of people with epilepsy in Victoria. As indicated previously, realisation had dawned that people with epilepsy did not belong in asylums, or even in mental hospitals. Elsewhere in the world this conclusion had already been reached, indeed the foundation of Germany's Bethel by Pastor Bodelschwinge in 1860 and of London's Hospital for the Epileptic and Paralysed the same year[23] suggest that there was already significant medical and sociological opinion that sufferers of epilepsy were best managed anywhere but in mental asylums.

Cat. 66 **Talbot Colony for Epileptics, Clayton**, 1907, photograph, 9.0 × 22.0 cm. Epilepsy Foundation Collection.

Cat. 68 **Talbot Colony for Epileptics, dormitory**, 1907, photograph, 17.0 × 21.0 cm. Epilepsy Foundation Collection.

Finally, the important report by London's Charity Organisation Society, *The epileptic and crippled child and adult*, published in 1893,[24] gave a detailed account of what needed to be done for those in society with epilepsy.

Despite the lack of any substantial outcome from the Zox Commission, the turn of the century saw more and more medical experts openly voicing concern about the pressing need for proper management of patients with epilepsy. Dr Norton Manning at the British Medical Association Conference of 1900, Dr Henry Barker in his textbook of 1902,[25] and the redoubtable Dr John Fishbourne in his address intended for the Australasian Medical Congress (published posthumously in 1911)[26] all mentioned the importance of the colony-farm format for this purpose. Institutions such as the Craig Colony in New York showed how successfully these could be run. But the question remained: where was the driving force to establish such a colony in Victoria?

The National Council of Women of Victoria

The National Council of Women of Victoria (NCWV), the local body of the International Council of Women, was founded in 1902, with its first congress held in 1903.[27] Among a wide range of presentations, a paper titled 'Epileptic colonies' presented by Dr Mary Page Stone, a graduate of the University of Melbourne Medical School, made such an impression on the audience that it was decided to form a committee to examine the possibility of founding a colony-farm for people with epilepsy, as a commemoration of this first historic congress. Just what inspired Stone to become interested in this particular medical problem is not recorded, but much is owed to her for inspiring the NCWV, 'the champions of the impossible', to take up this cause.

Dr Fishbourne and Dr Springthorpe were recruited to lend professional assistance and to explain to the NCWV what was needed and what were the aims of setting up such colony-farms.[28] From the record of their talks we learn that the principal aims were segregation and rural occupation for patients with epilepsy, rather than medical management or research. These latter were features of some such institutions overseas, but never in the Talbot Colony.

Fishbourne had a long-standing reputation for his efforts in establishing and supporting education of people with disabilities, and was revered in his home district of Essendon. He was now transferring his concepts to people with epilepsy, but these did not encompass medical treatment or research. Long-term secluded care of these patients was his aim.

The campaign fought by 'the champions of the impossible' was long and frustrating.[29] Attracting financial support proved difficult, and it was not until the NCWV recruited Lady Margaret Talbot, wife of the newly arrived governor of Victoria, as its

Cat. 69 **Talbot Colony for Epileptics, farm workers**, 1954, photograph, 17.0 × 21.0 cm. Epilepsy Foundation Collection.

president that it gained enough influence to launch a public appeal and to approach the premier, Sir Thomas Bent, with a direct request for public funding. Lady Talbot was a most effective campaigner, and 19 July 1906 saw the passage of legislation establishing the Talbot Colony for Epileptics. A mixture of public and private funding had given the project a strong financial foundation, and a Mr James Mason donated 'Masonsmeadows', his 165-acre property in Clayton, as the site for the colony-farm.

Much work remained before the Talbot Colony for Epileptics was established, including a great deal of building and the installation of basic infrastructure. The farm had its inaugural ceremony on 13 March 1907, with the governor of Victoria, Sir Reginald Talbot, presiding and Lady Margaret Talbot there to see the triumphal outcome of her great efforts.

Dr Fishbourne died in 1911, but by that time the numbers of residents in the Talbot Colony were slowly building up and the establishment was getting underway.

The Talbot Colony existed until 1961. It was run mainly as a farming establishment with a workforce of people who had epilepsy, not as a centre of medical activity or research. There were doctors in attendance when needed, but medical visitation was restricted to a few occasions per week. We are fortunate to have a first-hand nursing account of the day-to-day running of the establishment in the 1930s: Sister Alma Johnson was a nurse on duty; for the most part, the doctor on duty was only called if an accident occurred that demanded medical attention. The colony was primarily run as a chronic-care establishment, with a school attached, as about 20 children lived there at that time.

Overall, the Talbot Colony was smoothly run, offering a secluded, peaceful lifestyle set among rural nature, a pleasant existence for all who lived and worked there.

Closure of the Talbot Colony

The Victorian government's decision to establish a second university in the state led it to resume, in November 1958, the land occupied by the Talbot Colony, following passage earlier that year of legislation to establish Monash University. The government decided to shift the colony to North Kew, and the chairman of the Hospital and Charities Commission, Dr John Lindell, who was about to go overseas, assured the Talbot Colony's committee that he would actively consider options for the best format for the institution at this new site.

Unfortunately, Victorian authorities seem to have lacked all knowledge of how similar colonies overseas, such as Chalfont in England and Bethel in Germany, were developing into medical research and educational centres in epileptology. Indeed, it seems that they never intended for the Talbot Colony for Epileptics to continue. Dr Lindell's return saw the announcement that the site in North Kew was to become a rehabilitation hospital.

This was a savage blow for all concerned with the management of young people with refractory epilepsy. Only through the personal efforts of Greg Hirsch, president of

Cat. 120 **Construction of Monash University** (buildings of Talbot Colony for Epileptics in the background), 1960, photograph, 20.5 × 30.0 cm. Oakleigh and District Historical Society.

the Victorian Bureau for Epilepsy, were the authorities persuaded to accept patients with epilepsy into the new hospital. But the Talbot Colony in its former format was finished. Although there was a late decision to include some young patients with epilepsy in the early years of the new Talbot,[30] the writing was on the wall for its long-term investment in managing epilepsy. The Talbot today has nothing to do with people with epilepsy.

Professor Peter F Bladin

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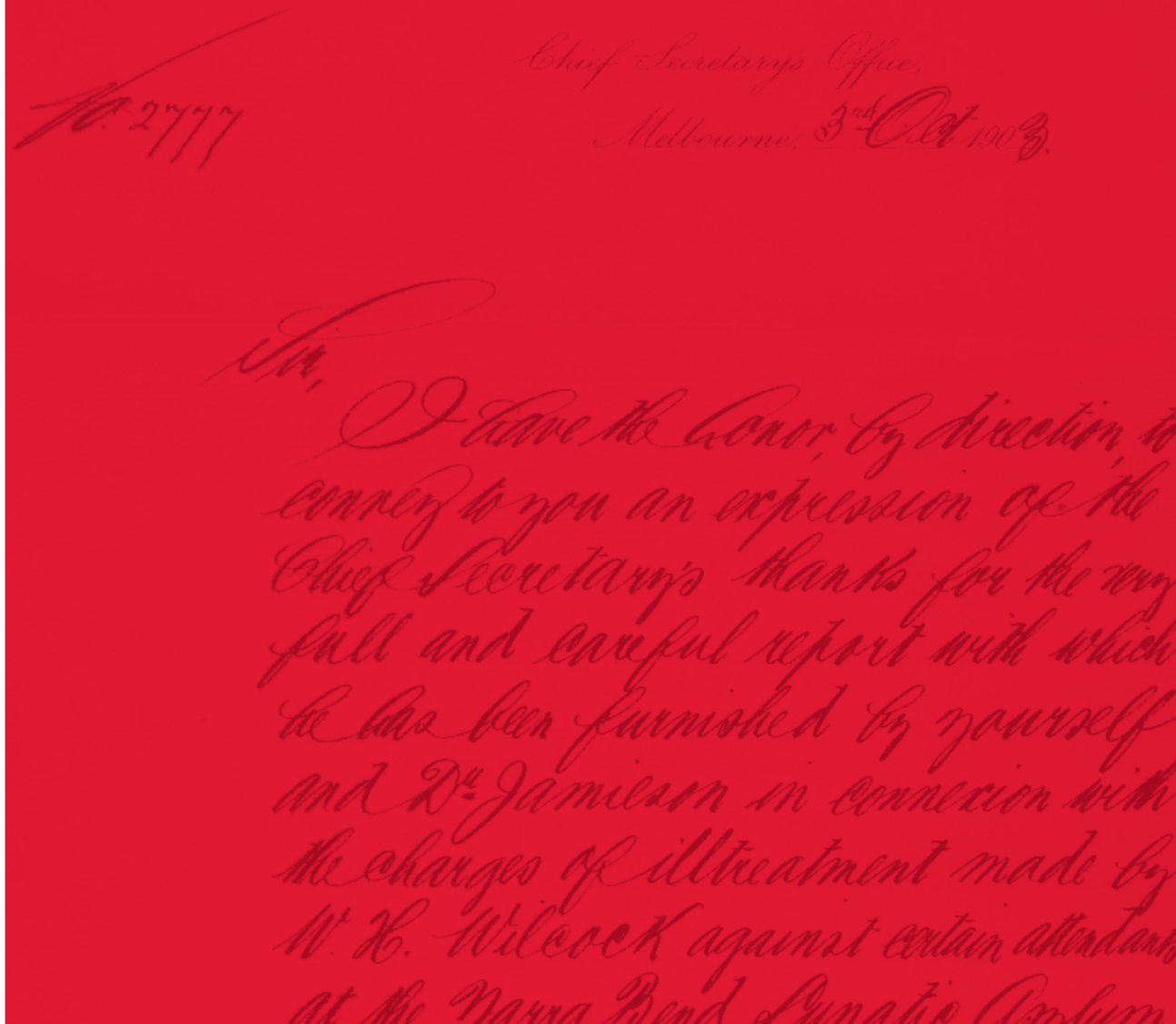
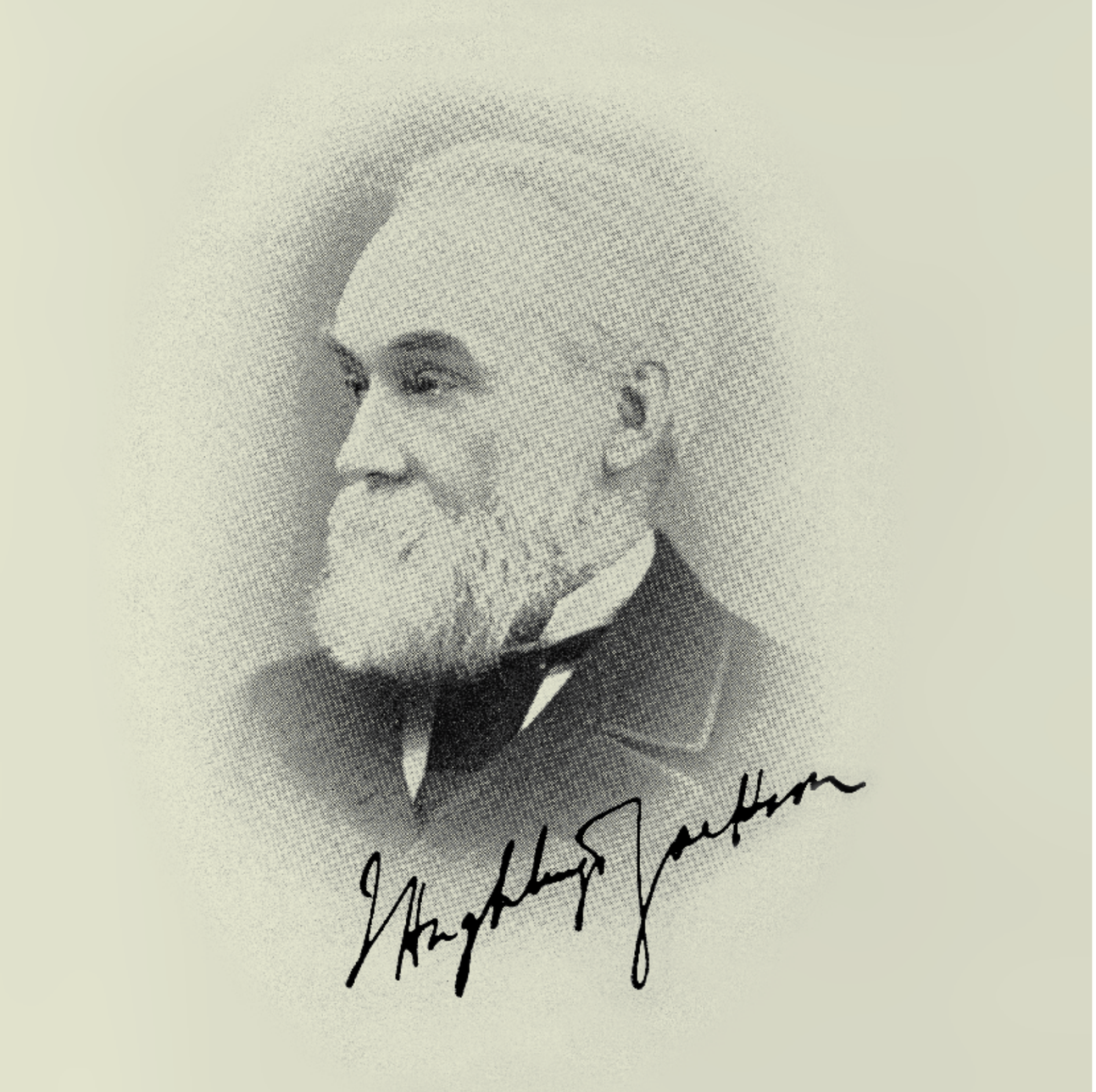
Cat. 99b Chief Secretary's Office, **Letter to Dr Joske** (detail, colour altered), to thank him for the report written with Dr Jamieson, which commented on charges of ill-treatment made by WH Wilcock against certain attendants at the Yarra Bend Lunatic Asylum, 3 October 1903, ink on paper (handwritten and printed), 33.0 × 21.0 cm. Gift of Dr William Joske, MHM03711.2, Medical History Museum, University of Melbourne.

TREATMENT AND DISCOVERIES





Overview: Epilepsy: Stigma, diagnosis and treatment

History

Epilepsy historically has been one of the most commonly recognised neurological disorders. Hippocrates, the father of medicine, in the fifth century BCE observed that a disorder of the brain was important for the occurrence of epilepsy and that the condition sometimes appeared to have a hereditary association. Modern care began with the observations and writings of John Hughlings Jackson (1835–1911), who became known as 'the father of epilepsy', at the National Hospital for Diseases of the Nervous System Including Paralysis and Epilepsy in Queen Square, London. Jackson postulated that seizures began in the cerebral cortex (the convoluted surface layer of grey matter of the brain, which coordinates sensory and motor information) and that the ictal behaviour (seizures) correlated with the region of functional anatomy. Further, he introduced the basic concept of focal epilepsy (partial seizures, or seizures involving only one side of the brain) with an area of cerebral cortex associated with a focal neurological deficit and focal seizure activity. Subsequently, in 1886 Dr Victor Horsley, a young surgical colleague of Hughlings Jackson, performed the first neurosurgery for treating epilepsy.

Today's perspective

Epilepsy is a chronic disorder characterised by recurrent and unprovoked seizures. It is one of the most common neurological disorders. One in 26 people will develop recurrent seizures during their lifetime. Community prevalence of epilepsy is twice that of multiple sclerosis, Parkinson's disease and autism spectrum disorders combined. Lifetime risk of developing epilepsy is estimated to be over 3 per cent.

Some 90 per cent of the incident cases in adults have focal seizure disorders. The most common seizure type in adults is the focal dyscognitive seizure of temporal lobe origin (associated with altered awareness or memory). Causes may include mesial temporal sclerosis (MTS: scarring in the inner portions of the temporal lobe), tumour, malformed blood vessels, malformations of cortical development (MCD), and head trauma. A diagnostic evaluation may confirm the classification of the seizure disorder and the seizure type(s), the underlying aetiology, and comorbid conditions and precipitating factors. We try to render the person seizure-free, while avoiding therapy-related adverse effects. The aim is for a person who has epilepsy to be a participating and productive member of society.

Unfortunately however, despite advances in diagnosis and management, approximately one-third of people with epilepsy will have a refractory seizure disorder (one that does not respond to treatment). Intractable epilepsy may be medically, physically and socially disabling and significantly impair a person's quality of life. People with epilepsy and refractory seizure

John Hughlings Jackson, frontispiece from JH Jackson (edited by J Taylor), *Selected writings of John Hughlings Jackson, vol. 1: On epilepsy and convulsions*, London: Hodder & Staughton, 1931. Wellcome Library.

disorders confront important challenges, including discrimination, under-employment and unemployment, restrictions on operating a motor vehicle, and difficulty living independently.

Diagnostic evaluation

Electroencephalography (EEG)

The scalp-recorded EEG is the most common diagnostic study used to evaluate patients with seizure disorders. The routine EEG study almost invariably records interictal EEG alterations (brain waves between seizures as opposed to an ictal EEG pattern) because of the intermittent nature of seizure activity. Interictal epileptiform activity may be sufficient to classify a person's seizure disorder and indicate their seizure type(s), and suggest appropriate medical therapy. The routine EEG recording, however, may be insensitive, that is, it may fail to identify epileptiform discharges, and yield nonspecific findings. Paroxysmal alterations (either nonspecific or potentially epileptiform discharges) may be identified in a patient with non-epileptic behavioural events. Patients with seizure disorders, even medically refractory epilepsy, may have repetitive 'normal' interictal EEG studies. The diagnostic usefulness of the routine outpatient EEG depends on multiple factors, including classification of the seizure disorder, localisation of the epileptic brain tissue, frequency and timing of seizure activity, duration of the EEG study, and the recording of activity during sleep.

Video-EEG monitoring may be used to confirm the diagnosis of a seizure disorder, classify seizure type(s), assess seizure frequency and precipitating factors, and for surgical localisation. This diagnostic tool is essential when evaluating patients with indeterminate clinical spells that may represent seizures. Video-EEG monitoring may be performed as an inpatient or outpatient procedure, and may be essential in the care and management of some patients' seizure disorders. Additional physiologic parameters that can be evaluated during video-EEG monitoring include the electrocardiogram, blood pressure, heart rate, and pulse oximetry (the level of oxygen in a person's blood). In adult patients, video-EEG monitoring is used mostly for diagnostic classification, that is, epilepsy as opposed to non-epileptic spells, and to evaluate surgical candidacy in patients with medically refractory seizure disorders. Surgery is usually restricted to patients with intractable focal epilepsy. The ictal EEG patterns may be difficult to interpret because of patient movement and eye blinking, and a subtle epileptiform discharge may be difficult to distinguish from the background. In selected seizure types, such as auras or focal nondyscognitive seizures, there may not be a definite scalp-recorded EEG alteration.

Neuroimaging

Magnetic resonance imaging (MRI) plays a pivotal role in evaluating people with epilepsy who experience focal seizures. MRI is highly sensitive and specific for selected abnormal alterations associated with focal epilepsy including MTS, vascular malformation, and tumour. The most common imaging alteration in adults with intractable focal seizures is an area of localised brain atrophy or volume loss in the structures of the medial temporal lobe reflecting MTS. Unfortunately, patients with MCD may have an unremarkable MRI study despite a history of intractable epilepsy. Techniques that may increase the diagnostic usefulness of MRI in people with epilepsy who have MCD include performance of a 7 Tesla study, voxel-based morphometry assessing structural thickness, and use of a double inversion recovery sequence.

Single-photon emission computed tomography (SPECT) has been used in patients with intractable partial epilepsy to identify a focal cerebral perfusion or blood-flow abnormality, which may indicate the localisation of the epileptic brain tissue. There are conflicting findings on the diagnostic usefulness of interictal studies in patients undergoing a presurgical evaluation with medically refractory partial seizures. Ictal SPECT studies are more sensitive and specific than interictal examinations for lateralising (confirming on which side of the brain the seizures commence). Computer-aided subtraction of the interictal from the ictal SPECT images, followed by co-registration to the MRI (SISCOM), is a recent development that is diagnostically superior to routine visual inspection of the interictal and ictal scans. Patients with SISCOM images that revealed a localised abnormality were more likely to experience an excellent outcome following epilepsy surgery than individuals with unremarkable studies or imaging findings that conflict with video-EEG monitoring findings.

Positron emission tomography (PET) may help to identify a focal metabolic abnormality that may assist in surgical localisation in patients with drug-resistant focal epilepsy. The most common study used in the evaluation of intractable epilepsy is the ^{18}F-deoxyglucose (FDG)-PET, and is diagnostically successful in patients with temporal lobe epilepsy. PET is a reliable indicator of the temporal lobe of seizure origin in patients being evaluated for epilepsy surgery. PET's sensitivity in these individuals approaches 90 per cent. The false lateralisation rate for PET in patients with unilateral temporal lobe epilepsy is low: about 1–2 per cent. The PET findings in patients with temporal lobe epilepsy may be of prognostic importance in patients undergoing epilepsy surgery. The most common FDG-PET abnormality is a region of focal or regional decreased brain metabolism that corresponds to the localisation of the epileptogenic zone. The anatomical region associated with PET abnormality is characteristically larger than the pathological findings underlying the epileptogenic zone.

Treatment

The goals of treatment for people with epilepsy are no seizures, no side effects and no lifestyle restrictions. Seizure remission permits the person to become a participating and productive member of society. In some patients, managing underlying medical or neurological disorders associated with increased seizure tendency is pivotal. Avoiding seizure-provoking factors, such as excessive sleep deprivation, may also be important. Patients with recurrent and unprovoked seizures or single seizures with significant risk factors for subsequent seizure activity may benefit from anti-epileptic drugs (AED). The usual strategy involves selecting one AED (monotherapy treatment), and increasing the dose as necessary until the patient either enters a seizure remission or develops AED dose-related toxicity. The choice of medication depends on many factors including classification of the seizure disorder, seizure type(s),

potential adverse effects of the therapy, other health problems and medications, and the patient's age and gender. First-generation AEDs, such as carbamazepine and valproate, are still prescribed for selected patients as initial therapy. Second-generation AEDs, such as lamotrigine and levetiracetam, are increasingly being used as monotherapy.

The most important parameters in assessing drug dosing are efficacy (reduction in seizure tendency) and safety (drug toxicity). Patients may tolerate 'high' AED levels with monotherapy. Unfortunately, approximately one-third of people with epilepsy may have a medically refractory seizure disorder. Individuals who have not responded to two or more AEDs with recurrent seizures for two or more years should undergo a comprehensive epilepsy evaluation at an epilepsy centre. Treatment options for these patients include trying other AEDs, epilepsy surgery, vagus nerve stimulation, ketogenic diet or investigational therapies. The most effective treatment for drug-resistant focal epilepsy is surgery.

Conclusions

The medical care and management of people with epilepsy must go beyond rendering the person seizure-free; it must also permit the individual to become a participating and productive member of our society. Current diagnostic techniques and therapeutic options allow most patients to experience a significant reduction in seizures and an improvement in their quality of life.

Professor Gregory D Cascino

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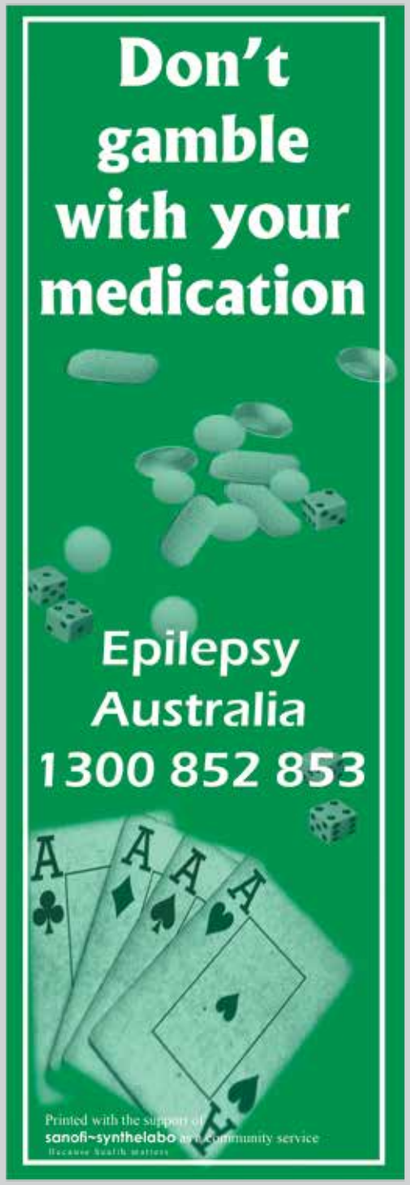
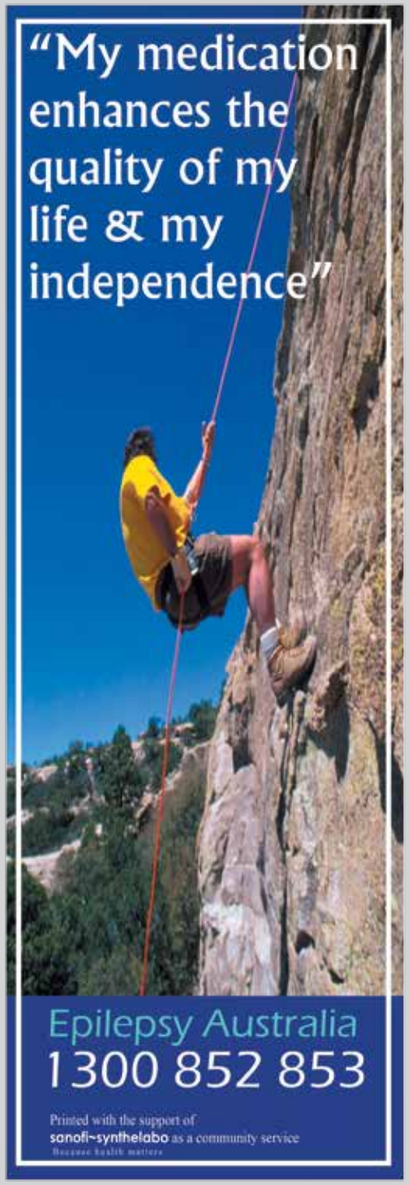




The evolution of medical therapies for epilepsy

Therapies for the treatment of epilepsy have been slowly evolving over the past century. From the time of recognition of the value of bromides in 1857 to the use of phenobarbitone in 1912, then to the gradual proliferation of medications currently available, this evolution has been a challenging journey of discovery, failure, scientific creativity and a major shift in the understanding of the condition of epilepsy and of what treatment is designed to achieve. The principles and strategies need to be illustrated first and some comments on the individual drugs will follow.

Epilepsy is a serious, life-threatening affliction, and early therapies, aimed at minimising seizure manifestations alone, were regarded as a major advance. Although these agents were able to reduce events, their side effects were serious, numerous and posed limitations on their use. Only much later was it realised that adverse effects could be reduced by newer agents and better strategies. The first systematic search for such agents yielded the discovery of phenytoin in 1938. This was effective in controlling seizures, and had fewer side effects than barbiturates, although it caused numerous dose-related neurotoxic side effects, metabolic and hormonal effects, gingival hyperplasia (enlargement of the gums) and occasional lymphomas (blood cancers). The combination of these two agents appeared superior in efficacy and up to the late 1960s phenobarbitone and phenytoin were marketed as a combination tablet.

Different types of seizures have been recognised for centuries, but it was only in the 1950s that drugs of the oxazolidinedione group were introduced for treating classical absences, known as ‘petit mal’. This condition was the first to be confirmed by specific electroencephalographic changes, the classic spike-and-wave pattern.

For the remainder of major seizure types, phenytoin held sway for almost 40 years. Only the formal classification of seizures, in parallel with new drug development, led to a clear therapeutic distinction, or rather a perception, recommending treating primary generalised seizures with phenytoin and partial-onset seizures with carbamazepine. This perception was slowed by the fact that in the United States carbamazepine, although developed by the early 1960s, was not approved for the treatment of epilepsy, but only for treating tic douloureux (trigeminal neuralgia—a severe, stabbing pain to one side of the face), because an early experimental batch of the drug may have been contaminated and caused blood abnormalities, which has haunted our American colleagues ever since. In Australia the benefits of carbamazepine versus phenytoin were recognised by the mid-1970s, and carbamazepine was established as the first-line drug for treating the most prevalent and difficult to treat condition of complex partial epilepsy, a role that now needs revision.

The syndromic classification of epilepsy helped markedly in understanding the complexities and manifestations of the various diseases called the epilepsies but, regrettably,

Cat. 113 German, **Lidded jar**, early 19th century, glazed earthenware with underglaze painting, 21.5 × 11.9 cm diameter, labelled *S Nitr: Dep:*. MHM01724, Medical History Museum, University of Melbourne.

nearly all clinical trials for evaluating new drugs express their results in terms of seizure types, rather than targeting syndromes. A further problem with clinical trial data as applied to therapies for patients with epilepsy is that the criteria for success—or partial success—is usually expressed as a percentage reduction in seizures, which is misleading. We ought to aim at seizure-freedom, rather than a reduction of seizures, because even a single seizure may have serious consequences for a sufferer in two ways: firstly, in terms of emotional effects and secondly, in practical terms, such as inability to hold a driver's licence.

Epilepsy is associated with multiple types of emotional disturbance. Depression, anxiety, sleep deprivation, personality changes and possibly psychotic features may need to be taken into account and may need treatment in their own right.

Tolerability of medications is a pivotal limiting factor for efficacy. If a drug is not well tolerated, patients will not take it as prescribed. While specific drugs may have preferential effects in some epilepsy syndromes, factors other than the seizures themselves may need to be taken into consideration, such as ethnicity, age, gender, childbearing status, other disorders and medications, renal disease and many more. For example, up to 30 per cent of patients of Chinese origin may have a positive HLA-B*1502 allele abnormality, posing the risk of an erythema multiforme-type skin eruption (a severe rash) with a potentially lethal outcome if carbamazepine is prescribed.

We recognise that specificity of drugs applied to treating specific syndromes is unimpressive. That is, they all seem to work for most types of epilepsy, and tolerability issues have become a main focus in individualising treatment.

A further issue is that of doses. To quote the words of Philippus Aureolus Theophrastus Bombastus von Hohenheim, known as Paracelsus, in 1493: 'Poison is in everything, and no thing is without poison. The dosage makes it either a poison or a remedy'. The dose of an anti-epileptic drug that is effective in seizure control is not much lower than the dose that causes potentially serious adverse effects. This is referred to as a narrow therapeutic range. The guiding principle is to start at a low dose and increase it gradually in order to prevent morbidity and to permit other drugs to be added in low doses to improve seizure control.

The practice of adding drugs to a regimen already stabilised, but not fully effective, has undergone major changes in the past 15 years. Edward H Reynolds in the 1970s was highly influential in proclaiming that monotherapy (using one medication only) was the ideal way to treat epilepsy. This was echoed by the experts, who proclaimed that treating secondarily generalised partial seizures with carbamazepine should consist of increasing the dose to the limit of side effects and beyond. In that era it was not uncommon to see patients so heavily over-treated that their quality of life was significantly undermined. Monotherapy has undoubted advantages in being often effective, free of drug interactions, and side effects can be readily discerned. More recently however, polytherapy has become an accepted reality, and following the availability of newer, better-tolerated medications, many patients

Cat. 70 **Talbot Colony for Epileptics, school room**, 1907, photograph, 18.0 × 23.0 cm. Epilepsy Foundation Collection.

are enjoying the benefits of individually adjusted combinations of lower-dose anti-epileptic therapy.

Epilepsy is often a genetically based disorder and because of the immense number of genes present, the number of possible abnormalities is endless, as is the number of combinations of anti-epileptic drugs in polytherapy. Hence efficacy in resistant cases is largely dependent on trial and error, although guidelines do exist.

In the 1950s ethosuximide, the most widely used succinimide, was developed specifically to treat classical absences. An effective drug of limited spectrum, it readily supplanted the diones, which produced severe adverse effects on kidney function, reported by our Adelaide colleague, the late Richard Rischbieth. The benzodiazepines, initially diazepam and later clonazepam, have played a major role since 1966 in controlling status epilepticus. Clonazepam was used as an adjuvant to the main drugs in chronic therapy and this was supplemented by the introduction of clobazam, which is structurally different, well tolerated, and used as a contingency measure, with few and rare adverse effects.

Sodium valproate was introduced in France in 1968, based on a discovery that the excipient (inactive substance) used in testing barbiturates five decades earlier was more effective in animal studies against induction of seizures than any of the barbiturate derivatives. Introduced in Australia in 1974, valproate has for 40 years been a most effective anti-epileptic drug for treating most types of epilepsies, generalised or partial, with convulsive or non-convulsive features, at all ages, but treatment has been achieved at the cost of significant side effects in some cases, mostly stemming from a failure to recognise dose issues. The common manifestation of valproate side effects are, fortunately, not life threatening, and include weight gain, hair changes, a tremor, potential blood abnormalities, multiple drug interactions, interference with elimination of other drugs, altered liver function (but only very rarely liver failure) and other serious complications such as encephalopathies (brain disorders). Another major issue with valproate, clearly dose related, is the higher incidence of teratogenicity (birth defects), so it must be used with caution by pregnant women. It may be the only drug capable of providing total seizure control, so abrupt cessation or replacement may be a hazardous undertaking. An intravenous formulation is available if oral intake is not possible, for example, after an operation.

Between 1974 and 1992 few new anti-epileptic drugs were discovered until vigabatrin, designed to inhibit the breakdown of GABA (the important neurotransmitter gamma-aminobutyric acid), was introduced in Europe but not in the United States. Targeted to treat localisation-related seizures, it was very effective. But in 1994 a British ophthalmologist, Tom Eke, reported loss of peripheral vision in patients treated with vigabatrin, and this was shown to affect possibly 30 per cent of patients, complicated by the fact that children exposed to this drug cannot be reliably tested. The eye changes were later shown to be due to the deposition of GABA in the retina, causing irreversible loss of peripheral vision. This led to a dramatic reduction in the use of vigabatrin.

Cat. 67 **Talbot Colony for Epileptics, billiard room**, 1954, photograph, 15.0 × 21.0 cm. Epilepsy Foundation Collection.The men's faces have been affected by long-term use of the epilepsy medication phenytoin, which was introduced in 1938.

Felbamate was developed in the United States in circumstances that remain unclear, but that appear to have involved only short periods of exposure to and withdrawal from the medication in a monitored hospital environment for a period of less than a week. The approval for its introduction stated that it appeared effective against partial epilepsy, but its main value appeared to be against Lennox-Gastaut syndrome (a serious form of childhood-onset epilepsy). Fortunately felbamate never appeared in Australia as a major drug, because soon after its launch it was reported to be associated with an unacceptably high level of liver- and blood-related side effects. Many of these were fatal. Felbamate was based on meprobamate, a drug used in psychiatry, which had similar adverse effects. These should have been foreseen, but the information was slow to reach the regulatory authorities. So felbamate, advertised in glossy brochures in medical journals as being the ideal drug for treating epilepsy, with very effective seizure control, no side effects, well tolerated, and requiring no plasma level monitoring, failed.

Plasma level measurements in anti-epileptic therapy deserve a mention. Based on the work of Bernard Brodie and Henn Kutt in the United States, the concept that clinical management of epilepsy is aided by knowledge of drug levels in the patient's blood became accepted largely on the basis of studies of phenytoin, where it appears to hold true. Efficacy is more closely related to plasma levels than to doses administered, and this applies also to the detection of toxicity. In Australia, Mervyn Eadie's pioneering use of anti-epileptic drug concentrations was a major advance at the time, and was introduced into practice nationwide in 1974. It appears to have a major attraction for practitioners, but the procedure, and judgements based on the results of drug levels, have significant limitations. Plasma levels should be used only as an adjunct, and not lead to changes in prescription unless clinically indicated. There should be a good reason for ordering the test.

The concept of a therapeutic range—a numerical set of levels associated with good seizure control—is only a statistical, population-derived set of figures, not strictly related to control in an individual patient, which may be achieved at levels higher or lower than the so-called therapeutic range. We treat patients, not levels!

After 1994 a plethora of drugs was approved in Australia. Lamotrigine—the 'drug of the decade' in Britain—offered significant benefits over existing drugs in treating all types of epilepsy syndrome. It was desirable to have a drug producing a euthymic effect (calmness) rather than depression. The developers of lamotrigine tried to find a marketing niche and soon targeted women, using unseemly advertising against its competitor valproate, which cited cosmetic side effects and claimed that valproate was responsible for polycystic ovarian syndrome, a claim that is only partly correct. What the makers did not reveal was that lamotrigine was subject to increased elimination in pregnancy, due to sex hormones, with lamotrigine levels also falling in women on the oral contraceptive pill. Hence lamotrigine use in pregnancy needs dose adjustments, making it difficult to use. But its synergism with valproate was proven by Martin Brodie, and lamotrigine is also the least teratogenic drug in pregnancy. Side effects include a rash related to Stevens-Johnson syndrome but, above all, lamotrigine is far less effective than valproate in patients with genetic generalised epilepsy.

Zonisamide, an American discovery, was to undergo clinical trials in the 1990s but kidney stones were reported in Caucasian patients. The drug was withdrawn from trials, but continued to be employed in Japan, where it has become an excellent broad-spectrum, well-tolerated anti-epileptic drug. Its reintroduction in Australia in 2011 followed an assessment of 20 years of successful exposure in Japan. It may yet become one of the best-tolerated drugs in our armamentarium.

Gabapentin replaced zonisamide in Australia in the late 1990s. Designed to treat partial epilepsy, its dose recommendations were markedly underestimated. Comparison with other anti-epileptic drugs showed a low efficacy, and later the doses were greatly increased. Its effects on neuropathic pain and psychological disorders were more striking, which led to the development of isobutyl GABA or lyrica, specially targeted to a population not with epilepsy, but suffering from pain and emotional problems.

Tiagabine, from Denmark, based on the GABA molecule, was short-lived in practice because of adverse gastrointestinal effects and development of absence-type seizures in patients who were treated primarily against localisaton-related seizures.

Topiramate is a very effective drug, with five mechanisms of action. Its side effects are not life threatening, but it is less tolerable than many other agents. It is associated with predictable weight loss, and appears to be effective across many epilepsy syndromes. Dosage requirements at the time of introduction were overestimated. It remains an excellent second-line drug, moderately safe in pregnancy.

Early in 2000, the most significantly effective new drug was approved. Levetiracetam has been shown in animal studies to have a better safety profile than any other anti-epileptic drug. It has shown efficacy predominantly in patients with partial epilepsy. It is effective even at doses lower than the recommended range, is well tolerated and the only question mark is a tendency to produce depression in a small percentage of patients. Levetiracetam is not teratogenic and is likely to replace carbamazepine as the first-line drug in the treatment of partial-onset seizure, although at present it is restricted to add-on status. It has no significant drug interactions, no catastrophic side effects, and is also effective in genetic generalised epilepsy. It is also available as an intravenous preparation, for people unable to take it orally.

Lacosamide has a novel mechanism of action, on slow voltage-gated sodium channels. It is an effective, well-tolerated drug, may be used in conjuction with others and has the potential to become a major addition to the armamentarium.

The field of anti-epileptic drugs is littered with agents that have tried, but failed, to make an impact. Second-generation drugs are more tolerable, hence they provide a new level of improved seizure control. We still only treat seizures, rather than the underlying epilepsy disorder. No drug devised so far has been proven neuroprotective (preventing loss of brain cells and function), but measures such as surgery are offering a great service in particularly difficult cases.

The stark reality is that over 90 per cent of patients with epilepsy are treated medically. Their treatment should be based on individual personal issues as well as seizure manifestations, and we must always be mindful of the severe limitations of each of the old and new anti-epileptic drugs.

Professor Frank Vajda

Surgery for treating epilepsy

Approximately 30 per cent of patients with epilepsy continue to have seizures despite taking anti-epileptic drugs, the primary therapy for controlling epilepsy. These patients with drug-resistant epilepsy have significantly impaired quality of life and independence, with an increased risk of injuries, medical complications, hospital admissions, mental health conditions and premature death.

The most effective treatment for these patients is resective epilepsy surgery, in which the region of the brain that is generating their seizures is surgically excised (removed). Resective epilepsy surgery is not suitable for all patients, but in those who are found to be ideal candidates, up to 80 per cent will have their seizures completely controlled by the surgery; this usually leads to dramatic improvements in their quality of life, safety, mental health and risk of death. Resective epilepsy surgery had been performed on selected patients for almost 130 years. But advances over the last two decades in technology to localise (identify very accurately) the source of the seizures in the brain have allowed surgery to be performed successfully on many more patients. For patients for whom resective epilepsy surgery is not possible or successful, a number of new technological devices—neurostimulators—can improve seizure control.

Resective epilepsy surgery

History

Surgical resection for mostly traumatic brain injuries was pioneered by Sir Victor Horsley at Queen Square Hospital, London, in 1886, using the clinical features of the patient's seizures to localise their source in the brain and guide the site of craniotomy (temporary removal of part of the skull) and resection. In the 1930s Wilder Penfield and Herbert Jasper at the Montreal Neurological Institute in Canada used intra-operative electrocorticography (brain wave recordings) during awake craniotomy to map the source of the seizures, and developed the techniques of resective surgery as it is known today.

In Australia, resective epilepsy surgery was first performed in 1894 at St Vincent's hospital by George Adlington Syme (see p. 72), but in modern times in the 1970s at the Austin Hospital in Melbourne, led by Professor Peter Bladin, and at the Prince of Wales Hospital in Sydney, led by Professor Rod MacKenzie. But epilepsy surgery remained a relatively uncommon procedure in Australia and worldwide until the 1990s, when the introduction of high-resolution magnetic resonance imaging (MRI) and other types of imaging, in particular

Cat. 101b Savigny & Co., **Trephining instrument set, in fitted case**, c. 1840; brass, steel, ebony, bristles, wood, paint, varnish and velvet; 5.2 × 20.6 × 10.2 cm (case). MHM00058, Medical History Museum, University of Melbourne. Trephining (trepanation), or making a hole in the skull, was used in many cultures in attempts to cure a range of mental, spiritual and physical conditions, including epilepsy. This set of instruments belonged to Edward Barker, first lecturer in surgery at the University of Melbourne.

positron emission tomography (PET) and single-photon emission computed tomography (SPECT), allowed the area of the brain causing seizures to be more readily identified. Since that time, epilepsy surgery has become much more widely available, with centres opening up across the country, including three additional centres in Melbourne associated with University of Melbourne research programs at St Vincent's, the Royal Melbourne, and Royal Children's hospitals. Resective epilepsy surgery is now considered the treatment of choice for suitable patients who have drug-resistant epilepsy.

Indications

Surgery should be considered for patients with drug-resistant epilepsy because of the increased death rate and progressive cognitive and psychosocial problems associated with uncontrolled seizures over many years. There is emerging consensus that once drug resistance is demonstrated (defined as the failure of two appropriately chosen and used anti-epileptic drugs), patients should be promptly referred to a specialty epilepsy centre that offers surgery. In some situations, such as catastrophic epilepsy in children, patients should be referred urgently because of the risk of severe developmental disability.

A case-by-case assessment is needed. In addition to results of diagnostic tests, the patient's and their family's perceptions of the severity of the epilepsy despite optimal medication, and their expectations for the future, are important considerations in reaching a decision on whether surgery is the right course of action.

Types of procedure

The type of surgical procedure performed depends on the indication. The most common procedure is removal of part of the temporal lobe, first described by Murray Falconer and colleagues in the 1960s. Different centres and surgeons vary in the amounts of temporal neocortex, parahippocampal gyrus, hippocampus and amygdala that they resect. In well-selected cases, 70–80 per cent of patients can become seizure-free, with the risk of serious complications—for example, hemiparesis (weakness on one side of the body) or hemianopia (visual field loss)—reduced to less than 5 per cent

Other potentially curative procedures include removal of discrete structural lesions such as glial tumors and vascular malformations. In a palliative procedure (for example, hemispherectomy, functional hemispherotomy, corpus callosotomy, multiple subpial transection), the focus of the seizure is not resected. Instead, the aim of the operation is to disrupt the pathways important for the spread of epileptiform discharges in order to reduce the frequency and severity of the seizures. Corpus callosotomy (separation of the two halves of the brain) is a treatment option for patients with severe generalised epilepsy, particularly atonic seizures (in which the person's muscles suddenly lose strength) with frequent falls and subsequent injuries. Multiple subpial transection (small cuts to disconnect parts of the brain's surface) is performed when the epileptogenic

lesion cannot be removed because of its close proximity to the eloquent cortex (areas with important functions such as motor and speech control), while hemispherectomy is a more drastic procedure in which an extensively diseased and epileptogenic cerebral hemisphere is removed, or left in place but electrically disconnected from other brain structures.

Pre-surgical evaluation

It is essential that all patients being considered for epilepsy surgery undergo a comprehensive, multidisciplinary pre-surgical evaluation in a centre that specialises in epilepsy surgery. This evaluation aims to establish whether the person has drug resistance, delineate the source of their seizures and demonstrate that its removal will not cause additional unacceptable neurological or cognitive problems. In practice, the evaluation involves a number of processes:

- A thorough review of the patient's seizure history and medication use is undertaken.
- Sophisticated video-EEG monitoring localises the onset of a number of seizures that are typical for the particular patient.
- High-quality MRI with dedicated 'epilepsy surgery protocol' increases diagnostic accuracy.
- Functional imaging such as SPECT or PET scanning, when necessary, delineates a potential epileptogenic zone.
- Neuropsychological testing where appropriate, aided by functional MRI and, less commonly, injection of the barbiturate amobarbital into the carotid artery, defines which side of the brain contains language and memory functions.

Intracranial EEG monitoring

If scalp EEG data do not clearly identify the seizure source, or if the neuroimaging and/or neuropsychological testing results are inconsistent with the findings of the scalp electrical recordings during seizures, 'invasive' electrodes may be implanted into the patient's brain for further, more accurate, seizure recording. Subdural strips and grids for intracranial EEG recordings have long been used to help find which part of the brain is causing seizures in patients with severe focal epilepsy. However, the manner in which intracranial recordings are now being used by many epilepsy surgery programs is very different from how they have traditionally been used. In contrast to the previously common use of 'blind implantations', when non-invasive techniques were non-localising, the strategy for the intracranial implantation now incorporates the results of modern neuroimaging. The intracranial EEG is used to test or refine the hypothesis for the localisation of the epileptogenic zone put forward by the results of structural and functional neuroimaging in a multi-modality image-guided approach.

Intracranial EEG recordings with surgically implanted subdural grids, strip or depth electrodes use stainless steel or platinum electrodes embedded in a synthetic material. These are implanted near the suspected epileptogenic zone via craniotomy or burrhole methods. In addition to recording EEG signals, subdural electrodes also facilitate 'mapping' of eloquent cortex by applying electrical currents, so that these areas may be avoided during the resection operation. Depth electrodes are implanted into the brain, and therefore have the advantage of being able to record EEG signals deep to the cortical surface, which may have not been detected with subdural electrodes. However, they are associated with a greater risk of bleeding in the brain and damage to brain structures. This risk can be reduced by the use of modern image-guided surgical implantation techniques.

Neurostimulation

Patients with drug-resistant epilepsy for whom resective surgery is not suitable may benefit from electrical stimulation therapies, including vagus nerve stimulation and, more recently, direct brain stimulation.

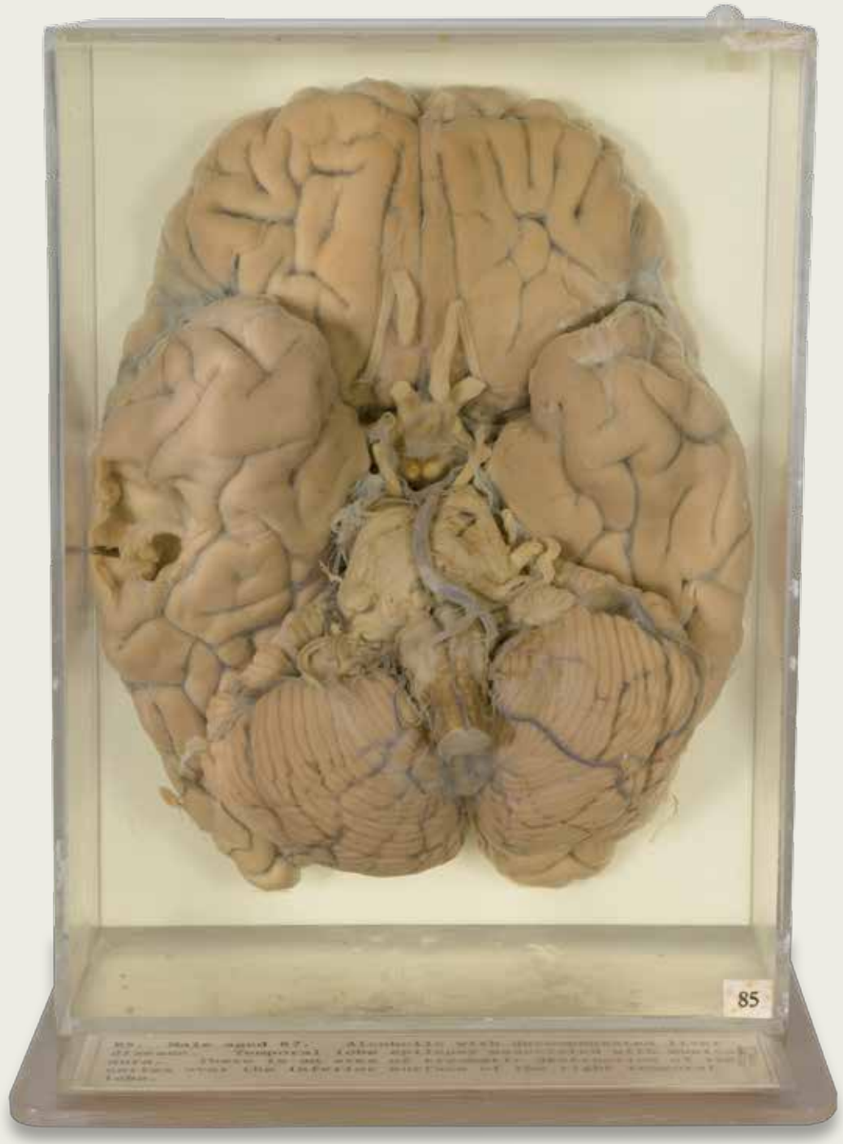
Vagal nerve stimulation

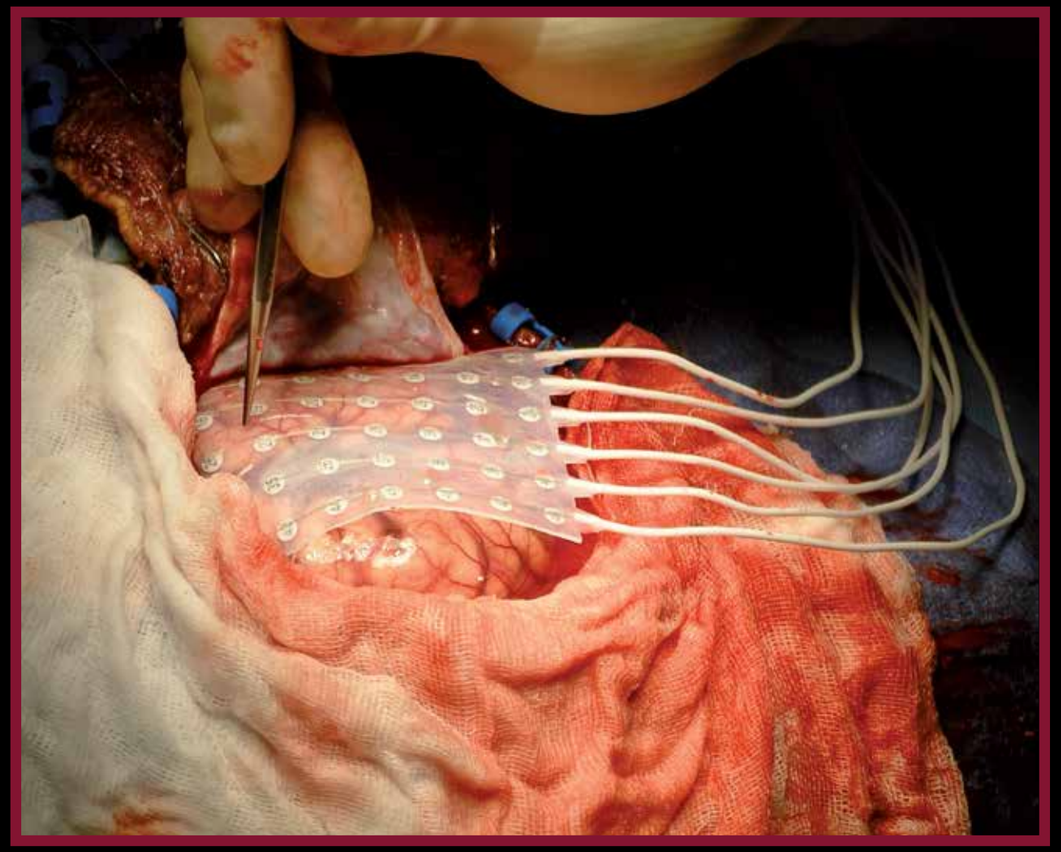
Vagal nerve stimulation (VNS), introduced in the 1990s, consists of a programmable signal generator that is implanted in the patient's left upper chest, a bipolar lead that connects the generator to the left vagus nerve in the neck, a programming wand that uses radiofrequency signals to communicate non-invasively with the generator, and a hand-held magnet used by the patient or their carer to turn the stimulator on or off. The mechanism of action of VNS is unknown.

Once programmed, the generator will deliver intermittent stimulation to the vagus nerve until any additional instructions are received or until the battery life is expended (typically after six years of operation with the latest model). In addition, the patient or a companion may activate the generator by placing the magnet over it for several seconds; in some patients, this may interrupt a seizure or reduce its severity if used near the start of the seizure.

Clinically important seizure reductions can be achieved with VNS in over 50 per cent of patients, but fewer than 10 per cent have become seizure-free. Side effects are transient and include pain at the site of the implantation surgery, coughing, voice alteration, chest discomfort and nausea. No cognitive, sedative, visual, affective, behavioural or coordination side effects have been reported; hence the typical central nervous system problems associated with anti-epileptic drugs are conspicuously absent with VNS therapy.

Cat. 98 **Human brain specimen**, 18.5 × 14.0 × 9.0 cm. 516-100713, Harry Brookes Allen Museum of Anatomy and Pathology, University of Melbourne. This was the brain of a 67-year-old man, an alcoholic with liver disease. He had temporal lobe epilepsy associated with musical aura. There is an area of traumatic destruction of the cortex over the inferior surface of the right temporal lobe.





Direct brain stimulation

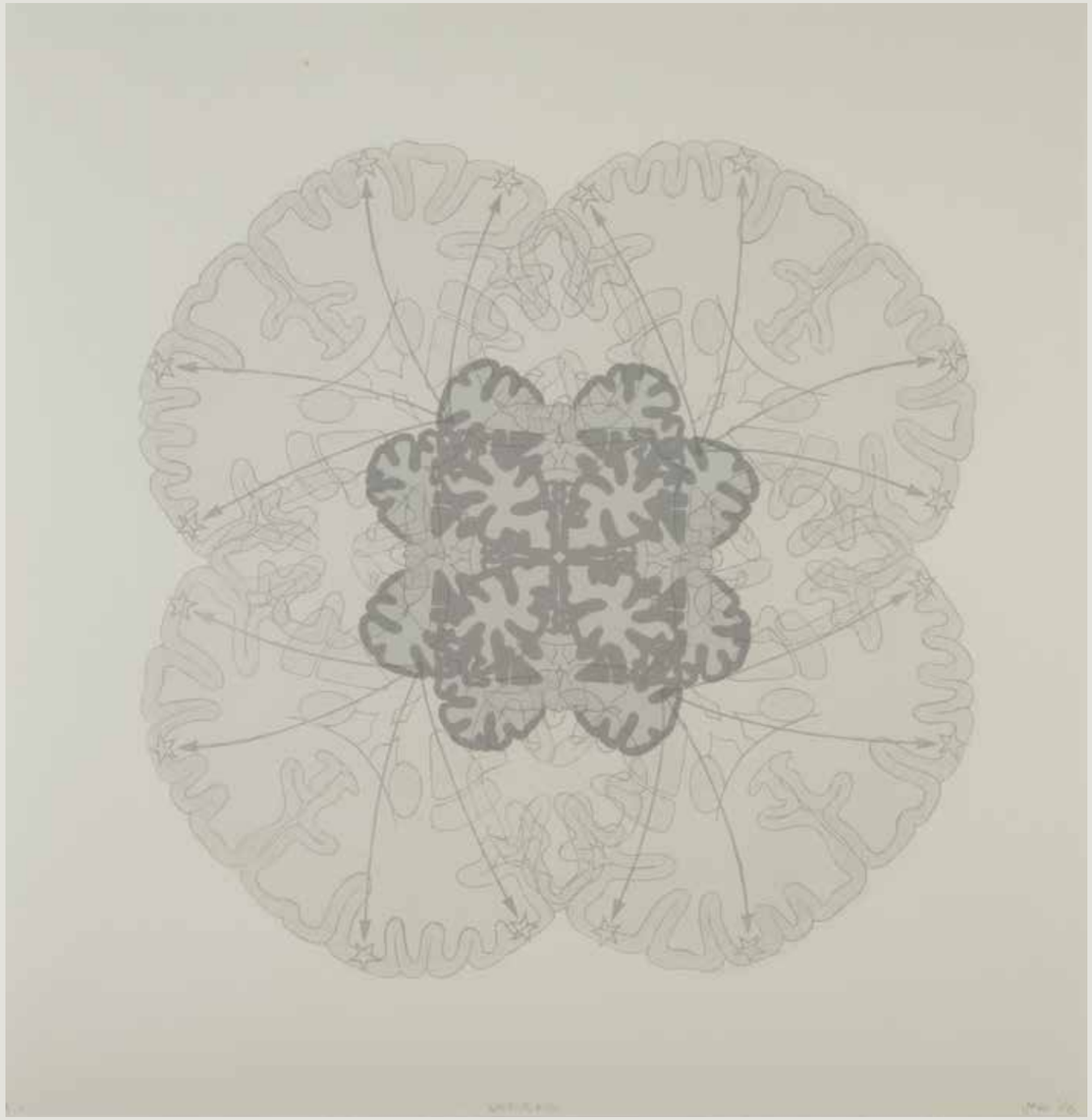
Following its success in treating movement disorders, direct brain stimulation (DBS) is under active investigation as a non-pharmacological therapy for patients with medically intractable epilepsy who would not benefit from resective surgery. Electrodes are inserted using a computer-guided system and connected to a battery-powered programmable stimulator located under the skin, which delivers intermittent signals of electric current. A variety of anatomic structures have been targeted. In the recently completed SANTE (stimulation of the anterior nucleus of the thalamus in epilepsy) trial, bilateral stimulation of the anterior nucleus of the thalamus in patients with severe focal epilepsy was associated with significant seizure reduction in the short term, resulting in approval of this device in Europe and Australia.

In another system, automated seizure detection is incorporated into an electric brain stimulator to form a closed-loop system so that stimulation is delivered to the epileptogenic zone when onset of seizure activity is detected. This system has undergone testing in a clinical trial and the results are under review by regulatory authorities.

Brain stimulation represents an exciting area of development, although further studies are needed to document its long-term efficacy and safety in order to define its place in the clinical management of epilepsy.

Professor Patrick Kwan and Professor Terence J O'Brien

A subdural grid of electrodes is placed in a patient's head in order to record electrical activity (brain waves) via electroencephalogram (EEG) directly from the brain surface, and to map areas with important functions by applying electrical stimulation. Image courtesy of St Vincent's Hospital, Melbourne.



Cutting edge: Genetics

The ugly past

Genetics has had an extremely chequered history in epilepsy. During the period in which people with epilepsy were thought to be possessed by demons, a period that sadly still extends to some pockets of the world today, the hereditary nature of many epilepsies was one of the major factors leading to discrimination against people with epilepsy. A number of the world's major religions, and certain governments, had prohibitions on people with epilepsy marrying and bearing children because of the 'hereditary taint'. Epilepsy was one of the conditions that attracted the attention of the eugenics movement, which reached its ugly peak of influence in Nazi Germany.

Against this background of appalling discrimination, with its dire psychosocial consequences for patients and families dealing with epilepsy, the medical and scientific analysis of the genetics of epilepsy was suppressed. Indeed, genetic factors were strongly denied. This of course was counterproductive to progress in the scientific understanding of epilepsy and in removing the stigma associated with epilepsy.

Genetic studies enable us to understand certain epilepsies by firstly, and importantly, establishing a cause. They give insights at multiple levels: from the fundamental level of molecules, which form the building blocks of the human body, to the brain and whole person. They provide understanding of other disorders, or comorbidities, that keep company with epilepsy, such as learning or psychological difficulties. Genetic studies offer the realistic hope that deep understanding of the mechanisms underlying seizure disorders will lead to the development of innovative and targeted therapies. Moreover, with some exceptions, most people with epilepsy where there is a major genetic influence can now be easily and effectively treated.

Present understanding

There is now good evidence that genetics plays a role in many patients with epilepsy. There are relatively rare examples where the disorder is due to a single gene and the rate of recurrence in the family is high; this is called Mendelian inheritance, after Gregor Mendel, who defined the basic laws of single-gene inheritance in peas over a century ago. Here it might be said that epilepsy runs through the family 'like a golden thread' akin to other Mendelian disorders, although in the epilepsies, this is uncommon.

Cat. 28 Jessica Merrell (USA), **Untitled**, 2006, lithograph on paper, 87.0 × 76.0 cm. Collection of the artist.

However, such Mendelian epilepsies provided the first examples where genes causing epilepsy could be found using the early technologies of molecular genetics. In the early 1990s we and other groups began studying rare large families in which many members had epilepsy, in the hope of identifying genes with the available techniques. We were successful and identified the first gene for epilepsy in 1995, plus numerous others since.

The key was our ability to intensively study very large families. Australia gave us a competitive advantage—people were often very cooperative and keen to help, and families often stayed together, unlike the situation in North America. Also, the stigma of epilepsy was somewhat less here than in other societies, which meant that families were happy to participate in our research in order to help their family and the wider society.

A fundamental observation that arose from this early work was that many of the genes responsible for Mendelian epilepsies encode proteins for ion channels. Ion channels are gateways into our cells that allow the passage of charged particles (ions). They regulate the transmission of sodium, potassium, calcium, chloride and other ions across cell membranes and regulate the excitability of nerve cells. This makes a lot of sense in a disorder of excitability like epilepsy, and knowledge of the ion channels involved is enabling new approaches to identifying novel drugs. Not all epilepsy genes affect ion channels; some epilepsy genes encode proteins important for the synapses or links between nerve cells, whereas others affect the way nerve cells grow and develop.

More usual is that people with epilepsy have so-called 'complex inheritance', where a number of genes and environmental factors contribute to the disorder in one individual. Here the risk to close relatives is much lower than for Mendelian disorders. Overall, relatives of people with epilepsy have about a threefold greater risk of having epilepsy, compared to the general population, but this depends on the epilepsy type—the risk is higher in those with generalised epilepsy (both sides of the brain involved) and lower in those with focal epilepsies (beginning in one part of the brain).

The revolution in genetics over the last 20 years with sequencing of the human genome and enormous technological advances in the speed and availability of genome sequencing has opened up a new world in epilepsy genetics. We have now identified numerous causes of Mendelian epilepsy, and the genes contributing to complex inheritance are slowly being unravelled. Many surprises have emerged. In addition to so-called 'point mutations', where one sequence change occurs in the DNA strand and thus changes the protein, some epilepsy mutations are known as copy number variants (CNVs), where large segments of DNA are either deleted or duplicated. These CNVs typically encompass a number of genes that might intuitively be expected to have very substantial clinical effects. While this is true for some CNVs, others, surprisingly, act not as genetic mutations that invariably lead to disease, but as factors that raise the risk for complex epilepsies where the subject may or may not develop a mild form of epilepsy.

The exciting future

The landscape of epilepsy genetics is rapidly expanding and its applicability is now well and truly entering the clinic. It is highly relevant to Precision Medicine, a new concept encompassing individualisation of diagnosis and treatment to the particular patient, rather than the one-size-fits-all approach to broad diagnostic categories that has been the general strategy in medicine to date.

The best example is that of the epileptic encephalopathies, a group of severe epilepsies that typically begin in infancy or childhood, and are associated with serious comorbidities including intellectual disability and autism spectrum disorders. Until recently, the cause of the majority of cases was unknown. The clinical approach was to classify patients on the basis of clinical and electroencephalographic features (EEG, brain wave recording), and treatment was empirical (based on observation of the treatment's effects, rather than on understanding the underlying processes). Moreover, the lack of a specific cause often set parents on a frustrating search for answers. Now we are learning that a large proportion of the epileptic encephalopathies are genetic in origin, and are explained by a new genetic mutation in the affected child. This finding explains the absence of epilepsy in the child's family: the disorder is genetic but not inherited. We have also found that the spectrum of disease due to defects in a specific gene can be wide. These observations led to increased identification of genetic causes in other individuals. Gene identification is now affordable and allows a Precision Diagnosis framework, which allows families to finish their search for a cause and to benefit from accurate family counselling. Moreover, the possibility of Precision Therapy is now real, with specific drugs or diets that may control the seizures available for some patients.

Further research is likely to enable Precision Diagnosis and Precision Therapy for an increasing proportion of cases. Moreover the usefulness of pharmacogenomics, where we can study an individual patient's genetic profile to predict their response to drugs and minimise side effects, will increase. For example, we know that the risk of Stevens-Johnson syndrome, a rare, but very severe, skin reaction to a commonly used anti-epileptic drug, carbamazepine, is much higher in patients of Asian origin who have a specific normal variant in a particular immune-response gene. Many similar examples will follow with increased understanding of pharmacogenomics.

Genetics has and will continue to greatly transform diagnosis and treatment for patients with epilepsies over the next few years. Clinical studies, together with laboratory science, will help us to understand what causes seizures and allow us to develop therapies targeting the genetic defect. This will not only help to control seizures but will improve the individual's outcome and lower their risk of associated comorbidities. The ultimate aim of genetic science is to prevent epilepsy from occurring in any person at risk of developing this serious disorder.

Professor Sam Berkovic and Professor Ingrid Scheffer

Epilepsy: Future directions

Despite a wealth of scientific advances in recent decades, there has been relatively modest progress in treating the condition of epilepsy. The number of available anticonvulsant medications has increased significantly, but the proportion of epilepsy patients whose condition remains inadequately controlled is about the same: around 30 per cent.

The ability to visualise the source of seizures with magnetic resonance imaging (MRI) and electroencephalography (EEG) generated great hopes for the more widespread application of surgical intervention, but this has not transpired, for a range of logistic and technical reasons. New pharmacological therapies will continue to be developed, and insights acquired through better understanding of the underlying mechanisms, particularly the genetic underpinnings of some of these epilepsies, will redirect drug research. Ultimately, more effective and better-tolerated pharmaceuticals will result.

Our ability to localise the origin of seizures though a variety of means has meant surgical resection is now a viable treatment option for a great number of individuals. Advanced stereotaxic methods harness developments in computing and imaging to make surgery more precise. Specific techniques such as functional speech and motor localisation, as well as tractography using MRI, have made surgery much safer, avoiding injury to eloquent cortex (areas of the brain involved in sensory processing, fine motor control, and linguistic ability) and identifying and avoiding the cabling joining the brain regions. The benefits of these techniques are immeasurable and, combined with other methods of seizure localisation, have unquestionably improved the outcomes of this type of therapy. The demanding nature of the surgical assessment and the small number of facilities possessing the necessary expertise have, however, limited access. But development of less resource-intensive methods and greater availability of the necessary surgical skills will make these treatments increasingly available.

Development of the EEG machine enabled us to visualise the electrical correlates of seizures and helped us diagnose and categorise the condition accurately—the first steps in management. More recently, techniques in mathematics, signal analysis and network theory have brought a new group of researchers into the field, bringing with them new insights into how seizure activity begins and spreads. Most noteworthy has been the identification of the changes in brain networks that underlie the manifestations of seizure, and perhaps the basis of the condition itself. At one time individual neurons were thought to be the key to understanding the process of epilepsy, but now we understand that vast ensembles of neurons are involved. Sufficiently powerful computers and the ability to store and manipulate the enormous amounts of data necessary to understand these relationships have only recently evolved, and we are currently at the edge of understanding these relationships.

Professor Mark Cook (standing left) with colleagues Dr Dean Freestone and Dr Amy Halliday participating in epilepsy research, 2013. Digital image, photographer Gavin Blue.

Imaging advances, particularly in the field of MRI, have dramatically changed the landscape. In the days of computerised tomography (CT) scanning, only infrequently could the abnormality causing epilepsy be accurately identified, and until the late 1980s the location and nature of the underlying pathology was established by analysing the signs and symptoms of the seizures, much as the pioneering English neurologist John Hughlings Jackson (1835–1911) had done a century earlier. The EEG brought about a dramatic change, but it was not until MRI technologies became widespread that the pathological basis of seizure activity could be defined in most people with epilepsy. The techniques have been further refined and elaborated, with functional MRI techniques revealing not only how normal brain functions are distributed, but even seizure activity itself. Very powerful high-field systems can resolve to the cellular level, and are becoming increasingly available. Further improvements in these methods will undoubtedly be critical, not only for maximising the outcomes of surgery, but to expose the fundamental processes of the condition.

Apart from MRI, there is a huge variety of methods for imaging the brain and its activity. Magnetoencephalography measures extremely small changes in magnetic fields produced by brain activity. This supplies information complementary to EEG data but, unlike routine EEG, can record non-invasively the activity of remote parts of the brain. Because much of the surface of the brain is hidden in folds and under complex surfaces, this provides a unique means of visualising function. Near-infrared spectroscopy can directly measure changes in blood flow through the skull, and can identify these changes throughout the brain. In some ways similar to functional MRI, it is a much smaller device than an MRI machine, and significantly less costly.

Techniques using radionuclide imaging, such as positron emission tomography (PET) and single-photon emission computed tomography (SPECT) have already proven their worth. SPECT is of immense value in the surgical assessment of epilepsy, by localising seizure activity, but PET permits more elaborate analysis of cellular and regional function, with specific labelling compounds developed to characterise various aspects of function, such as blood flow, metabolism and receptor density. Sophisticated ligands (introduced substances that bond with biomolecules) could, in the future, be linked to known abnormalities in the cellular properties of specific pathologies, identifying the regions involved; it is conceivable that similar techniques could even be used to deliver therapies.

Genetic studies have become one of the most important components of our new understanding of epilepsy. Identification of specific genetic changes underlying certain seizure disorders caused a paradigm shift in our approach to analysis and classification, and will no doubt eventually do the same to diagnosis and therapy, as the new age of Precision Therapies promises the possibility of designing specific treatments for unique genetic abnormalities. Professors Sam Berkovic and Ingrid Scheffer discuss the development and application of these methods elsewhere in this book. Linking these developments to sophisticated imaging and computational analyses will no doubt offer even more penetrating insights.

Inflammatory diseases, including the group of illnesses known as limbic encephalitis, are very well known but were previously thought to be very rare. New testing methods have shown that these conditions are not as rare as we once believed. Because their treatment may include therapies not generally used in epilepsy, with implications for the prognosis, this will no doubt be a diagnosis of increasing importance. Understanding better the causes of these conditions and their optimal therapy will have major diagnostic and therapeutic consequences.

In the late 1980s, systems of modulating brain activity through stimulation of cranial nerves were shown to be effective in treating epilepsy. The earliest devices (a pulse generator implanted in the patient's chest, similar to a cardiac pacemaker) stimulated the vagal nerve in the neck, delivering a regular pulse that is thought to stimulate deep-brain structures in a way that supresses seizure activity. The technique does not work for a significant proportion of patients, and at present there is no way to identify responders in advance, though it is more effective for patients with dangerous 'drop attacks' as a feature of their seizures. Nevertheless, its relative lack of serious side effects make this an attractive proposition for many.

Two years ago a similar device, also stimulating a cranial nerve but in this case the trigeminal nerve of the face, became available. This system employs a similar strategy to vagal nerve stimulation, but has the advantage that it is entirely external, and very safe. Stimulation is carried out while the patient sleeps, and so is much better accepted by patients.

Direct electrical stimulation of the brain has been recognised as a potential therapy for epilepsy since at least the 1950s. Only recently has the means of powering and ensuring the functionality of such devices, as well as the engineering techniques required for electrodes suitable for long-term use, been available. In the last four years a deep-brain stimulation system, with electrodes implanted in the thalamus (an area near the centre of the brain), has become commercially available. This system delivers regular stimulation to deep parts and seems to modulate cortical activity, reducing seizure activity. A more sophisticated system has become available only this year, which can detect the onset of seizure activity and deliver electrical counter-stimulation directly to electrodes implanted in the abnormal region of brain. We can expect to see more devices of this kind because surgical implantation, although it involves some risk, has the potential to avoid the many unwanted consequences of chronic medication use. As yet the clinical efficacy of such systems is incompletely studied, and the more widespread use and analysis of these systems in the years ahead will be critical. As with vagal nerve and trigeminal nerve stimulation, the ability to identify those individuals likely to respond to such therapies will be a great advance, and a considerable amount of research is being carried out in this area.

Only recently have we been able to predict seizure activity accurately. Potentially this could have significant consequences, not only for day-to-day safety issues, but also for determining when to apply therapy. Sophisticated mathematical techniques underpin this but, at the moment, intracranial data (and hence invasive procedures) are required, which

hamper its widespread use. Researchers are currently exploring innovative solutions to this problem.

Another by-product of computer technology has been the development of small devices cooled by the Peltier effect (thermoelectric cooling). Originally intended to cool computer chips to improve their performance, such devices can be applied to focal epilepsy, with the resultant cooling suppressing seizure activity. Significant challenges remain in this work however, particularly around the practicalities of heat transfer and the energy source. Also, as it takes some time to effect the cooling, it may not be suitable for event-triggered treatment. Nevertheless it is an interesting system that may prove useful.

Finally, there are dietary therapies. The oldest described therapy for epilepsy, fasting, is recorded in the Hippocratic texts, and its rediscovery in the late 19th century ultimately led to the ketogenic diet (which forces the body to burn fats rather than carbohydrates). Recognition of the other effects of manipulating the body's metabolism in a way that influences brain function has brought about considerable interest in dietary treatments, and more recently a special oil additive has been proposed as therapy for epilepsy. Though long regarded as alternative medicines, such approaches may have great potential. We are, however, still very early in the discovery process.

Conclusions

Advances in engineering, computing, and imaging technologies are driving many new areas of research, many novel and innovative, into the causes and treatments of epilepsy. But significant challenges lie ahead, and we must overcome many technical obstacles before device-based therapies are widely available. Genetic studies hold the promise of insights that will improve both diagnostic and therapeutic aspects of management, and although personalised treatment is still not here, it is within our grasp.

Professor Mark Cook

Cat. 75 Victorian Bureau for Epilepsy, ***Expectation*** (detail, colour altered), quarterly magazine: issue no. 2, May 1966, print on paper, 30.5 × 23.0 cm. Epilepsy Foundation Collection.

Traditional treatments for epilepsy

It is not surprising that a condition that is so intractable and that has such a high profile as epilepsy should attract a host of treatments, some of them bizarre. But it is surprising that these persisted well into the 19th century and were documented in the medical literature of the time. Admittedly most of these reports were written in a somewhat light-hearted manner, but the fact that they were given valuable space in the journals is worthy of note.

The *British Medical Journal* in 1872 carried a note from Dr James Sawyer describing the recipe of Sir John Floyer of Lichfield, which had been published in 1710:

> The foetid parts of animals: powder of bore's bones, or of a horse, cock, ram or man's skull; powder of a secundine [placenta]; blood powdered 3ss [half an ounce]; the bones of a ferret's back; the spine of a fish. Take these with sugar in wine and black-cherry water for twenty days; drink four ounces of blood, taken in a warm porringer: dung of a peacock; the liver of an ass; the coagulum of a hare, the powder of a swallow, raven, jay, cuckoo, dry'd; the gall of a whelp; the liver of frogs; the warts of colts; crows' eggs.[1]

In addition, says Sir John, '*Augenius* cured an epileptic after 25 with two ounces of sanguinus mustelae in vinegar 3j [one ounce], musk in wine, human bones'.

It speaks volumes that this would be thought entertaining in a prime medical journal in the last quarter of the 19th century, although it can be said that the medical journals of today take themselves very seriously.

The *British Medical Journal* in 1880 quoted a *Daily Telegraph* report that Princess Bismarck praised dried magpie dust as a cure for epilepsy. She had been asking local landowners to shoot as many magpies as possible and send the lot to her.[2] It is well known that royalty and political power often turn to alternative medicine, and it is no surprise that the doings of Germanic royalty were of interest to late 19th-century English doctors. But these examples give us some idea of the resistance to jettisoning old-fashioned medicine cures for epilepsy, simply because of the lack of alternative medication.

Professor Peter F Bladin

1 'Column for the curious: Treatment of epilepsy', *British Medical Journal*, vol. 1, no. 576, 13 January 1872, p. 55.
2 'Letters, notes, and answers to correspondents: Magpie dust', *British Medical Journal*, vol. 1, no. 998, 14 February 1880, p. 270.

Reference:
Bladin PF, *A century of prejudice and progress: A paradigm of epilepsy in a developing society: Medical and social aspects, Victoria, Australia, 1835–1950*, Melbourne: Epilepsy Australia, 2001, p. 247.

Cat. 114, 115 and 116 **Three 19th-century French drug pots**. MHM2009.5, MHM2009.41 and MHM2009.40, Medical History Museum, University of Melbourne.

Death on the Ballarat Express, 1877

This report and illustration from Richard Egan-Lee's *Police News* of 30 June 1877 give us little information on whether the demise of Mr David McDonough was witnessed or not, nor any clinical history to tell us whether the unfortunate man was a known sufferer from epilepsy. It is clear however that this event was sudden and unexpected.

Whatever the circumstances, whether Mr McDonough died from a series of seizures witnessed by his travelling companions, or as the result of a single seizure, the event illustrates two aspects of epilepsy that have very significant, long-term effects on the community: the perception of the lethal significance of a diagnosis of epilepsy and the condition's implicit threat to public order.

This latter perception stemmed from, and was subsumed in the concept of, epilepsy being a process that could gravely distort brain functioning. Indeed, those people with epilepsy who were managed in the public health system in 19th-century Victoria were housed in 'mental asylums'. Happily, the days when this perception was widespread are now long gone in Victoria. But sudden death in epilepsy is still with us, albeit on a significantly reduced frequency, and demanding our keenest attention to proper management.

Anticonvulsant medication in 1877 was the merest shadow of what it is today, and the use of bromides, the first efficacy-proven medication, was just beginning to be accepted as part of epilepsy management. The use of bromides was by no means without complications and side effects, often reducing patient acceptance. Chronic bromism, a form of long-term brain poisoning, was common.

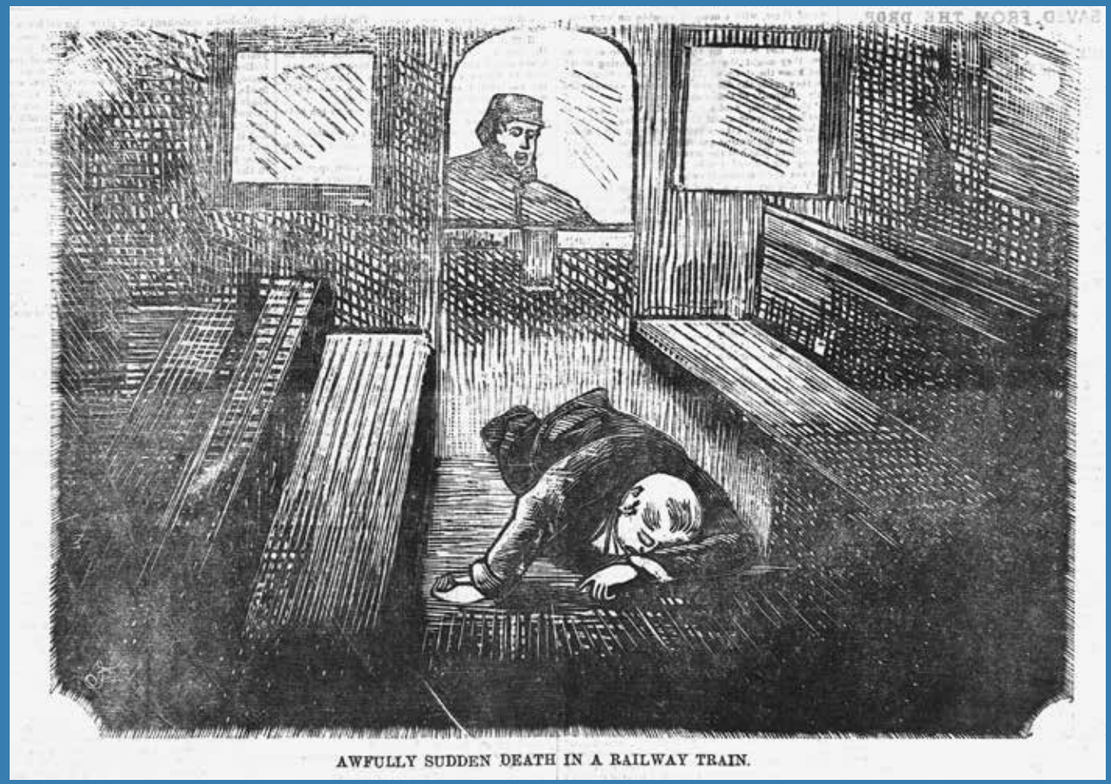
A lethal outcome could be the result of a single seizure or of a rapid series of seizures. We do not know whether anybody witnessed the demise of the unfortunate Mr McDonough, but the time needed for a train to travel from Melbourne to Ballarat was sufficient for either alternative to have occurred.

It was well known in asylums that seizures could pose grave problems for inmates, and that despite the use of bromide anticonvulsant medication this lethal disaster could occur.

Professor Peter F Bladin

References:
Bladin PF, 'Status epilepticus, the grim reaper of the mental health system in early Victoria', *Journal of Clinical Neuroscience,* vol. 10, no. 6, November 2003, pp. 655–60.
D'Souza W, 'Deaths from epilepsy: SUDEP', this volume, p. 84.
Smith P, 'On the treatment of epilepsy by large doses of bromide of potassium', *Australian Medical Journal*, vol. 18, 1873, pp. 258–63.

Cat. 121 ***Awfully sudden death in a railway train***, *Police News*, 30 June 1877, wood engraving. PN30/06/77/00, courtesy State Library of Victoria.



AWFULLY SUDDEN DEATH IN A RAILWAY TRAIN.

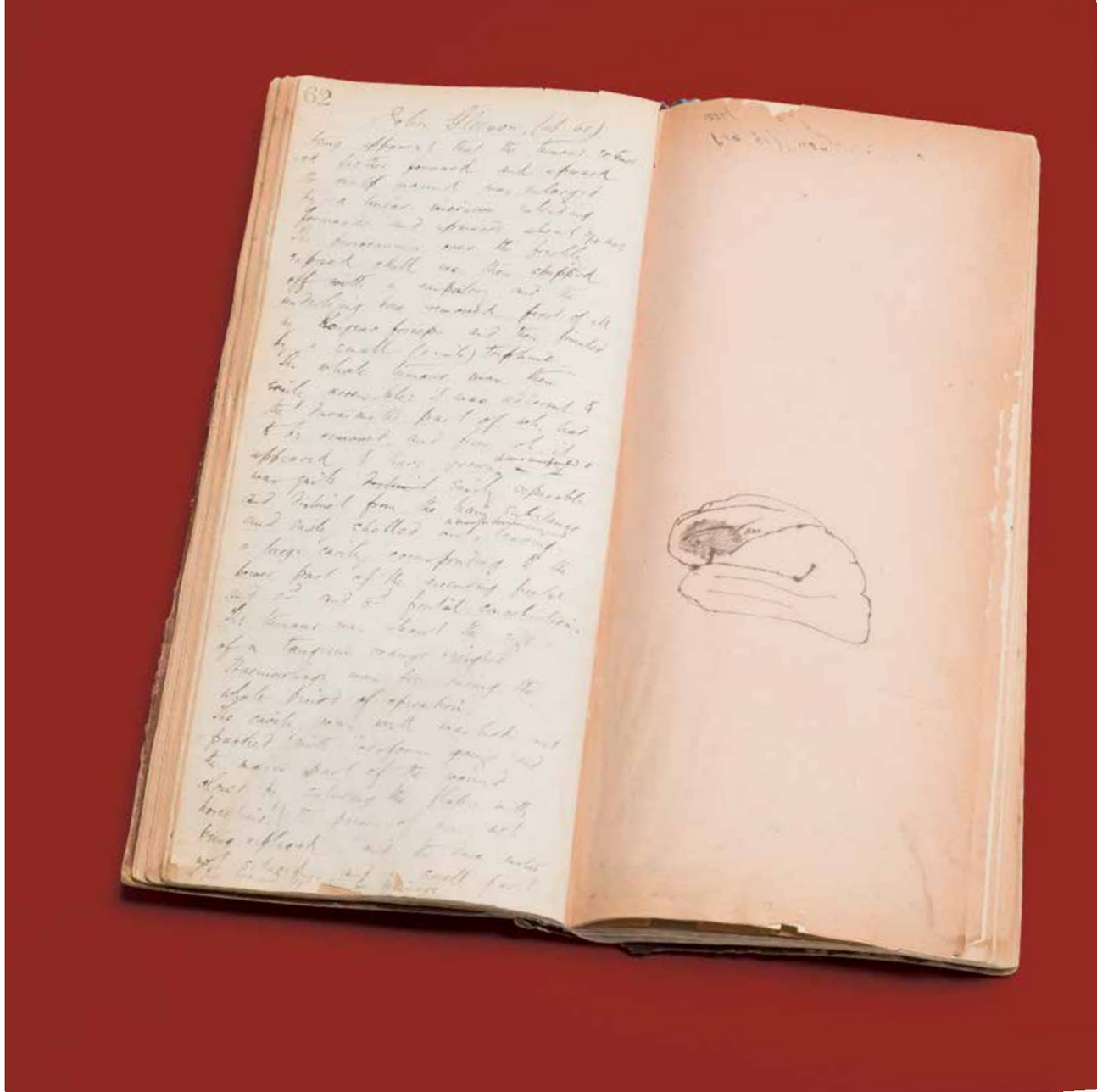
‘A twist in the tale’ of a surgeon and his patient: An Australian first in seizure localisation

In 1894 at St Vincent’s Hospital in Melbourne, Dr George Adlington Syme removed a meningioma from a patient with symptomatic focal epilepsy. The operation stands as the first surgery based on seizure localisation in Australia. It is also the country’s first documented successful resection of an intracranial meningioma. It followed by 18 years William Macewan’s landmark cerebral localisation case on the boy ‘John M’ and Victor Horsley’s first epilepsy localisation surgery on John Hughlings Jackson’s patient ‘James B’ by a mere eight years.

The case notes offer an insight into Syme’s surgical prowess in the era before X-ray technology. Syme’s patient, ‘Constable John G’, survived the operation by some 24 years, eventually dying from a gunshot wound to the head in 1917. Newly discovered inquest papers reveal that the coroner’s judgement that his death was accidental completely fails to address the more credible scenario of suicide. The suspicious death of an upstanding member of the police constabulary, whose history was ‘tainted’ by the ‘falling sickness’, may have been seen as too awkward a problem to tackle with any kind of forensic rigour. The story, with its newly discovered final twist, makes for an intriguing epilogue to an important piece of Australian neurosurgical history.

Dr Christopher Plummer

References:
This is an abbreviated version of an article published as C Plummer *et al*., ‘A twist in the tale of a surgeon and his patient: An Australian first in seizure localization’, *Journal of the History of the Neurosciences*, vol. 17, issue 1, 2008, pp. 33–45.



Cat. 125 Sketch by Dr Syme’s resident, Stanley Docker Read, showing the position and size of the lesion at the left frontoparietal convexity. The letters *RSC* presumably indicate the line of the Rolandic Sulcus. **Casebook of George Adlington Syme**, 1893–96, leather-bound register (opened at p. 62), 31.7 cm × 14.0 cm. St Vincent’s Hospital Archives and Heritage Centre.

Merric Boyd: A creative life lived

Merric Boyd (1888–1959) spent virtually all of his adult life in the Melbourne suburb of Murrumbeena. Arriving there in 1913 as a fit and energetic 25-year-old and establishing a studio residence he called Open Country, he died 46 years later in the same home. In between those events, he built a pottery studio, married artist and poet Doris Gough, with whom he had five creative and artistic children, and produced some of the finest and most original pottery made in this country or any other. He survived service in World War I and an explosion and fire in 1926 that destroyed his pottery, supported his family through the Great Depression, and despite being affected by epilepsy, which affected his physical and mental health, continued to be highly creative until the end of his life.

While Boyd may or may not have had seizures before the loss of his pottery, he was certainly subject to them afterwards. These progressively worsened as he aged. His ability to remain creative was in large measure due to the love of Doris—his 'rock', upon whom both he and their children relied for her strength and devotion. He was also supported by his doctor, John Springthorpe, who came to live in Murrumbeena five years before Boyd (to whom he was related). Merric's parents had bought the property for their son and while they may not have been aware he had epilepsy before then, they had enough concern about his health to believe he might require special care from a trusted doctor. John Springthorpe was that doctor.

Merric Boyd's ability to be creative, even as his health was increasingly affected by his condition, is one of the more significant and dynamic aspects of his life. He never lost the desire to be expressive. The drawings he did prolifically in his latter years are remarkable in their diversity. Images of rural landscapes recalling his days as a jackaroo, coastal scenes of Port Phillip and Westernport bays, trees wild and windswept, portraits of friends and family, all display his interest in his world and his sensitive and sympathetic portrayals of it. His creativity never dimmed or faded.

There is no better insight into Merric Boyd than through his art. He has left a rich legacy for us all to enjoy.

Colin Smith

Cat. 48 Merric Boyd, ***Tree***, n.d., pencil on paper, 25.5 × 20.0 cm. 95-0588-001-01, Bundanon Trust Collection.

St Vincent's Hospital's first electroencephalograph machine

I was a medical student in Melbourne when St Vincent's Hospital's first electroencephalograph (EEG) machine arrived in the 1950s. I was not involved in neurology in a significant way until my final year of studies in 1960, and that was only with clinical cases. The EEG machine was, I think, made in South Australia to an original design that was not very good; but because there was an Australian manufacturer a licence to import an EEG machine could not be obtained—the local industry had to be protected. Attempts to circumvent this problem failed, until the Australian manufacturer ceased trading.

I was much more impressed by the 1936 Grass EEG machine I used in the United States: lots of valves, tons of heat, and a good chance of breaking down any time it was used. That original St Vincent's EEG shared some of these features. There was an interim EEG machine between the 1960s and early 1970s when I returned, but we were able to acquire the all-transistor 16-channel model to replace the older 8-channel EEG. The more I try to look back to the early days, the more I realise I am remembering the setup at the Boston City, the Gibbs old lab, and the Boston Veterans' Affairs and not St Vincent's. As far as I know, all the old machines have gone, though some parts were used for other projects in the hospital. I cannot recall any of the instruction manuals, suggestions for maintenance, or even the valve requirements, and certainly not the manufacturer's name.

The EEG reports were prepared by a triumvirate: the head of neurology, John Billings; the psychiatrist, Eric Seal; and the neurosurgen, Keith Henderson. The neurological world was very different back then, and psychiatrists, neurosurgeons and neurologists were all very involved in the still relatively new science of EEG. The expectations for the general clinical utility of the EEG were unrealistically high, and were thought to include behavioural neurology. Many of the early texts on epilepsy were written by psychiatrists, which was the result of the American practice of combining psychiatry and neurology as a single specialty. The separation came in the 1940s and thereafter psychiatric involvement declined, and the management of epilepsy became a neurological skill.

At its introduction, the EEG was a simple procedure; the technological developments that subsequently appeared owed much to the development of the computer and its application to the clinical setting. The reports issued at that time were often vague, which avoided the problem of 'over-reading', that is, reaching conclusions unwarranted by the evidence, a practice unfortunately common at the time. These EEGs had variable usefulness, and possessed few of the benefits we presently have.

Dr E Bruce Tomlinson

Cat. 126 **EEG machine in use**, 1955, photograph, 18.5 × 24.7 cm. St Vincent's Hospital Archives and Heritage Centre, Clinical Photography Department Collection.

Early brain scans in Melbourne

In 1975 the Royal Melbourne Hospital obtained its first computed tomography (CT) images of the brain, using a then state-of-the-art EMI scanner. This was the first CT scanner in the eastern states of Australia and was purchased at a cost of $320 000. Scans took 30 minutes to obtain, whereas today's scanners take less than a minute, and were by today's standards of coarse resolution. The scanner printed computerised images of the brain onto paper. Radiologists at the time regarded the images as a spectacular advance in radiology, and one of the greatest advances since the discovery of X-rays in 1895. For epilepsy patients this was the best way to diagnose tumours, infections and vascular malformations that are some of the causes of epilepsy.

In 1985 further progress was made when the Royal Melbourne Hospital became one of the first sites in Australia to purchase and install a magnetic resonance imaging (MRI) scanner. This was a 0.3 Tesla Fonar scanner and represented another major leap forward in imaging the brain. As with the early CT images, the initial MRI images from 1985 would now be considered substandard, but at the time they provided previously unseen detail of the brain. For the first time, clear definition of grey and white matter was possible and images could be obtained in the sagittal (vertical from front to back) and coronal (vertical from left to right) as well as the axial (horizontal) plane. With MRI it was possible to see the hippocampus, and a common cause of temporal lobe epilepsy, mesial temporal sclerosis, could now be diagnosed on the basis of imaging. The improved imaging with magnetic resonance meant that cavernomas (clusters of abnormal blood vessels found mainly in the brain and spinal cord), and congenital abnormalities of grey and white matter responsible for seizures could be seen and diagnosed.

All patients with seizures now undergo an MRI scan as part of their diagnostic process. Imaging is an essential part of trying to locate a source for the seizure and it also acts as a guide to choosing the most appropriate therapy.

Professor Patricia Desmond

Cat. 127 **CT machine**, 1976, photograph, 15.5 × 20.5 cm. St Vincent's Hospital Archives and Heritage Centre, Clinical Photography Department Collection.

A home of their own

We all expect to have a say in our medical care. For people with epilepsy this was a right that was a long time coming.

The start of the 20th century saw Australia's only farm colony, the Talbot Colony for Epileptics, come into being. Although outdated by today's values, at the time it confirmed that Victoria was at the forefront of epilepsy management in Australia. The concept of 'A home of their own' for Victorian people who had epilepsy was born in Dr John Springthorpe's speech to the National Council of Women in 1903 when, in answer to the question of the purpose of the colony, he decreed 'the object of establishing a Colony here was not so much for cure as to provide a home for epileptics …'[1]

The Victorian Bureau for Epilepsy was formed in May 1964 after the demise of the Talbot Colony. (The site is now occupied by Monash University.) The families involved believed that the medical profession and the state government had been instrumental in their exclusion from the new Royal Talbot facility at Kew. On the back of that exclusion, one of the key tenets of the new Bureau established in 1964 was that funds would be raised to purchase a new permanent home. Once achieved, the Bureau would then exist so that people living with epilepsy would always have a home, no matter what.

After renting an office in the old Gas and Fuel Corporation premises, the Bureau moved to a terrace house in East Melbourne where it also provided accommodation for girls from regional areas. After a short stint in the city, the Bureau purchased a Camberwell property; this became 'that home' in 1975. In 2012, after a six-year search, a new home was bought in Canterbury Road, Surrey Hills.

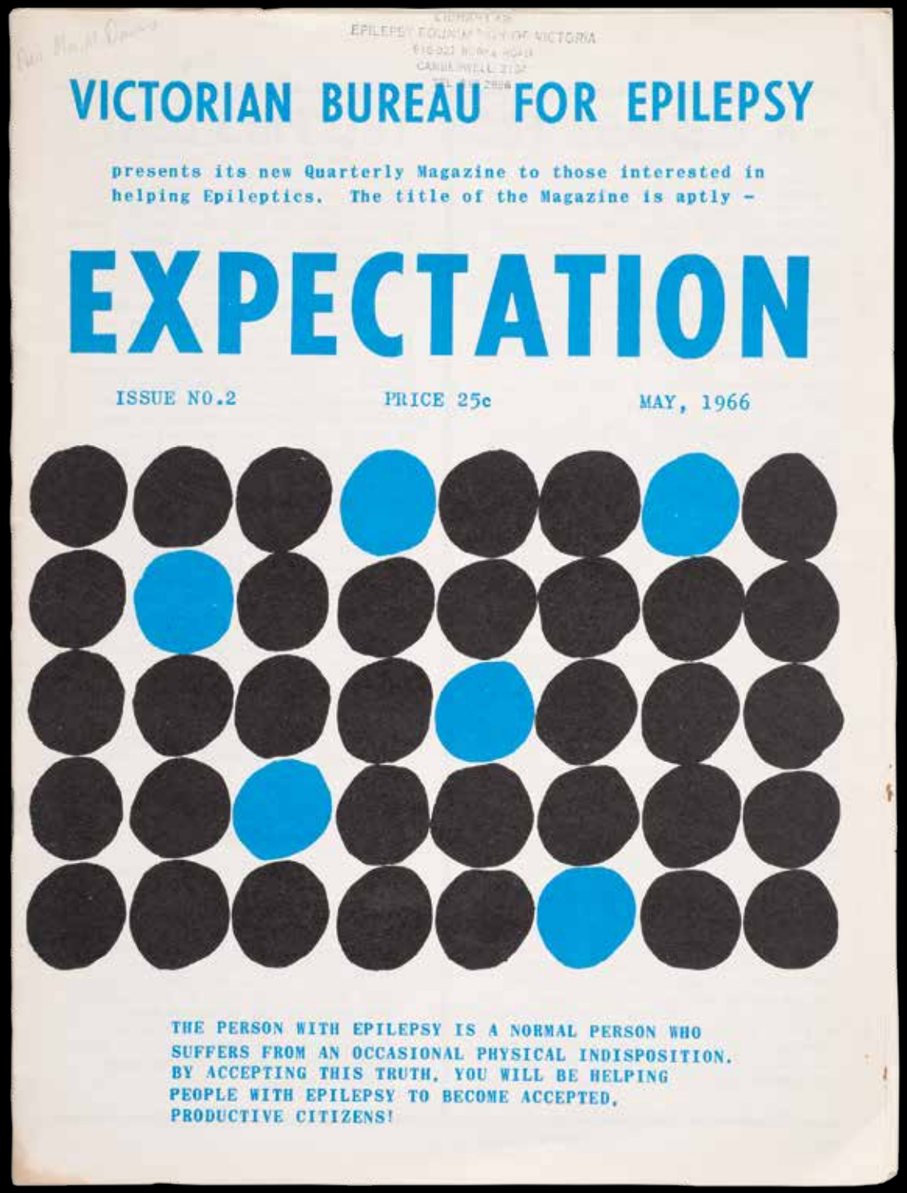
In 1990 the Victorian Bureau for Epilepsy changed its name to the Epilepsy Foundation of Victoria, to reflect the broadening of its work to include psychosocial research. It was the first of the consumer-based epilepsy organisations to provide a range of services including individual support, advocacy, education and public information programs.

Today, the Epilepsy Foundation is the largest member of a network of independent epilepsy organisations that cooperate as part of the Joint Epilepsy Council of Australia, which is Australia's member of the International Bureau for Epilepsy.

Jeremy Maxwell

1 J Springthorpe, 1903, quoted in PF Bladin, *A century of prejudice and progress: A paradigm of epilepsy in a developing society: Medical and social aspects, Victoria, Australia, 1835–1950*, Melbourne: Epilepsy Australia, 2001, p. 119.

Cat. 75 Victorian Bureau for Epilepsy, ***Expectation***, quarterly magazine: issue no. 2, May 1966, print on paper, 30.5 × 23.0cm. Epilepsy Foundation Collection.



Epilepsy, discrimination and legal restrictions

I have two friends who are both severely disabled, one through multiple sclerosis and the other who describes herself as a thalidomide survivor. Both are in wheelchairs and, although surprisingly independent, rely on the care of others. Each has received a mainstream education, has been employed, undertaken extensive voluntary community work, has a partner and, in one case, children.

Why then, one must wonder, is it that many people with epilepsy who are no more physically and intellectually restricted than my friends, find that they are not able to complete an education, find employment, get married or even undertake voluntary work? While these are my observations, research demonstrates that people with epilepsy constantly underperform in these areas. The effects of medication or seizure activity play a role for some people, but do not explain why it is the case for people with well-controlled epilepsy. Yet research confirms that three times more Australians with self-reported epilepsy are likely to be unemployed than those who do not have epilepsy;[1] that educational attainment is lower in this group than in the overall Australia population;[2] that more than half of those with epilepsy who are in employment fail to earn the minimum wage;[3] and, regardless of culture, research consistently demonstrates that all people with epilepsy are less likely to marry and more likely to have failed marriages.[4]

Discrimination explains much of this and, like all Australians, people with epilepsy have recourse to redress through the anti-discrimination and equal opportunity legislation. But people with epilepsy rarely report discrimination because under these Acts they must personally make a complaint, making their humiliation and diagnosis public. The legislation thus creates restrictions for those very people it should most assist.

Perhaps the greatest legal restriction relates to driving. The regulations in this area are inconsistently administered and medical practitioners are the instruments of their administration, which puts them in difficult relationships with their patients.[5] Being desperate to work or to be seen as 'normal', many people choose not to disclose their epilepsy to the wider world.

Truly the law has a long way to go to remove the discrimination that is endemic in the diagnosis itself.

Dr Christine Walker

1 K Brown, 'Indicators of social consequences of epilepsy', in J Pinikahana and C Walker (eds), *Society, behaviour and epilepsy*, New York: Nova Biomedical, 2011, pp. 17–28.
2 Epilepsy Foundation, *'Out of the shadows': Needs, perceptions and experiences of people living with epilepsy in Australia: Findings from Wave 2 of the Longitudinal Survey*, 2012.
3 *Ibid*.
4 L Andermann, 'From public to personal: A social view of epilepsy', in Pinikahana and Walker (eds), *Society, behaviour and epilepsy*, pp. 29–43.
5 R Beran, 'Epilepsy and law', *Epilepsy and Behavior*, vol. 12, 2008, pp. 644–51.

Cat. 74 ***Grapevine***, newsletter of Epilepsy Group, October 1988, print on paper, 30.0 × 21.0 cm. Epilepsy Foundation Collection.

GRAPEVINE Oct 88

EPILEPSY GROUPS NEWSLETTER

Patients lose drug ruling

The Sun, Thursday, August 18, 1988

UPDATE ON EPILIM

Yes folks, in spite of representatives from Epilepsy Self Help Groups, the Epilepsy Foundation of Victoria, the National Epilepsy Association of Australia and a number of eminent neurologists, the Federal Government has placed epilim on the "Authority Only" listing. This was effective from August 1st. One concession made was that in emergency situations, doctors can prescribe epilim and seek authority after the prescription has been filled. There is also an emergency out of hours telephone approval system. The Victorian after hours number is (03) 3470103 (doctor's use only).

This "concession" in theory should remove the problem of people running out of epilim and not being able to get a new script immediately. The alternative would have been sudden withdrawal of epilim possibly precipitating status epilepticus.

However, the other problems associated with the "Authority only" listing remain. Grapevine understands that there are discussions taking place and epilim may get come off the Authority Only list. We will keep you posted.

It is very interesting to note that the Health Department was considering removing tegretol from the Free Listing of Medications to be replaced by a generic drug. The secretary of the Pricing Authority, however, has indicated that this will not happen. They seem to have changed their minds. Grapevine wonders if the complaints regarding epilim were foremost in their minds.

Ed.

THE Community Services and Health Department's decision to place 33 drugs on the Authority Required list is a retrograde step that will seriously disadvantage some of the most vulnerable people in the community.

The anti-convulsant drug Epilim (sodium valproate) is used by 30 to 40 per cent of the 360,000-plus Australians who have epilepsy.

While many hesitate to use the term "wonder drug", Epilim has made it possible for many thousands of disabled people to live a completely normal life.

The older type of medication had the potential to cause serious side-effects in learning ability and permanent liver damage, while behavioral and cosmetic changes were experienced by some persons who were, of necessity, forced to use the "older generation" of drugs over a period of years.

We sympathise with the Government's need to reduce costs in every area.

But we must weigh the cost of the effective medication against the increased cost of invalid pensions and unemployment benefits for those unable to work due to an inability to receive the 100 per cent protection from the cheaper medication available.

Doctors have immense amounts of paperwork now and the necessity to obtain permission to prescribe Epilim will increase the load to an extent.

Many doctors may take the easy way out and prescribe the older, now more readily available but less satisfactory and cheaper type of anti-convulsant.

The families, support groups, teachers, friends and employers ask the department to reverse this decision to restrict the use of an excellent drug, so the people affected by epilepsy may lead normal lives, keep their financial independence, self respect and health.

— Evanna Lake
Sale Epilepsy Support Group (Sale).

DRIVING AND EPILEPSY

EDITORIAL - PICTURE

The issue of Driving and Epilepsy is still unresolved and at the moment it is "Each case will be judged on its own merits". This phrase is as original as the car above.

Happy Birthday to you. Happy Birthday to you.
For more details see page 6
"Oh how could you forget".

Deaths from epilepsy: SUDEP

Sudden death in epilepsy is a long-recognised phenomenon. In 1868 an article in *The Lancet* written by G Mackenzie Bacon, based on his experience as medical superintendent of the Cambridge County Asylum, categorised deaths due to epilepsy itself as arising from 'sudden deaths in fit', 'deaths after a rapid succession of fits' and 'deaths from accidents'.[1] This early interest by a medical practitioner perhaps occurred because people with epilepsy were institutionalised in asylums, which allowed members of the medical profession to witness when a person with epilepsy died.

Despite these observations, over the following century there was a widely held belief that epilepsy itself could not be fatal. This sometimes amounted to complete denial of the possibility of death from epilepsy and has meant that research into the causes of death in epilepsy has been largely neglected.

The term SUDEP was first coined to describe this phenomenon by Dr Lina Nashef and Dr Stephen Brown in 1996 at the first International Workshop on Epilepsy and Sudden Death, held in London. This definition was later published as:

> the sudden, unexpected, witnessed or unwitnessed, nontraumatic and nondrowning death in patients with epilepsy, with or without evidence for a seizure and excluding documented status epilepticus, in which postmortem examination does not reveal a toxicologic or anatomic cause for death.[2]

Critical to this definition is the term 'unexpected' rather than 'unexplained', as it is likely that the major cause of SUDEP may be explained as being the consequence(s) of a seizure. In addition, despite post-mortem examination being central to identification, pathologists' use of the term SUDEP in valid cases still remains too low. These ambiguities probably lead to under-recognition and under-identification of SUDEP as a post-mortem diagnosis.

The first steps in understanding and preventing SUDEP remain consistency of recording epilepsy-related deaths on death certificates, coding of cause of death by central statistics offices, reporting of deaths to the coroner, actions of the coroner in deciding further examinations, completeness of post-mortem investigations, access to these sources of information by those retrospectively researching SUDEP, and the availability of prospective data-capture systems. To quote Bacon again: 'If practitioners would adopt some such system … we would not have to lament such a meaningless blank as the word now represents in lists of mortality'.

Associate Professor Wendyl D'Souza

1 G Mackenzie Bacon, 'On the modes of death in epilepsy', *Lancet,* vol. 91, no. 2331, 2 May 1868, pp. 555–6.
2 L Nashef, 'Sudden unexpected death in epilepsy: Terminology and definitions', *Epilepsia*, vol. 38, supplement 11, 1997, pp. S6–S8.

Epilepsy Foundation, **Seventh memorial service in remembrance of those who have died with epilepsy**, 2014, digital photograph. Epilepsy Foundation Collection.

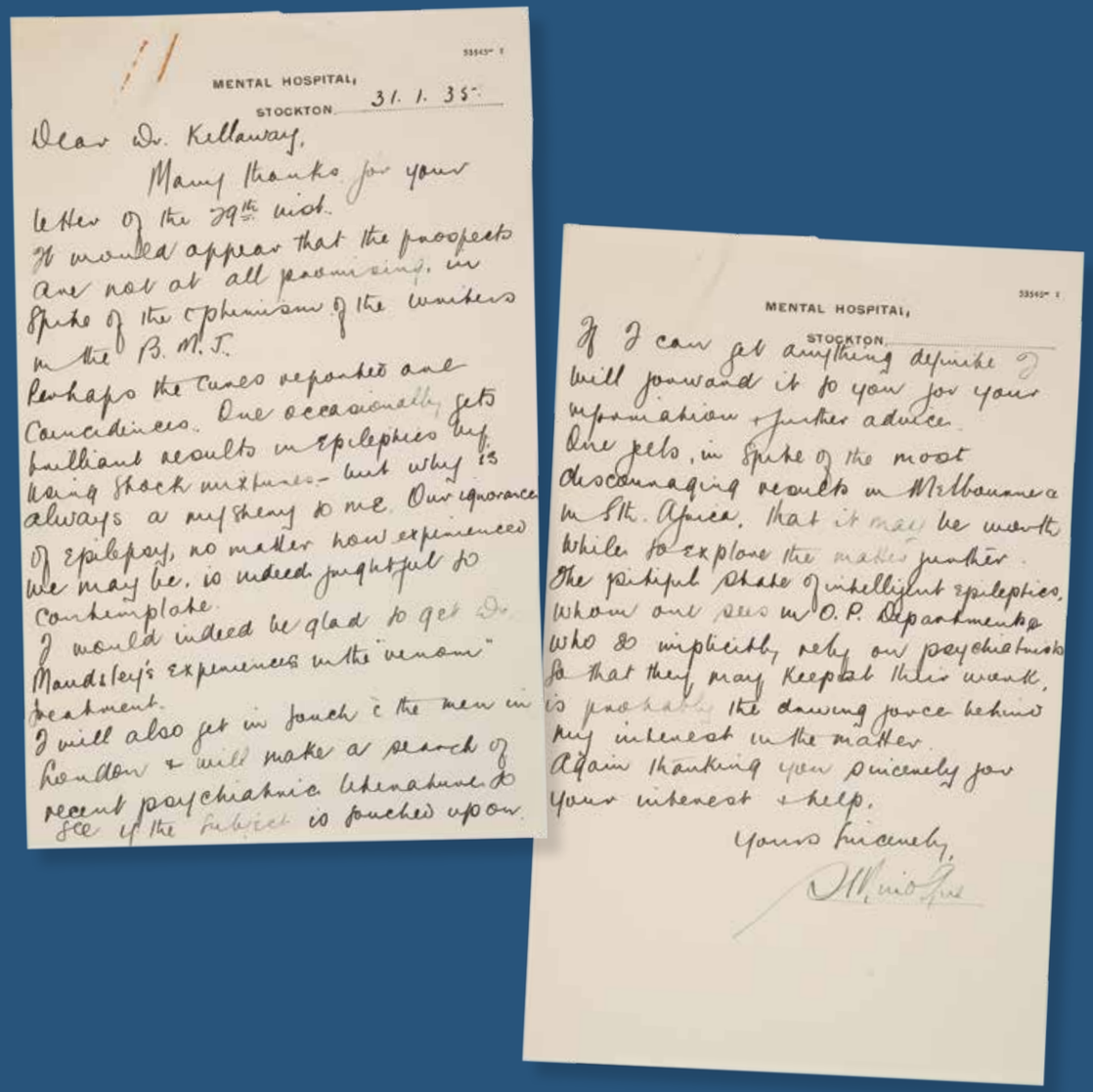
Poisonous optimism: Snake venoms for epilepsy

As is true for many medicines, the origins of treating epilepsy with snake venom were anecdotal. In 1908, Philadelphia physician Ralph Spangler heard of a Texan with epilepsy who became seizure-free after being bitten by a moccasin viper. Theorising that 'crotalin' (dried rattlesnake venom) might interfere with coagulation and thus reduce blood clots—which were believed to provoke seizures in individuals with susceptible nervous systems—Spengler injected it into more than 100 patients.[1] Thanks to claims that crotalin therapy reduced seizures and improved general and mental health, his approach gained favour with many American doctors. By 1914 however, crotalin was largely dismissed as merely proof of 'the abnormal craving for medicine which all epileptics have'.[2]

But in South Africa, the director of the Port Elizabeth Museum and Snake Park, Frederick FitzSimons, convinced the Pretoria Mental Hospital to trial a purified mixture of local venoms for epilepsy in 1919, and again in 1929.[3] Injecting 'venene' merely made these patients irritable, yet FitzSimons attracted a loyal following among private practitioners. Treatment required increasing doses of venene over nine months, but its mechanism remained unclear: was its effect primarily haematological, immunological, neurological or psychological? FitzSimons remained evasive, simply suggesting that venoms were 'powerful anti-spasmodic and anti-convulsive agents'.[4]

By 1930, FitzSimons began campaigning to convince Australian and New Zealand health authorities to adopt his expensive treatment for epilepsy. Bolstered by several British experts familiar with Indian serpents,[5] this approach coincided with experimental use of Indian cobra and Australian black snake venoms as neurotoxins to treat cancer pain.[6] In 1934–35, all lines of inquiry led to Charles Kellaway, director of the Walter and Eliza Hall Institute and Australia's acknowledged venom expert.[7] His friend, neurologist Henry Maudsley, had treated epilepsy cases with purified tiger snake venom, but the results were evidently unfavourable. Furthermore, the Commonwealth Serum Laboratories were reluctant to incur the costs and dangers of supplying venoms for clinical use.[8] Despite these 'most discouraging results in Melbourne & S[outh] Africa', compassionate Newcastle psychiatrist Sylvester Minogue confessed that the 'pitiful state of intelligent epileptics' led him to hold out hope.[9] Kellaway, however, expressed little optimism, and the venom treatment for epilepsy soon slithered from favour.

Dr Peter Hobbins


MENTAL HOSPITAL,
STOCKTON. 31. 1. 35.

Dear Dr. Kellaway,

Many thanks for your letter of the 29th inst.

It would appear that the prospects are not at all promising, in spite of the optimism of the writers in the B. M. J.

Perhaps the cures reported are Coincidences. One occasionally gets brilliant results in epilepsies by using shock mixtures - but why is always a mystery to me. Our ignorance of epilepsy, no matter how experienced we may be, is indeed frightful to contemplate.

I would indeed be glad to get Dr. Maudsley's experiences in the "venom" treatment.

I will also get in touch with men in London & will make a search of recent psychiatric literature to see if the subject is touched upon.

MENTAL HOSPITAL,
STOCKTON.

If I can get anything definite I will forward it to you for your information & further advice.

One gets, in spite of the most discouraging results in Melbourne & in Sth. Africa, that it may be worth while to explore the matter further. The pitiful state of intelligent epileptics, whom one sees in O.P. Departments who so implicitly rely on psychiatrists so that they may keep at their work, is probably the driving force behind my interest in the matter.

Again thanking you sincerely for your interest & help.

Yours sincerely,

Cat. 132 SH Minogue (Medical Superintendent, Mental Hospital, Stockton), **Letter to Dr Kellaway** (Director, Walter and Eliza Hall Institute), 31 January 1935, ink on paper, 20.0 × 13.0 cm (two pages). Walter and Eliza Hall Institute Archives.

1 RH Spangler, 'The crotalin treatment of epilepsy. Chemical composition of snake venom. Its possible effect on blood coagulation. Report of eight cases', *Epilepsia*, A4, no. 3, June 1913, pp. 315–18. See also J Turner, 'Further observations bearing on the supposed thrombotic origin of epileptic fits', *Journal of Mental Science*, vol. 54, no. 227, October 1908, p. 639.
2 CL Jenkins and AS Pendleton, 'Crotalin in epilepsy', *Journal of the American Medical Association*, vol. 63, no. 20, 1914, p. 1750; Laurence M Klauber, *Rattlesnakes: Their habits, life histories, and influence on mankind* (2nd edn), Berkeley: University of California Press, 1997, p. 166.
3 JL Dunston to TG Gray, 30 June 1930 (copy). WEHA00049, 1929–34, Snakes (2), series 1, box 1. Walter and Eliza Hall Institute of Medical Research Archives (hereafter WEHA).
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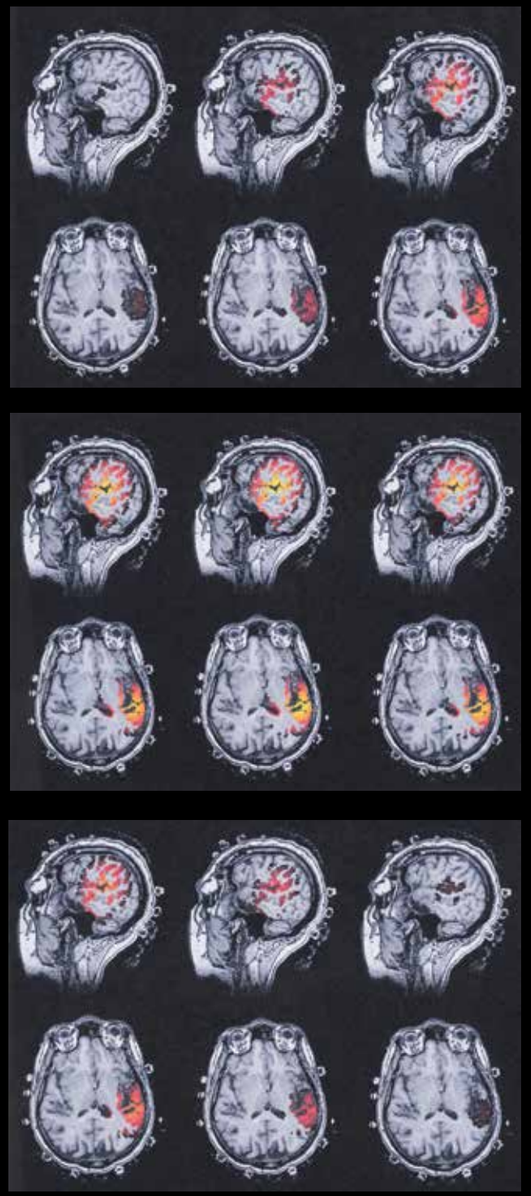
5 RH Elliot, 'Treatment of epilepsy by snake venom', *British Medical Journal*, vol. 2, no. 3857, 8 December 1934, p. 1073.
6 CH Kellaway to JH Cumpston, 9 August 1934. National Health and Medical Research. Walter and Eliza Hall Institute—application for subsidy, 1927–34. Series A1928/1, control 690/20, section 1, National Archives of Australia (hereafter NAA).
7 PG Hobbins, 'Serpentine science: Charles Kellaway and the fluctuating fortunes of venom research in interwar Australia', *Historical Records of Australian Science*, vol. 21, no. 1, 2010, pp. 4–16.
8 FG Morgan to JHL Cumpston, 4 October 1934. NAA A1928/1, control 690/20, section 1. See also P Hobbins, ' "Immunisation is as popular as a death adder": The Bundaberg tragedy and the politics of medical science in interwar Australia', *Social History of Medicine*, vol. 24, no. 2, August 2011, pp. 428–39.
9 SJ Minogue to CH Kellaway, 31 January 1935. WEHA00049.

Cat. 7 Emma Brockett (Australia), ***Linear confusion***, 2003 (detail, colour altered), mixed media on paper, 101.0 × 63.0 cm. Collection of the artist. See p. 115.

CREATIVE SPARKS: ART FROM PEOPLE WITH EPILEPSY



Insights into epilepsy and creativity through visual art

The exhibition *Epilepsy: Perception, imagination and change* offers a rare glimpse into how epilepsy has influenced the works and lives of artists who participated in an in-depth study of the influences of epilepsy on creative art. They share their intimate experiences and insights into the condition, along with their artworks, with the intention that people with epilepsy will be better understood, respected, appreciated, and accepted in society.

Over the centuries, people with epilepsy have been saddled with false perceptions of being possessed by demons, engaging in witchcraft, or being mentally ill. These misguided views led to people's institutionalisation, persecution and sometimes death.[1] The rights of people with epilepsy to procreate and to live among society were also violated, in part because of misguided beliefs in eugenics,[2] a philosophy advocating the improvement of human genetic traits through promoting higher reproduction among people with desired traits and discouraging reproduction among people with less-desired traits.[3]

On 14 July 1933 the German government instituted the 'Law for prevention of progeny with heredity diseases', which specifically included 'hereditary epilepsy'.[4] As a result, many people with epilepsy were castrated or prevented from bearing children. In 1939 Hitler began euthanising people with epilepsy, disability, mental illness and other conditions that fatally labelled a person as 'unfit'.[5]

Hitler and Nazi propaganda also claimed that some forms of art, which they labelled as 'degenerate', were signs of possible genetic defect or madness in the artist. Use of the term 'degeneracy' in art came from the author Max Nordau (1849–1923), who advocated in his book *Entartung* (Degeneration, 1892), that modern art styles or movements, such as Surrealism, Impressionism, Cubism and Abstraction, were products of a diseased visual cortex.[6] The Nazis defined 'degenerate art' as that which would 'insult German feeling, or destroy or confuse natural form or simply reveal an absence of adequate manual and artistic skill'.[7] Some artists who produced 'degenerate art' were dispossessed of their works or forbidden to paint.[8] In a speech delivered in the framework of the Day of German Art in Munich in 1938, Hitler declared that artists were forbidden to represent anything but forms seen in nature, under threat of ending up either in an asylum or before the courts.[9]

As the Nazi list of degenerate artists expanded to include non-Germans, the names and works of three notable artists, who most likely had epilepsy, were added:[10] Vincent van Gogh (Netherlands),[11] Odilon Redon (France),[12] and Giorgio de Chirico (Greece/Italy).[13] Much of their so-called degenerate art was also confiscated for destruction or for sale at undervalued prices, for the personal gain of corrupt Nazi authorities or to help finance the war.[14]

Fig. 1: Images of changes in cortical activity in interictal discharge for Mr Howard Smith (2010). Images produced by Simon Vogrin and formatted by Jim Chambliss, Melbourne Medical School and St Vincent's Hospital.

Portrait of Dr Gachet (1890) by Vincent van Gogh was one of the confiscated works designated as degenerate. Nazi leader Hermann Göring realised the painting had some monetary value and sold it, while not appreciating its true artistic value. Siegfried Kramarsky bought it from an intermediary for $53 000 before he fled to the United States. In 1990, *Portrait of Dr Gachet* surpassed the record for the highest price paid for an artwork sold at auction when purchased by Japanese paper magnate Ryoei Saito for US$82.5 million.[15] The current location of the painting is unknown.[16]

Portrait of Dr Gachet, which went from being unacceptable for sale during the lives of Vincent and Theo van Gogh to being the most expensive artwork sold at auction, serves as an example of how art from a person who likely had epilepsy was undervalued.

Context of the art exhibit

The exhibition *Epilepsy: Perception, imagination and change*, in conjunction with scholarly research, looks at artists with epilepsy and examines their creative contributions. Firstly, it demonstrates that people with epilepsy or migraine deserve admiration, respect and empathy. Secondly, it evaluates the extent to which art made by people who experience seizures can serve interpretive purposes in medicine, psychology and art history. Thirdly, it offers objective and subjective proof that focal epilepsy can sometimes significantly enhance creativity in visual art.

In 2006, my study of the art and lives of people with epilepsy began at the University of Melbourne through the PhD academic research 'Creative sparks: Epilepsy and enhanced creativity in visual arts' (2014), supervised by Professor Mark Cook and Associate Professor Barbara Bolt.[17] More than a hundred artists from around the world, who produced their work as a hobby or profession, participated in the study. Most of them provided images of their works for evaluation, performed creativity tests, and provided extensive personal information through surveys and interviews.

The works in the current exhibition were assembled as part of the 'Creative sparks: Art and epilepsy' project, which produced the largest ever collection of digital images of works by artists with epilepsy. Many of the more than 100 works can be viewed at www.artandepilepsy.com. Each of the artists whose work features in this book participated fully in the research, which validates the artists' actual experiences with epilepsy and supports the interpretation of the artworks.

This exhibition at the Medical History Museum is shaped by what is essentially a visual and verbal dialogue between the artists who have experienced seizures and the audiences, who mostly consist of people learning about medicine, psychology, art history and creativity theory. Many, if not all, of the artists represented in this exhibit and associated educational programs have thought, 'I'd like to give my doctor a piece of my mind, so that he or she

Fig. 2: Cat. 42, 43 and 44: Howard Smith (Australia), **Experimental drawings 1, 2** and **3**, 2010, pen on paper, each drawing 30.0 × 21.0 cm. Courtesy the artist, Melbourne Medical School and St Vincent's Hospital.

better understands what I am thinking, feeling and experiencing'. If a picture indeed speaks a thousand words, these artistic expressions say much that cannot be covered in a doctor–patient interview or written text.

Most of the artworks in this exhibition depict influences from a wide range of pseudo-hallucinations, illusions and unusual experiential manifestations sparked by epilepsy. During personal discussions about these unusual phenomena, most artists said something to the effect of 'I would not tell this to my doctor!' or 'I have not shared this information with anyone until now'.

One legacy of the 'degenerate art' label associated with the development of German fascism and the horrific culmination of that enterprise was the denunciation of psychopathological interpretations of art. For decades, art historians excluded psychiatric or psychodynamic theories from art analysis, because of fear of refuelling a potential condemnation of contemporary artists.[18]

This exhibition has gone through an extensive process of ethical review in order to display the artworks in a way that balances the need to know with a genuine respect for the unique artists. It does not proliferate stereotypes, clichés and stigma, but has the opposite effect: it demystifies epilepsy, contests reductive stereotypes and provides real, evidence-based explanations for the unusual transient phenomena that epilepsy can generate. Some of the steps taken to achieve this ethical recognition, display, discussion and appraisal of the participants' art—consistent with recommendations in *Framing marginalised art*—are as follows:

- Artists describe their artworks in their own words.
- Artists receive named acknowledgement for their art and written insights.
- The manner in which the works are displayed and discussed was approved by the artists.
- I, as the main lecturer in the associated educational programs, am also an exhibiting artist with epilepsy. As such, based on similar personal experiences, I understand most of the sensitivities of the participants.

Epilepsy and intrinsic perceptions

An epileptic seizure is a transient occurrence of signs and/or symptoms caused by abnormal excessive or synchronous neuronal activity in the brain. Epilepsy is a disorder of the brain characterised by an enduring predisposition to generate epileptic seizures, and by the neurobiological, cognitive, psychological and social consequences of this condition. A diagnosis of epilepsy requires the person to have had at least one epileptic seizure.[19]

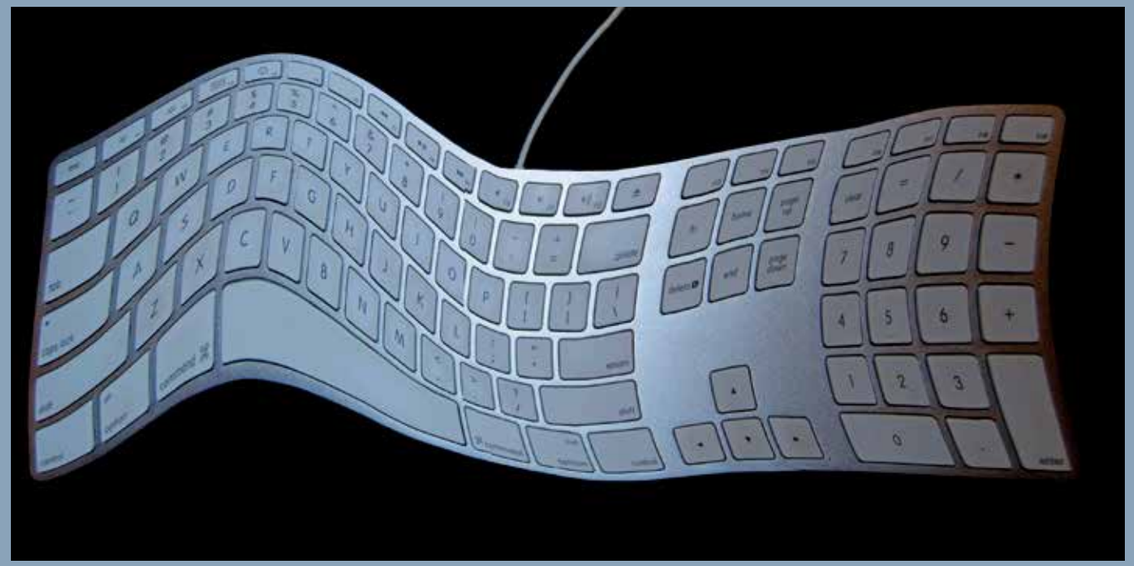


Fig. 3: Jude Rouslin (USA), ***Keyboard in a visual seizure***, 2014, digital image.

To understand how epilepsy can influence art requires one to acknowledge that the cycle of epilepsy changes like weather patterns.[20] The influences of epilepsy on cognition are not limited to a seizure (ictal event) or the relatively brief pre-ictal and post-ictal stages immediately before and after a seizure. The electrical mischief of epilepsy can cause a prodome (early symptoms) of epileptic discharges that can build up for hours or continue in a clustering of seizures,[21] or interictal epileptic discharges that can exhibit no visual signs of, or connections with, seizure activity—even to a trained observer or the individuals themselves.[22]

In the 1970s Norman Geschwind noted that the behaviour of some people with temporal lobe epilepsy (or focal epilepsy) shared 'a tendency to increased and extensive writing (or drawing) often of a cosmic or philosophical nature', even when they are not having a seizure.[23] The more prominent behavioural changes are hypergraphia, deepening of emotions, preoccupation with religious or mystical concerns, viscosity (stickiness or extreme talkativeness) and hyposexuality (reduced sexual drive).[24] Hypergraphia is a tendency to extensive, and in some cases compulsive, writing,[25] drawing[26] and painting.[27]

The phenomena of interictal behavioural changes led to a prominent theory of how epilepsy could enhance creative potential, developed by researchers such as Eve LaPlante, Oliver Sacks[28] and Julien Bogousslavsky.[29] Until now, little attention has been paid to how intrinsic perceptions like the 'cosmic' phenomena mentioned by Geschwind—not necessarily related to heightened emotions or hypergraphia—can influence visual art.

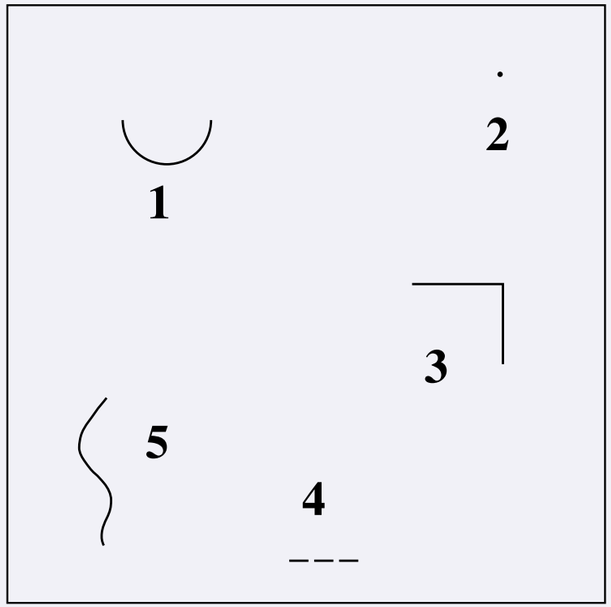
Some of the artists in my research reported that they have clusters over an extended period of time—even days or weeks—when the transient phenomena of epilepsy are more intense and frequent. During this time their desire to produce art becomes so compulsive that they continue working into the later hours of the night. If they push the threshold of fatigue to a point that it triggers an ictal event, they sometimes experience a crash of creative motivation that in some ways deceptively resembles the patterns of bipolar disorder.

One of the artists in our study, Howard Smith, was also involved in a study involving advanced brain imaging of interictal discharges, undertaken by the University of Melbourne and St Vincent's Hospital (Fig. 1).

The brain images in Fig. 1 model a sequence of changes in cortical activity, corresponding to an interictal discharge in Howard Smith's brain. The recording in 2010 showed many such EEG spikes of spreading hypersynchronous electrical activity in Smith's left hemisphere, each lasting less than two seconds. Smith made three drawings (Fig. 2) during the capture of these ongoing epileptic discharges, when there was no observable change in physical movement or outward signs of altered consciousness. The drawings

Fig. 4: K Urban and H Jellen, extract from the Test for Creative Thinking—Drawing Production form, 1996. © Harcourt Assessment Inc. (2005).

Fig. 5: Anonymous participants in family member control group, TCT—DP sample drawings. Administered by Jim Chambliss, Melbourne Medical School and St Vincent's Hospital.



Standard TCT—DP drawing form

Identification of fragments

1	Semi-circle
2	Point
3	Large right-angle
4	Broken line
5	Curved line
6	Small open square

Father's TCT—DP score = 24

Mother's TCT—DP score = 22

Brother's TCT—DP score = 27

responded to requests to draw, firstly, his memory of imagery that came to his mind's eye when recalling previous seizures; secondly, the face of a person he knows; and thirdly, simple geometric figures. He showed a tendency to diverge from drawing realistic, commonly expected images. I propose that there is a unique correlation between this activity of interictal epileptic discharges in some artists with focal epilepsy and a cognitive pull towards creating art that is dreamlike, surreal or extraordinarily imaginative.

In the two decades from 1870 to 1890, John Hughlings Jackson recognised and described focal epilepsy in terms of a 'dreamy state', 'psychic seizures' and 'double consciousness'.[30] The 'double consciousness' refers to a state where a person with focal epilepsy is vaguely aware of their reality-based consciousness, involving ongoing events, while being preoccupied with an invading sense of familiarity, like déjà vu or psychic phenomena.[31] Uncommon images, thoughts and experiences, formed within the 'dreamy state', have for centuries perplexed and fascinated people, contributing to the misconception that those with epilepsy were blessed with mystical, religious or philosophical revelations or—to the contrary—cursed by demons, witchcraft or insanity.

In the study of science, medicine and psychology, such other-worldly imagery and experiences are termed somatosensory or special sensory hallucinations,[32] illusions (cognitive distortions)[33] or experiential manifestations sparked by epilepsy. In this article these phenomena are called 'intrinsic perceptions'. They comprise imagery, visions and thoughts, derived automatically and impulsively in a pre-cognitive stage, from within one's brain and mind, rather than from what is observed in the extrinsic, real and tangible world, or learned from common knowledge and instructions. The term intrinsic perceptions has less negative association with reductive and impersonal medical and psychological pathology.

Intrinsic perceptions can result from a hyperstimulated and hypersynchronous spread of epileptic discharges. Truly original images, experiences and phenomena can be formed because of the complexity of the process whereby thousands (or more likely hundreds of thousands) of neurons meld together in unusual combinations while a person is conscious or in a state of dual consciousness, able to remember and later integrate such rare and novel imagery into visual art.

Intrinsically derived perceptions can be simple or complex hallucinations. Most simple hallucinations do not have significant and reliable interpretive or diagnostic value, when looking at artworks alone. For a person with epilepsy, a wide variety of neurological, visual and environmental conditions can produce simple hallucinations such as seeing spots or flashes of light. In art, imagery similar to simple hallucinations can be purely coincidental. In contrast, complex hallucinations involving surreal, dreamlike or fully integrated imaginary scenes are statistically rare outside the influences of epilepsy, a few other neurological conditions, psychological illness, or responses to substances such as hallucinogenic drugs.

Intrinsic perceptions also transpire when what is seen, felt or experienced is so altered by neurological processes that what would be commonly perceived or understood as 'real' takes on surreal or dreamlike qualities (illusions). Intrinsically altered perceptions include distortions of shape, size, proportion or colour.

Intrinsically experienced perceptions include phenomena such as numinous auras, unique mystical or spiritual qualities,[34] ecstatic seizures,[35] out-of-body experiences, and other experiential manifestations of epilepsy, such as distortion of spatial awareness or surreal events.

Interactive intrinsic perceptions occur as imagery develops during the course of producing visual art. Some artists with epilepsy feel a compulsive, free flow of motivation to create art that is remarkably detailed or elaborative.

More than 90 per cent of the 51 artists with focal epilepsy who completed all aspects of the Creative Sparks research had experienced intrinsic perceptions and integrated such fascinating imagery and experiences into their art.[36] The statistics are reported in the table below in three categories: whether a type of intrinsic perception was experienced; whether artists consciously integrated influences of the specific intrinsic perceptions in their works;

Intrinsic perceptions experienced and integrated into art[37]

Type of intrinsic perception	Actual experiences	Artist reporting use in art	Independent evaluation
Complex intrinsically derived perceptions Entirely imagined people or things Entirely fictional scenes Dreamlike/surreal narrative Demons, ghosts or monsters	73.47%	> 1% = 98.08% > 25% = 78.85%	88.46%
Intrinsically altered perceptions Metamorphopsia (altered shape) Distortion of human form Size distortion Fragmentation Wavy lines Halo or ripple effect Colour intensification or muting	95.83%	> 1% = 94.23% > 25% = 79.00%	75.00%
Intrinsically experienced perceptions Mystical or cosmic experiences Religion/spirituality Vertiginous, extra-sensory perceptions Multiple or divided person Out-of-body experiences Unprovoked fear	95.74%	> 1% = 86.27% > 25% = 52.94%	78.85%
Interactive intrinsic perceptions Automatic drawing, painting or sculpting Incorporation of text within imagery	86.54%	> 1% = 86.54% > 25% = 87.00%	61.54%

and whether an experienced and informed art therapist identified possible influences of intrinsic perceptions in a blind study of 10 artworks from each participant. The artists disclosed the frequency with which they included intrinsic perceptions in their art. The table distinguishes between intrinsic perception being integrated into art once, a few times or more than 25 per cent of the time. Such frequent use of an intrinsic perception indicates that it is highly significant in an artist's personal inventory of images, ideas and life experiences that contribute to their individual style and portfolio of works.

The statistical frequency of intrinsic perceptions in the art of people with epilepsy was assessed by comparison with the artworks from two art exhibitions, not subject to the exclusivity of a jury selection process. Fewer than 3 per cent of the artworks in those exhibitions depicted dreamlike or surreal imagery that could reasonably be inferred as linked to complex hallucinations. Fewer than 4 per cent of the artworks depicted influences of any intrinsically altered perceptions, except for altered colour use, which was seen in fewer than 10 per cent of the artworks. Fewer than 3 per cent of the artworks showed signs of the integration of intrinsically experienced perceptions. Frequency of interactive intrinsic perceptions was not calculated, because the artists for comparison were not available for validating interviews.[38]

Participants with epilepsy often simultaneously integrated multiple intrinsic perceptions in a single work, either in whole as a theme, or in part as an influence. Fig. 3 shows how a computer keyboard appears to Jude Rouslin when her epileptic discharges cause cerebral metamorphopsia (distortion of shape). Rouslin's self-portrait *Partially where?* (see cat. 35, p. 145) is an example of how this artist integrates her experiences with metamorphopsia into her painting. It also integrates her feeling of spatial distortion, confusion, swirling lines and segmentation of intensified colours.

Self-expression through art is, according to Carl Stafstrom, 'a vastly underused tool in neurology'.[39] The statistical frequency of intrinsic perceptions in the art of people with epilepsy compared to their rarity in the works of people who do not have seizures warrants valued consideration in medical study and practice.

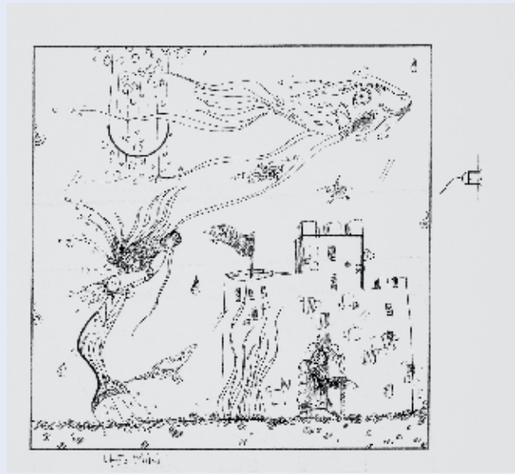


Fig. 6: Matt Rees (Australia), TCT—DP drawing. Administered by Jim Chambliss, Melbourne Medical School and St Vincent's Hospital.

Fig. 7: Anonymous, TCT—DP drawing. Administered by Jim Chambliss, Melbourne Medical School and St Vincent's Hospital.

Fig. 8: Anonymous, TCT—DP drawing. Administered by Jim Chambliss, Melbourne Medical School and St Vincent's Hospital.

Fig. 9: Cat. 20 Cinders Gott (USA), **Test for Creative Thinking—Drawing Production** (TCT—DP), 2008, pen on paper, 15.0 × 14.5 cm (image). Administered by Jim Chambliss, Melbourne Medical School and St Vincent's Hospital. Compare with Gott's ***Primordial womb*** (Cat. 19, p. 129).

Epilepsy and enhanced creativity in visual art

Creativity, for the purposes of this article, is defined as the ability to produce novel or original material that appropriately responds to a motivational drive or need.[40] Novelty and originality are dependent on statistical rarity and remoteness from common, predictable or mundane representations.[41]

Colin Martindale's Biological Theory of Creativity describes the brain as consisting of millions of nodes, each of which has corresponding functions, processors, memories or inhibitors.[42] In order for a person to think most creatively, as many nodes as possible need to be activated simultaneously, to produce truly original images and novel experiences. The basic process underlying the generation of novel ideas occurs during a strong increase in neural complexity and activity, reflecting higher degrees of freedom within the brain's neuron assemblies.[43] The simultaneous activation of nodes in the brain is more free-flowing and widely distributed when a person is in a defocused or chaotic state, such as that caused by transient focal epileptic discharges. Such positive phenomena can occur while people are conscious, able to remember, and capable of incorporating their intrinsic perceptions into visual art, whether at the time of the perception or at a later date. The original and novel nature of the intrinsic perceptions, when substantially integrated, makes the artistic expression inherently more likely to be creative.

In the Creative Sparks research we used the Test for Creative Thinking—Drawing Production (TCT—DP) (1996)[44] as a quantitative and standardised method to compare creative potential in a holistic manner. The TCT—DP provided a way to look at 15 different characteristics of creativity, making it possible to see if and how focal epilepsy significantly enhances creativity. The TCT—DP uses a standardised sheet of six fragments (Fig. 4).

For the control group we chose family members: parents, siblings and children over the age of 18, as they are likely to have the most similar genetic and environmental backgrounds to the artists. An additional reason for using family members as a control group was to ascertain whether epilepsy was a variable that might lead a person to create art as a hobby or profession. That question could not be answered by recruiting artists without epilepsy, at a similar level of experience and accomplishment, for a control group.

Members of the one family tended to produce similar drawings that had correspondingly similar scores. Fig. 5 demonstrates the similarities of the TCT—DP drawings and the total scores of three family members who tested at the median level of the control group. Figs 6–8 are TCT—DP drawings of artists with focal epilepsy, representing some of the higher scores from the test group.

Participants with focal seizures scored at a highly significant level on the TCT—DP for characteristics in their drawing that showed more elaboration and attention to detail; humour and emotional expression; and depiction of surreal, dreamlike or atypical content. The TCT—DP results also showed the following significant correlations:

- Increased frequency of intrinsic perceptions—as reported by the artist—significantly enhanced creativity.
- Increased frequency of integrating intrinsic perceptions into their other visual art significantly enhanced creativity.
- Higher variety of intrinsic perceptions, as measured by the independent evaluation, corresponded with higher TCT—DP scores.

Many of the TCT—DP drawings were very similar to some of the existing artworks of participants. For each of the examples, the completed artworks were linked to the artist's reports of experiencing intrinsic perceptions, which they consciously integrated into their visual art. The inclusion of similar imagery, linked with intrinsic perceptions by the artists themselves, indicates that higher creativity scores resulted from experiencing intrinsic perceptions sparked by epilepsy.

In conclusion, original and unique intrinsic perceptions, when integrated into visual art, can enhance creativity. But this does not occur in a direct cause-and-effect relationship. The influence of epilepsy is just one of the many factors that contribute to the creative process, yet it is a highly significant one. The question of whether intrinsic perceptions or hyperstimulated neural activity from focal epilepsy will enhance an artist's creativity is subject to the free will, artistic talent, experiences and motivation of unique individuals with epilepsy.

Dr Jim Chambliss

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Fig. 10: Cat. 3: Sharon Anderson (Australia), **Test for Creative Thinking—Drawing Production** (TCT—DP), 2009, pen on paper, 15.0 × 14.5 cm (image). Administered by Jim Chambliss, Melbourne Medical School and St Vincent's Hospital.

Fig. 11: Sharon Anderson, ***Ornamental face***, print on paper. All rights reserved by the artist.

Fig. 12: Cat. 1: Sharon Anderson, ***All seeing eye***, 2008, pastel on paper, 27.5 × 38.0 cm (image). All rights reserved by the artist.

Sharon Anderson

I have temporal lobe epilepsy, which for me is an intense and overwhelming feeling of déjà vu. It is then followed by nausea and headaches. The déjà vu feelings are so intense and sometimes it feels as if I am thinking another person's thoughts. These feelings must have somehow shaped the way I create art. I remember as a university art student wondering if I were mystical or had some unique insight. It was not until many years later that I was diagnosed with epilepsy. I had no idea at the time that epilepsy can take many forms and also how the brain can play tricks on what I thought was my aware and conscious mind.

I believe that on a subconscious level my subject matter may show an influence of epilepsy, particularly with the drawing *Created mind*. Looking at these artworks now, I see the use of strong colour and fantasy subject matter links with the type of epilepsy I have (temporal lobe). I don't think about this when I am actually in the process of creating art, but later on I wonder what is really going on in my creative subconscious.

My pastel drawing *Created mind* was created without a thought of it being linked to epilepsy. It evolved as I was drawing it. Things were added, colours were changed and it became me. When I had finished, the reaction from others made me look at it with a new perspective. Because of temporal lobe epilepsy my mind creates its own, sometimes confusing, reality.

Cat. 2 Sharon Anderson (Australia), ***Created mind***, 2009, pastel on paper, 50.0 × 52.0 cm. Collection of the artist.

Patricia Bernard

One might say that my artistic background comes from my desires to create, which are intensified during times of migraines and seizures. Inspiration for my art comes out of many events and symptoms of hallucinations and illusions associated with epilepsy. Sometimes my compulsive energy to make art flows with more activity during times when there is more frequency of seizures or auras from migraines and/or seizures. During those periods, which can extend into days, there are powerful sensations of fear that come out of nowhere, that can go hand in hand with feelings of out-of-body experiences and/or sinking sensations.

I compulsively isolate myself from other people because I want to simultaneously transfer into art the visions and pictures that are going through my head and that could be lost through interruptions. Things are out of perspective, like being in a fun house with wavy lines contorted by warped mirrors. I see the detailed, intricate segmentation of lines mixed with intensified colours and flashes of light.

I develop a passion and unquenchable desire to create even after the event is over, which makes me impulsively paint until the late hours of the night. There is somewhat of a dual consciousness, when my focus on painting the illusions is more intense as my focus on reality and self-control dissipates.

Just pulling me in shows a feeling of being drawn forward in a tunnelling, disconnected rush where everything feels sinking and surreal. I feel compelled to run, but my feet are not following me. The trees come alive with an unstable swirling movement that is out of perspective. The trees are rooted, but not stable or within my reach to grasp for bracing myself to regain control. The frightening intrinsic perceptions are a precursor to what is usually a major shaking convulsion.

Cat. 4 Patricia Bernard (USA), ***Just pulling me in***, 2012, acrylic and ink on canvas, 50.0 × 40.0 cm. Jim Chambliss Collection.

Nadine Binder

Deeply embedded in spontaneity and intuition, my drawings are reflections of my 'need' to draw and are direct representations of my thoughts and feelings.

FACET is a representation of myself as a young woman. I have drawn myself in semi-abstract form with half my face exposed, perhaps because of my split personality. It is as though I make a conscious effort to portray the happy side, whilst keeping the sad side hidden and locked away. The gaze of my eye depicts how shy and vulnerable I have been at times. The fixed blank stare of my eye is a direct link to what I experience before the onset of an epileptic seizure, a warning sign for many other people living with epilepsy.

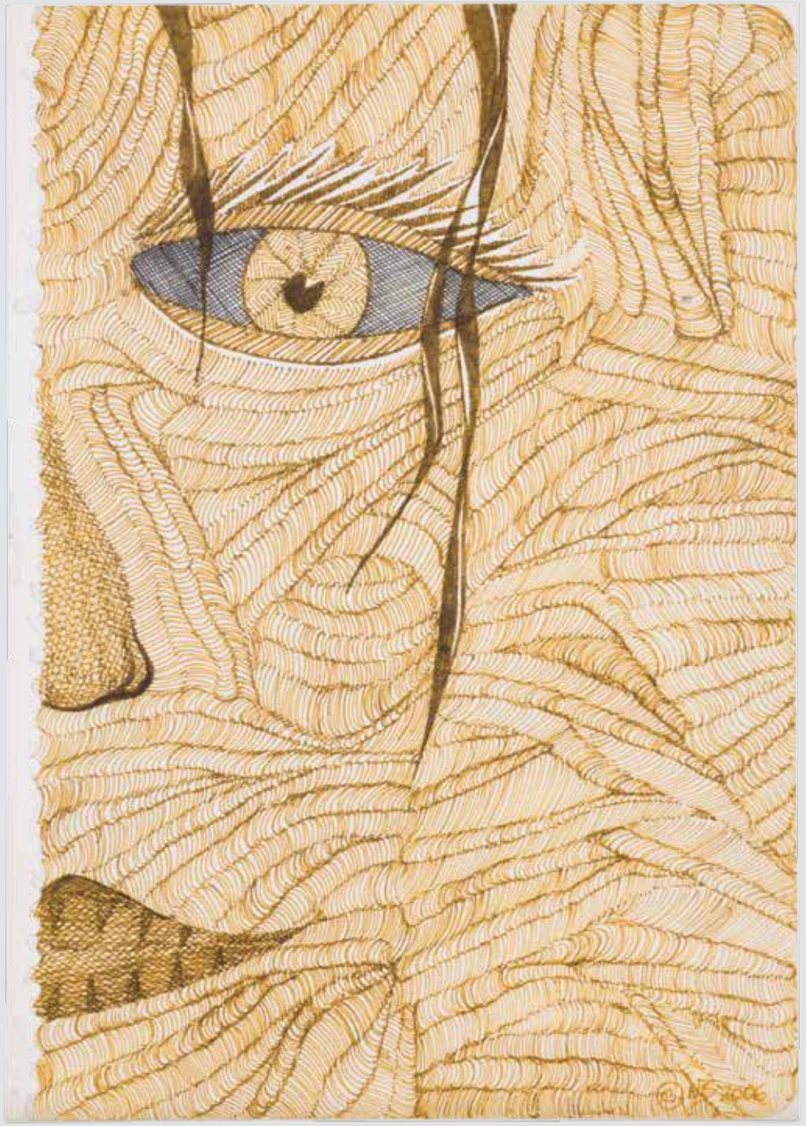
My drawing manner shows that I'm a perfectionist and appreciate detail to the highest degree. This can be seen in the dense line-work, the close-knit crosshatchings and the bold streaks of hair. The layering of the line-work represents my impulsive 'need' to draw. Repetitive swirly lines that connect and overlap are the essence of my drawing and show an inclination towards rounded forms. Nothing in the drawing incorporates angles, which I have found to be a repetitive aspect in my work.

I often feel a lot of tension being released during the drawing process and a wonderful sense of achievement with the end result. *FACET* is a reminder of a very creative time in my life and inspires me to stay true to myself.

The greatest influences in my art are recurring dreams, the natural environment and innermost thoughts and feelings. The connection between my drawings and my condition lies in the idea that they form order out of the mind's disorder.

Sharing my story and artwork has helped me to gain greater insight into myself whilst raising general awareness about people living with epilepsy. People consider epilepsy a disability; I consider it a gift of creativity and express myself using this gift through my artworks.

Cat. 5 Nadine Binder (Australia), ***FACET***, 2006, pen on paper, 21.0 × 14.8 cm. JTA Australia Collection.



Marea Breisch

The *Orange nude* represents my struggle to recreate my life after a horrific fall down some stairs resulting in an acquired brain injury and epilepsy. In 1995, from June to December, I was in a vegetative state. I could not walk, stand or speak. I did speech therapy for 10 years, Monday through Friday.

I was, before then, a successful VCE teacher (painting and history), artist, mother and sportswoman. I had to start again. I wanted to teach and I couldn't. I wanted to talk and I couldn't. I wanted to drive and I couldn't. I wanted to paint and I couldn't. Epilepsy, difficulty with communication and reading, and inability to pursue a career all destroyed my self-worth.

My determination and will to be the person I was helped me through this difficult time. I started to paint again four or five years after my accident. I was frustrated with the differences in my art before and after acquiring epilepsy. I had to get help in mixing colours, because I could not read the labels. I had problems with hand and eye coordination. Painting nudes was different.

My art now represents freedom and a return to a small part of what I valued in life. I want my children, Miranda and Richard, to be proud of their mother.

Cat. 6 Marea Breisch (Australia), ***Orange nude***, 2005, oil on canvas, 112.0 × 87.0 cm. Collection of the artist.

Emma Brockett

I had meningitis when I was six months old, and was 14 years old when I was finally diagnosed as having epilepsy, with complex partial seizures.

All my life I have been making intensely detailed mandalas and geometrical pictures in vivid colours, in addition to black-and-white patterns, highlighted with gold and silver. I started drawing 'dot pictures', in Textas, at a very young age. Despite medications failing to control the 40-odd seizures I can endure every month, my artwork has been the one consistent form of expression throughout my whole life.

Linear confusion came from a need, or desire, to 'mess with people's minds'. I find it interesting that people always seem to have an interest in 'putting things right' or pointing out 'mistakes'. The picture consists of a background design painted in black ink on white cardboard. Small movable pieces, cut into different shapes, which mirror the pattern beneath, are attached with split pins. A person can opt to have the pieces lined up with the identical pattern below or twisted to make the pattern beneath obscured.

Epilepsy is much like *Linear confusion*. When not having seizures we are all 'lined up'. After or during seizures, the patterns of ourselves are quite 'messed up' or 'out of line'. It may take time, a few minutes or a few hours, but we come back to line up with the pattern of our real selves again.

This picture reflects the way I am the majority of the time, until another seizure strikes and puts me all out of focus again. Upon reflection, epilepsy is not the only part of my life which has been 'misaligned'. I am adopted and the only Vietnamese person in my family. People make assumptions about me.

All in all, the question has to be asked: 'Does the pattern need to be "fixed" in the first place?'

Cat. 7 Emma Brockett (Australia), ***Linear confusion***, 2003, mixed media on paper, 101.0 × 63.0 cm. Collection of the artist.

Jim Chambliss

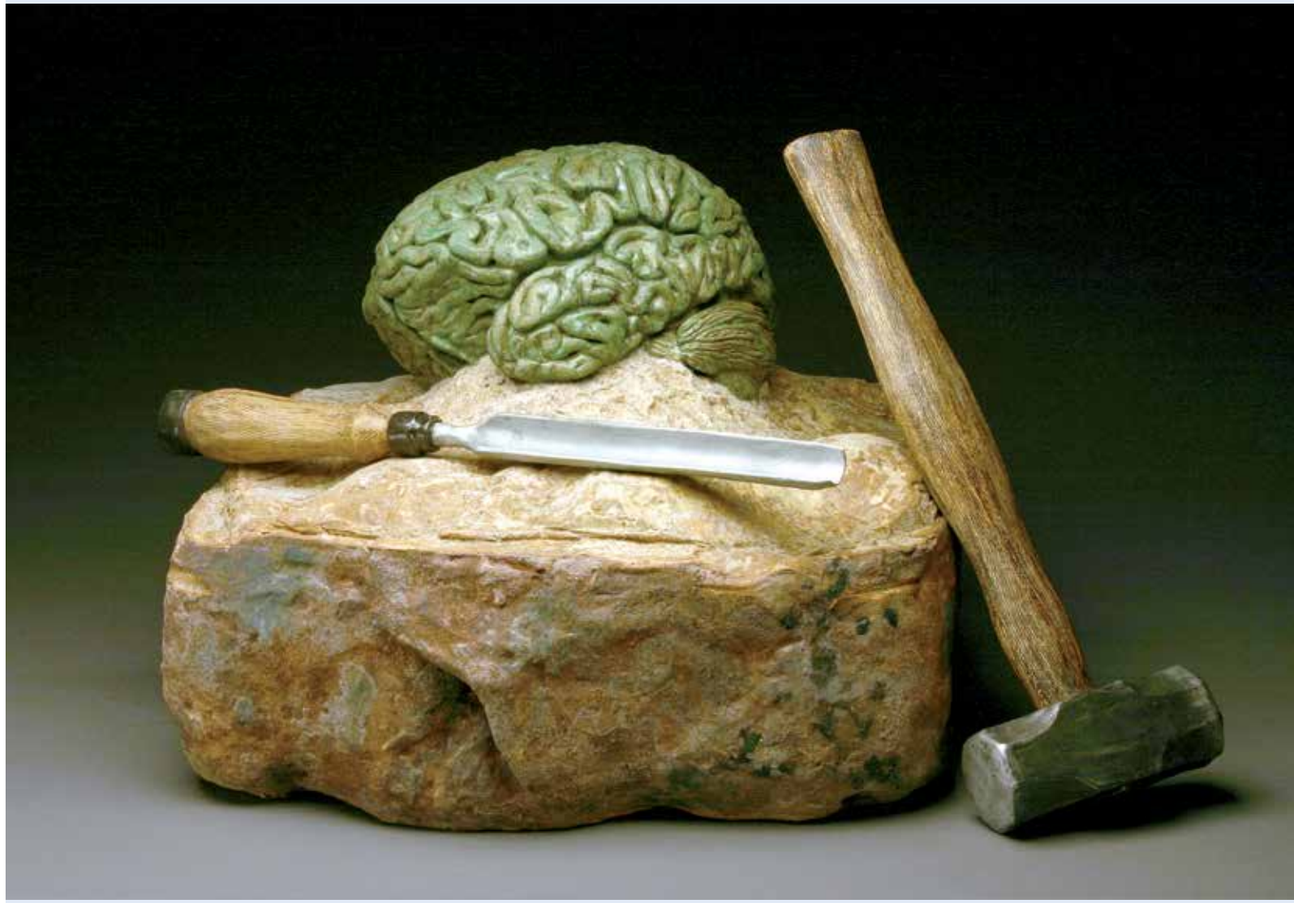
Discovering the source represents the process of frustration and persistence to discover what happened to my brain, and who I became in adjusting to devastating challenges, and how I evolved to be more creative following traumatic brain injury and acquired epilepsy.

The stone represents how hard it is to break through inadequate scientific paradigms and social barriers of ignorance, myths and prejudices towards people with epilepsy.

The psychosocial impacts of epilepsy can often be more hurtful and impeding than the transient neurological symptoms. It takes enormous effort to understand and break through the misconceptions of what happens beneath a skull encasing an injured brain with altered functioning from epileptic discharges. No enduring process of sweat and persistence can completely reveal a solid and tangible object that can be viewed, touched and measured to reveal the flaws that can be reconstructed. After years of chiselling through to glimpse a cerebral surface, the functions and feelings remained beyond my grasp.

Under the cumulative layers of sandstone is a metaphorical brain of jade, which is widely regarded as a symbol of healing. For me the healing needed was to move forward to make the most of a brain that was altered, not broken. From that came a quest to discover my new pathways when the rigid and constrained practice of law was no longer my best fit. I discovered that my cognition became remarkably visual, with unique imagery impulsively coming into my mind's eye. I pursued an education in art and quickly began accumulating awards and publications. I had no recognisable talent or training in art prior to the onset of epilepsy.

My art conception and production, along with my writing, were excessively detailed and circumstantial, which I later learned to be consistent with interictal behavioural changes that were never mentioned or explored by my initial neurologists. My experiences with epilepsy and the psychosocial changes associated with it would have been less hurtful had these doctors been trained to recognise the diagnostic value of creative art and writing.



Cat. 10 Jim Chambliss (USA/Australia), ***Discovering the source***, 2005, ceramic, 38.5 × 45.0 × 45.0 cm. Dr Steven Schachter Collection, Harvard Medical School. Photograph by Todd Burns.

Vicki Deutsch

This is a piece I created many years ago while I was still having seizures. I have not had a seizure in almost 15 years, due to the medication that I take.

Fear of fear represents the various emotions that I was going through at the time of my seizures: great sadness, fear, anger, anxiety and occasionally a smile to try and forget what I was going through. There are waves of tears, accompanied by the feeling of being wrapped like a mummy in something I could not control.

The cloud with lightning is like the aura I would have seconds before a seizure. Jagged orange and yellow slanted lines like the lightning would flash before my closed eyes and then the seizure would begin.

My hand grips the material in a sign of fear.

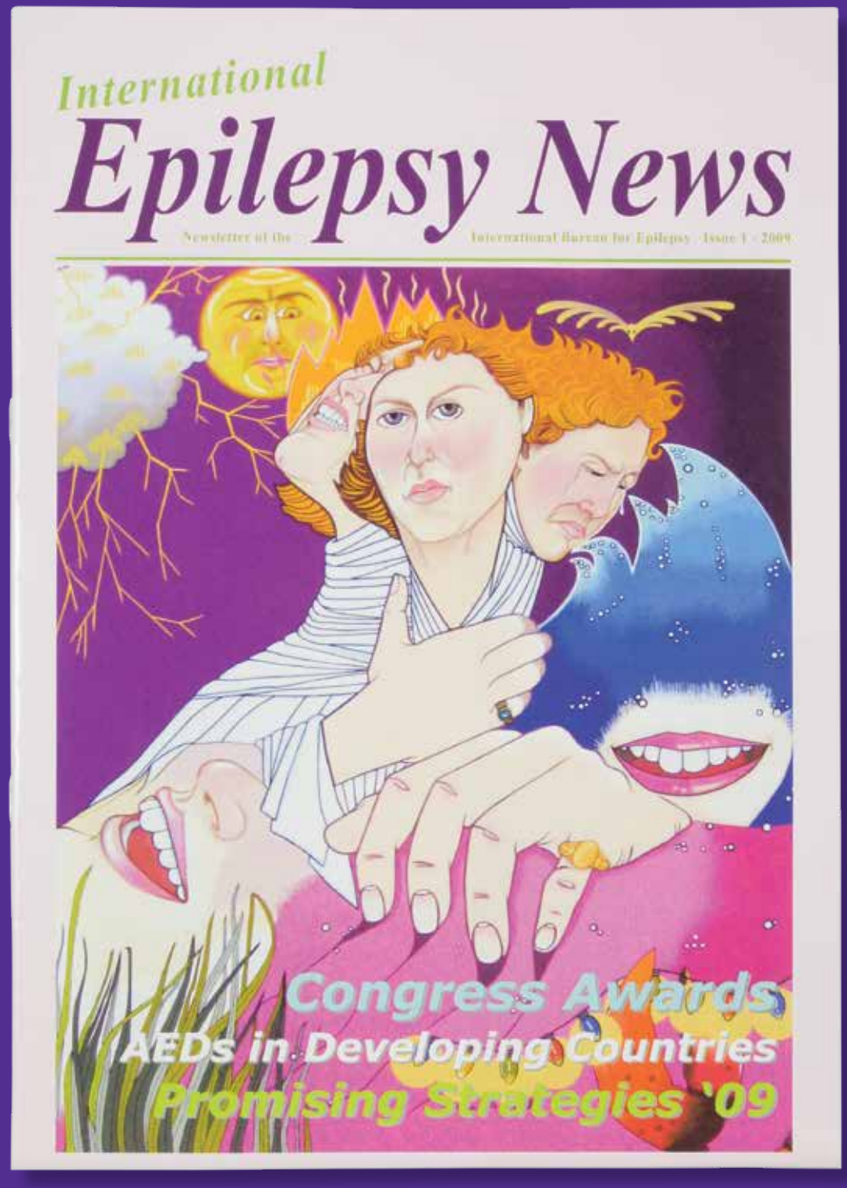
Christmas is a favourite time of the year for me. The Christmas lights represent this. It is also the time of year that I was born. The lobster claw represents the loss of joy as it cuts the colourful Christmas lights.

The Moon looks both a little surprised and angry amid the sky that is the overwhelming darkness in my life at the time.

I don't remember what the sea grass represents but it adds to the colour and design of the piece.

I think the bird was like the Holy Ghost that was coming down upon me to get me through the worst of times.

Cat. 12 Vicki Deutsch (USA), ***Fear of fear***, reproduced on cover of *International Epilepsy News*, issue 1, 2009, print on paper, 22.3 × 18.5 cm (image), 29.5 × 20.7 cm (cover). Jim Chambliss Collection.



Myron Dyal

I have TLE (temporal lobe epilepsy). It resides in my hippocampus and has been there since I was four years old.

The painting *Face out of the wall* was a hallucination that I experienced during one of my seizures. I was walking in the mountains and I began to 'feel' different. The world began to change and I saw before me what looked like a wall. The wall began to change its shape and slowly became the image that you see in the painting. My mind seemed to 'forget' where I was, but I did know who I was, but 'time' and the world in front of me was lost in a different dimension. It lasted for some time (I am not sure of the amount of time involved), and then it seemed to fade and was gone. I have been seeing these, what I call 'events', all my life and have made a record of them in my art.

I draw, paint and do sculpture and have shown all over the world. The pure fact of the matter is that it is the seizures themselves that have been the catalyst for my art and even though my life has been difficult, in the final analysis it has been worth it all.

Cat. 13 Myron Dyal (USA), ***Face out of the wall***, 2006, oil on canvas, 97.0 × 66.0 cm. Collection of the artist.

Tremain Farrar

My art is the vehicle through which I express my obsession with detail and the rendering skills I have honed to capture the verisimilitude of the subject. In addition to my epilepsy, I have been diagnosed with clinical depression and obsessive-compulsive disorder. These afflictions have manifested themselves in various forms and I partially contribute the development of these psychological ailments to the negative attributes of inherited epilepsy. The artwork I create is autobiographical and therapeutic; the lines I scrutinise and meticulously draw on the paper are invigorating and enable me to focus my emotional and physical attention on a single task. I want to divulge my neurological disorder through my artistic creations, exposing myself unabashedly to the appraising public; my epilepsy does not hinder me from expressing genuine feelings but enables me to truthfully explore topics that some artists would be hesitant to address, such as psychological and medical maladies.

Ascension no. 3 depicts how I feel immediately after a tonic-clonic seizure: alone, disoriented, frightened and, above all, disconnected from reality and sentience. The horrifying aura that precedes a seizure can be likened to a feeling of extreme disorientation, a disembodied sensation with limited control of kinaesthetic abilities. The meticulously drawn human figure, juxtaposed to the grey, nebulous background, is intended to encapsulate the eerie sensation I experience after a seizure; although I'm rising towards consciousness, my awareness of the surroundings is distinctly blurred. The self-portraiture that is framed within a costumed, staged tableau is a component of my larger interest in constructed identity and fragile consciousness. The theatrical gestures and facial expressions of the figures are intended to heighten the drama of the surreal environment, a suggestively domestic arena from an estranged perspective. The work ultimately seeks to navigate through the reeling psychological journey of epilepsy.

Cat. 14 Tremain Farrar (USA), ***Ascension no. 3***, 2010, charcoal on paper, 73.0 × 110.0 cm. Collection of the artist.

Ryan Fletcher

Sacrificing service depicts my convictions of understanding epilepsy through spirituality. On the right side of the painting you will notice two separate sets of comments. The first reads: 'Think to be before you act (act once thought upon)', which means envisage the state of existence you aim to achieve and act accordingly (remaining cautious of what you wish for, as you may receive it). This is a response to L. Ron Hubbard's position that 'Thought was boss' (mind dictates the structure of the body).

The second comment (located in the bottom right-hand corner) was the conclusion I took from Scientology, which I crossed out to deter the condition of PTS (Potential Trouble Source) from occurring. The comment reads, 'opposing the imposition of will' (with an added WARNING resting beneath it). What this says to me is, 'do not form a position rooted in aggression against another being's self-survival', as Scientology teaches that survival is the common denominator upon which we all share. I ceased association with the Church of Scientology after hearing a lecture by Hubbard state that 'epileptics' were to cease their medication in order to undertake auditing services.

The symbolism of the Freemasons, 'G', as shown in the top left, symbolises the Great Architect of the Universe. The square and compasses, at the top right, symbolise square dealing and keeping on course with one's community, with the all-seeing eye of God watching over summarises a range of faith exposure, overseeing the portrayal of Jesus Christ in the centre of the painting, pertaining to my baptism.

The bottom left of the painting, which reads simply as 'The Pot Market', relates to the potential benefits of entheogenic healing. To me cannabis is a natural treatment that isn't tied to the petrochemical production line of the transnational pharmaceutical companies.

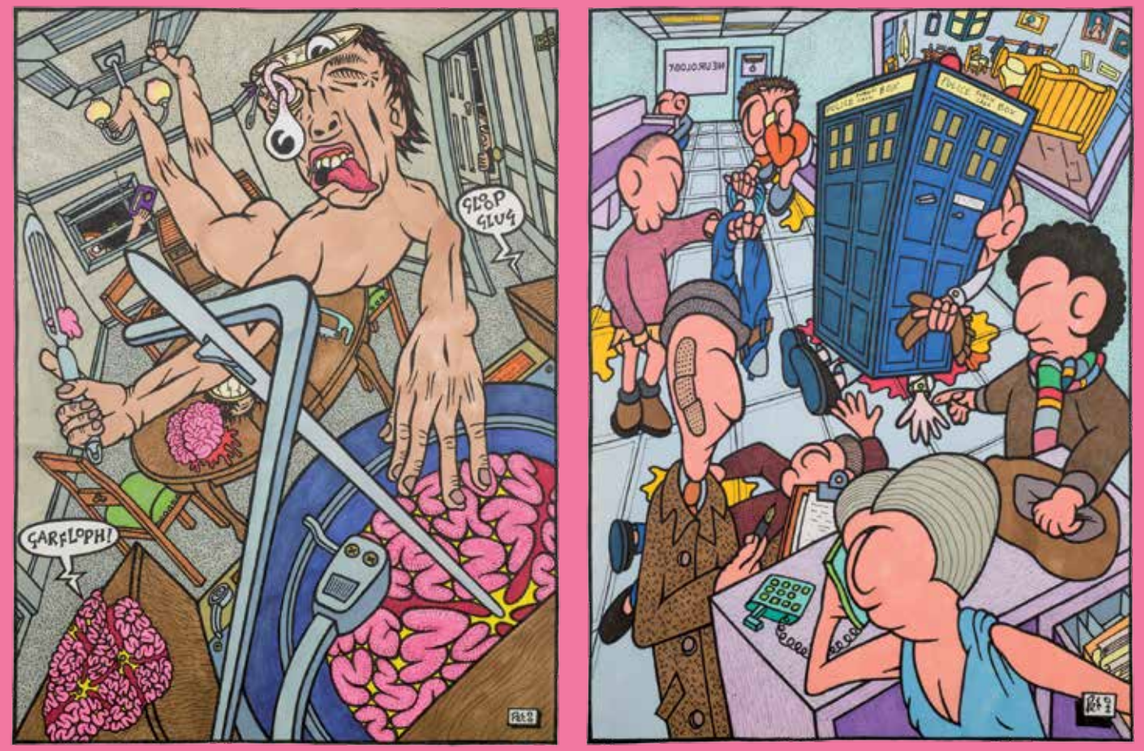
Cat. 15 Ryan Fletcher (Australia), ***Sacrificing service***, 2011, acrylic on canvas, 60.0 × 45.0 cm. Collection of the artist.

Peter Goodman

Epilepsy
It is strange for a person with epilepsy to view his brain via MRI and PET scans. How could these photo-like slices of his brain represent the pain or déjà vu-like experiences his seizures give him? The disk-like MRI scans in particular, with their black plastic appearance, could be individually cut from their photographic multi-image host and played on a record player similar to the vinyl discs people of my age grew up with. Perhaps a special stylus could show the auras or déjà vu-like moments? This drawing shows not only the old phonograph player of my childhood but also furniture and other memories from those earlier years.

Blood Bank
If the phenomenon of time travel was real, could people with intractable illness travel to a time in the future when a cure was available? What if Vincent van Gogh could be transported from the past to a modern-day consultation with a specialist? What would Vincent think if he saw reproductions of his paintings in the waiting rooms of the future? Would he observe other patients in waiting rooms lose bladder control or disrobe in seizure state? Would the receptionist of the future recognise the person from the past or maybe consider him a patient suffering from post-ictal psychosis? I think this synopsis brings new meaning to the phrase 'time heals all wounds'.

After having right temporal lobe surgery early in 2012, I found my mind empty of ideas for art and the inspiration to make new works or even continue pieces I had started pre-surgery. Approximately 18 months after surgery I began having seizures again. With the return of a seizure state, my mind became full of new ideas for art. With a little pleasurable effort, I worked to translate these new images to the visual world, with results I consider as good as, or better than, my pre-surgery work.



Cat. 17 Peter Goodman (Australia), ***Epilepsy***, 2000, pen on paper, 43.0 × 31.0 cm. Collection of the artist.

Cat. 18 Peter Goodman, ***Blood Bank***, 2001, pen on paper, 43.0 × 31.0 cm. Collection of the artist.

Cinders Gott

Floating weightless, timeless, in the quantum World Egg cradled by the All+Mother in liquid healing warmth … a Sacred Space of nurturance, sustenance and pure acceptance … we are in a symbiotic dance with our Creatress … yin then yang … always striving for balance … living to love … gently gliding in the Wishing Well of human spiritual existence.

Mother Goddess of the Primordial Waters suspends blissfully, while contemplating her existence. It is no small thing to give and receive simultaneously and consciously, with unconditional love. Yet she has inner strength and a flexible resiliency. She gingerly seeps between rocks and particles of earth as does she eagerly devour air in the surge of a chaotic wave. Curvilinear tendrils of seaweed, her liquid essence, reconnect with her life-giving womb.

The archetypal imagery of the Birthing Goddess calls to us with its universality. If there is one thing humans have in common, we are all born from a 'wom(b)an'. She is at times the vessel in which to bring one back into a new life path or incarnation. Her womb … the Holy Chalice, or Cauldron of Life, held us each in her keeping. Now it is our time to reciprocate, to make the Gaian Connection for the purpose of gratitude and healing.

As a Priestess of the Goddess, and shamaness in training, my spiritual link to nature and Mother Earth is my solace. Healing information often comes to me in the form of visions that surface during altered states of consciousness. I now utilise these opportunities for viewing the world between worlds, parting the veil, as I dance 'Into the Mystic'.

Since science can now monitor how nature can help heal our psyches, why not integrate nature into the healing process? E(ART)H! Join your organic world … take the plunge!

Cat. 19 Cinders Gott (USA), ***Primordial womb***, 2007, ink and watercolour on paper, 24.0 × 14.0 cm. Collection of the artist.

Sylvia Heuge de Seville

I tend to always depict people and their feelings in my art. *For those we loved* depicts a sense of loss. It was painted after the Tsunami in Samoa and Tonga in 2010.

Much of my art addresses problems of colonisation and racial difference. This could stem from my own ancestral roots, which go back to generations of African slaves, caught between two worlds on the British colony of Bermuda (only a recent discovery as, for years, I believed I had Indigenous Australian ancestry).

In much of my work, I have a tendency to place a halo around the heads of some of my painting subjects. To me, this depicts an essence of spirituality (as in paintings of the Madonna), but another explanation could stem from my own social sensitivity. I pick up on people's thoughts and feelings, so much so, that a loud or intense person can sometimes cause me to have 'auras', or what I call an epileptic 'episode'. Before I have a seizure, my brain is so over-charged that I feel like it is short-circuiting electrically. I have small 'absences' or auras, until my brain finally 'blows a fuse' and I have a full-blown seizure, of which I have no memory afterwards. Since being around people increases the 'noise in my head', I tend to seek out the quiet and solitude of my own space (usually my room).

Having epilepsy turned me into a virtual hermit, as I lacked the confidence to go any place where I might have a seizure in public. During the long hours by myself, my art became my solace. The halo in my paintings represents a 'quietness', a 'softness' and a more spiritual aspect of one's *being*. It is also a reminder of the auras that I experience prior to a major seizure.

Cat. 21 Sylvia Heuge de Seville (New Zealand), ***For those we loved***, 2010, mixed media on canvas, 110.0 × 152.0 cm. Collection of the artist.

Sherion Jones

Art has always been my greatest passion. My art is my place to escape … to dream … to dance … to shout … and the only place where it is impossible for me to make a mistake. There is no greater freedom.

I have multiple sclerosis, epilepsy and migraines. My body and my mind are not as reliable as those around me. Art makes me feel 'normal'. The viewer does not see that I may be clumsy and fall, that I may not be able to see, that I may feel pain or suffer uncontrollable seizures. The parts of my life that are difficult for others to understand are near invisible to those who view my creations.

I am a self-taught artist and enjoy working in pencil, watercolour, acrylic and mixed media. My techniques are not perfect, as they are primarily learned via trial and error. My greatest inspiration comes from people. I look for that fleeting moment when the ordinary becomes extraordinary. I know in that moment that I have to capture that look or that gesture and illustrate the effect it had on me. I find a certain beauty in awkwardness, vulnerability and things most people find unattractive.

The biggest change in my art came when I realised that my creations and my techniques do not have to be perfect. I am not a square peg trying to fit in a round hole. I am a round peg and I fit perfectly in a square hole. I strive to encourage others, with and without disabilities, to find their passion in life and lose the fears that keep them from accomplishing their dreams. Your dreams belong to you. You cannot make a mistake.

Cat. 24 Sherion Jones (USA), ***Mystified***, 2008, mixed media, 57.0 × 47.0 cm. Collection of the artist.

Maggie Keegan

Temporal zone 1 is an attempt to show what I see, literally, when I have seizure activity. It is not a view of a particular instance but an amalgamation of various 'scenes' I have experienced. In reality the images would be moving and changing all the time. The elements present can change, but eyes, mouths and hands are usually present. The 'light source', always to the left of my field of vision, here is half hidden behind the hand. The intense, colourful light that extends through my visual perception is always present and sometimes the only thing I see; it is a warning of a period of increased seizure activity.

Most of my art is influenced by epilepsy, especially my *Seraphim* series and my *Spirit cat* series. A Seraph, the plural of which is Seraphim, is a member of the highest order of Angels. Often before and during seizures I see an intensifying brightening of my environment emanating from behind me to the right, which I have always thought of as the presence of an Angel.

I don't usually discuss the influence of epilepsy on my art with people viewing my art. I believe that while I know what the works represent and what they mean to me, anyone viewing them should be allowed to see what they see in them without being told what to see or think of them. I have made an exception in this case because I think that the study of art and epilepsy is very important, as most people still have a very poor view of epilepsy and epileptics.

Cat. 25 Maggie Keegan (Australia), ***Temporal zone 1***, 2008, ink and watercolour on paper, 60.0 × 48.0 cm. Collection of the artist.

Serene Low

I am a person with epilepsy and migraine. I had febrile epilepsy at infancy. I outgrew febrile seizures at the age of seven and later suffered a relapse of epilepsy with grand mal seizures at the age of 18. I started having migraine at the age of eight and have lived with epilepsy and migraine ever since.

My life during the earlier years of my epilepsy and migraine was a tormenting, neglected and embittered one. Mention quality of life and I can testify that it was a derogative and almost meaningless lifestyle that I had to put up with. It was truly very tormenting to have to live life with epilepsy, a medical condition that is shrouded with pitch darkness and wrongful conceptualisations. A great lack of information and social support with acceptance had made life extremely dreadful.

With no cure in sight, coupled with my loved ones' inability to ease my sufferings, rendered me a deep sense of neglect. Every time I regain consciousness after a seizure, I wished for life to end as abruptly as the unpredictable and unforewarned seizures. Depression ensnared the manifold beauty of life.

Epilepsy embitters. To be living and deprived of the freedom to engage in many social aspects of a normal life, life with epilepsy is downright degrading. Friends belittle and shun us. Employers threaten to terminate our employment. Driving is mostly a 'No, no'. Dating and marriage are a taboo. Pregnancy should be avoided. With so many myths and misconceptions, how reasonably well can a person with epilepsy live life happily and meaningfully?

A picture tells a thousand words. It is through painting that I am able to express my epilepsy and migraine in beautiful and abstract ways. Undeniably, I have found painting to be one of the most effective methods of disseminating information.

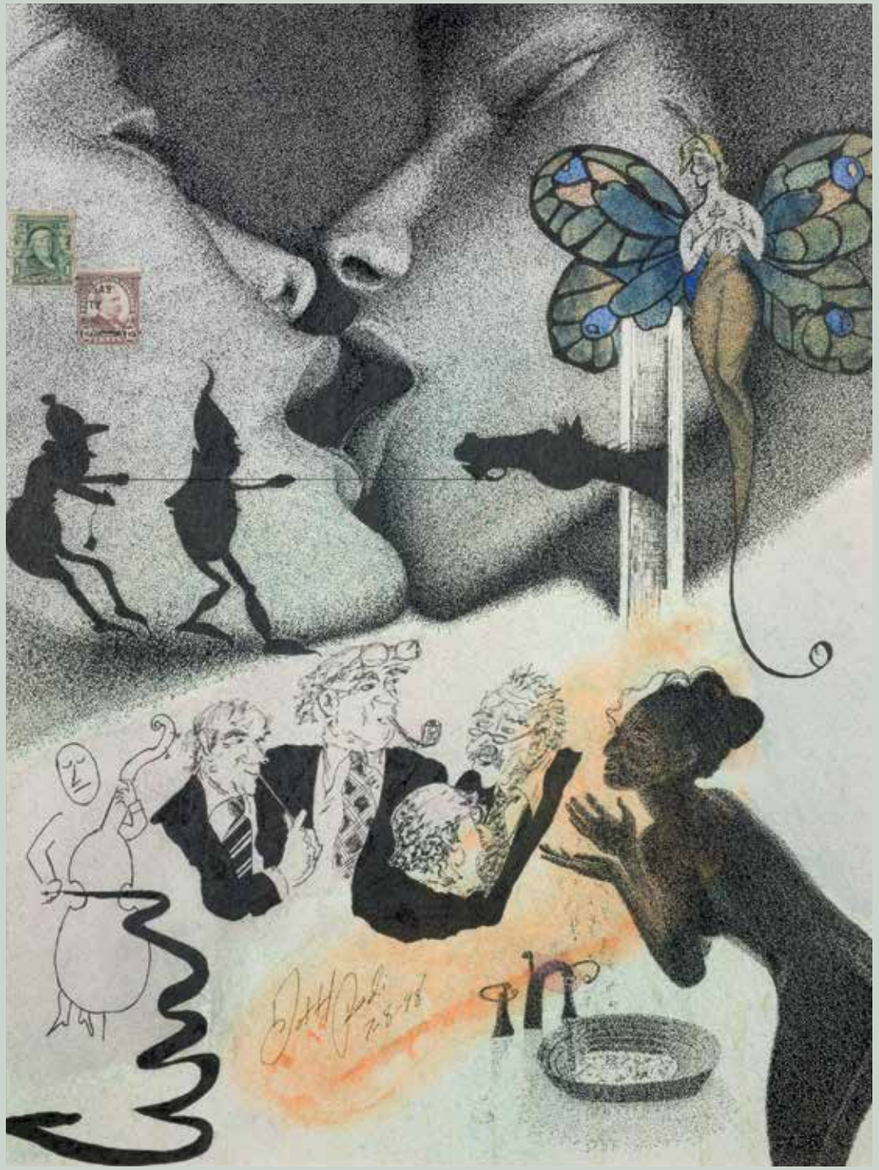
Cat. 26 Serene Low (Malaysia), ***Epilepsy and migraine***, 2008, acrylic on canvas, 74.0 × 56.0 cm. Collection of the artist.

Dotty Pedi

I have been an artist my entire life. Before epilepsy took over, I worked for a major bank, in the accounting department. It is now 17-plus years post-surgery. I am still unable to work a 9-to-5 job, but was able to keep my artistic ability post-surgery.

Almost all of my artwork is connected to epilepsy. In *Wandering thoughts* you can almost feel the fear the horse has of coming out of the stable. Much like the fear I had when facing brain surgery, the woman in the corner is trying to wash it all away, but it's not going anywhere.

Having epilepsy I feel has made me incredibly creative. I see things in objects other people don't. I look up at the clouds in the sky and see things others don't. I doodle/draw almost every day and if I'm unable to then I think about art every day. Post-surgery, I find I am more creative with my hands. I like to assemble things out of found objects and work with clay.



Cat. 31 Dotty Pedi (USA), ***Wandering thoughts***, 1998, ink and watercolour on paper, 65.0 × 53.0 cm. Jim Chambliss Collection.

Fiona Pringle

I've not been diagnosed with epilepsy, though I have experienced migraine with seizures. I understand that a difference between migraine and epileptic seizure is speed of onset, but I seem to go into such a sensory overload (sometimes for quite prolonged periods) that it's hard for me to assess where one might begin and the other end!

For the main part my migraines begin with dull, familiar pains behind the left eye. I might see flashing lights and zigzag lines.

However sometimes, independent of my migraines or sometimes as they fade, I will experience a momentary state of disoriented confusion, altered thinking, déjà vu or an out-of-body experience. Unlike the gradual onset and auras of migraine, these images appear in pings or flashes of fantastic imagery, occupying my entire visual field and overlaying any scenes that are before me.

Like bright visual jewels, these images enlighten, providing glimpses into unique, surreal environments. It's a wondrous experience, so complex and detached from the real world. Scenes appear with the sort of flowing reality that you might see in a well-made film or animation. Briefly captivating, they make me feel as if I'm in an undiscovered and fabulous dimension! At other times images appear in static form, dissected and segmented like an illogical jigsaw.

Many years ago I had an MRI, and one EEG that did not show seizure spikes. From that one EEG the neurologist concluded that I did not have epilepsy. But what if I have a combination of epilepsy and migraines and the 'electrical mischief' of the epileptic discharges was just silent during that exam?

If so, perhaps I'm lucky to have come so far through life without that particular label, which evokes such negative social perceptions and consequences. Or perhaps it might have been helpful to recognise earlier how differently I 'see'!

Cat. 33 Fiona Pringle (Australia), ***Contemplation***, 2001, ink and watercolour on paper, 44.0 × 65.0 cm. Collection of the artist.

Matt Rees

Forgotten lands is a fictitious landscape set in a surreal world of land merging with biology. My initial goal for this piece was to show that every element seemingly out of place had a purpose. 'Drawing the viewer in for a second look' was secondary. Thinking in that way simplified the idea in my mind and made it easier for me to complete when extra detail was required.

Forgotten lands took two weeks to finish amongst my graphic design studies, and is one of two landscapes I've created with use of lyrical line. I used the full spectrum of colour in pencil and pastel, completing the work with both warm and cool tones. In short, my entire pencil set was used and another needed to be purchased for the next work. The themes in the artwork are not meant to be frightening, even though *Ghostbusters* is one of my favourite films. Developing my skill in line art was at the forefront at all times.

Regarding the mediums used, much like my epilepsy, pushing the limits of colour pencil via the combination of land and biological formations was too much to think about. At the time, putting pencil to paper with little planning was my best option. In saying that, comparing a seizure or migraine aura to this work is very easy, as both required decisions of action with very little time to think things over. Also, the prediction of a major atonic fall from minor headache used to be as difficult to judge for me as the decision to not use an eraser, 'at all', for the first time. As a result, *Forgotten lands* had no edits, reworks or structure changes and was completed without anxiety or indecision.

The idea that art and epilepsy can be compared closely is valid here. The initial thought process was daunting but eventually it was easy to put those thoughts aside and relax into a consistent pace.

Cat. 34 Matt Rees (Australia), ***Forgotten lands***, 1997, pencil and pastel on paper, 76.0 × 96.0 cm. Collection of the artist.

Jude Rouslin

Partially where? is both a statement and a question. It demonstrates a particular aspect of focal epilepsy (partial seizures). It's an episodic event that runs through a dual state of consciousness and altered reality. Each partial seizure, whether complex or simple, can be different, as my multifocal epilepsy affects various skills, memories and sensory processing areas of the brain.

I have an intractable multi-focal seizure disorder with unusual variety of sensory and motor manifestations. Sometimes images that I call 'visual seizures' are distorted shapes, lines, grids and intense colours. The novelty of the 'visual seizures' allows me to go beyond the expected and well-established norms and truly create from within. There is a heap of combined experiences and strange images that last seconds. It's analogous to a sudden bottlenecked traffic jam of short duration (like my seizures) and it's over when normal traffic resumes. I can look at what is actually unfolding as a partial seizure continues and remember the dynamic chaos of the bottleneck as an observer of images detached from reality. Once the 'visual seizure' has played out, I can bring it back, like a strange photographic experience.

There are times when I have clusters of seizures and become more active in creating art. However, interestingly enough, just as I was riding that rollercoaster when misfiring neurons were most out of control, my art also seemed to have followed that same ride. Before the medication brought my seizures more under control, I sometimes didn't know my colours, and drawing smooth lines was futile.

The sheer number of seizure experiences over decades has allowed me to become less affected emotionally and unafraid to integrate such divergent thoughts into my art. Perhaps my greatest challenge is to use those experiences to visually document what the uniquely fascinating images that accompany my seizures can be.

Cat. 35 Jude Rouslin (USA), ***Partially where?*** 2005, oil on canvas, 50.0 × 40.0 cm. Jim Chambliss Collection.

Janet Y Shagam

According to the World Health Organization, malaria affects 3.4 billion people, or 50 percent of the world's population. Therefore, antimalarial medication is often required when people living in the United States travel to places where malaria is endemic. Malarone, the medication I took while visiting Senegal, Africa, is the only antimalarial that is safe for *most* people who have epilepsy. Unfortunately, I was not one of them.

Loon Lake is an etching I made shortly after returning home. The etching has been exhibited in several galleries and in one museum. Frequently, viewers want to know more about the image. To most, I say it is an emotional response to seeing an impending storm hovering over the horizon. To a select few, I do explain further.

An awkward silence often follows. A few people say, 'But you look so normal', and every now and again a subtle and knowing nod of the head follows my answer.

Without question, *Loon Lake*, an image depicting the feeling of impending doom and gloom that often precedes a seizure, does have links to my epilepsy. However, until I participated in the Sparks of Creativity study, I believed that *Loon Lake* was one of only two images that made direct or indirect references to epilepsy.

I now appreciate that epilepsy may have more influence than I thought possible. According to study findings, people who have epilepsy often incorporate scratchy lines into their work and use a palette where dark colours, with an occasional bright splash, predominate.

On the whole, I prefer to think of myself as an accomplished person who happens to have epilepsy. Separating accomplishment from the condition helps me, as well as others, understand I am the one who defines the relationship between me and my different brain.

Cat. 36 Janet Y Shagam (USA), ***Loon Lake***, 2006, etching on paper, 92.0 × 74.0 cm. Collection of the artist.

David Sharp

This self-portrait is, simply, an act of creativity. One which plays with modern styles of artistic expression (Picasso's use of profile) and a personal expression of caricature then applied to me. Before epilepsy (idiopathic) I was always into the arts. With epilepsy I simply continued with a broader oeuvre of experience to draw upon.

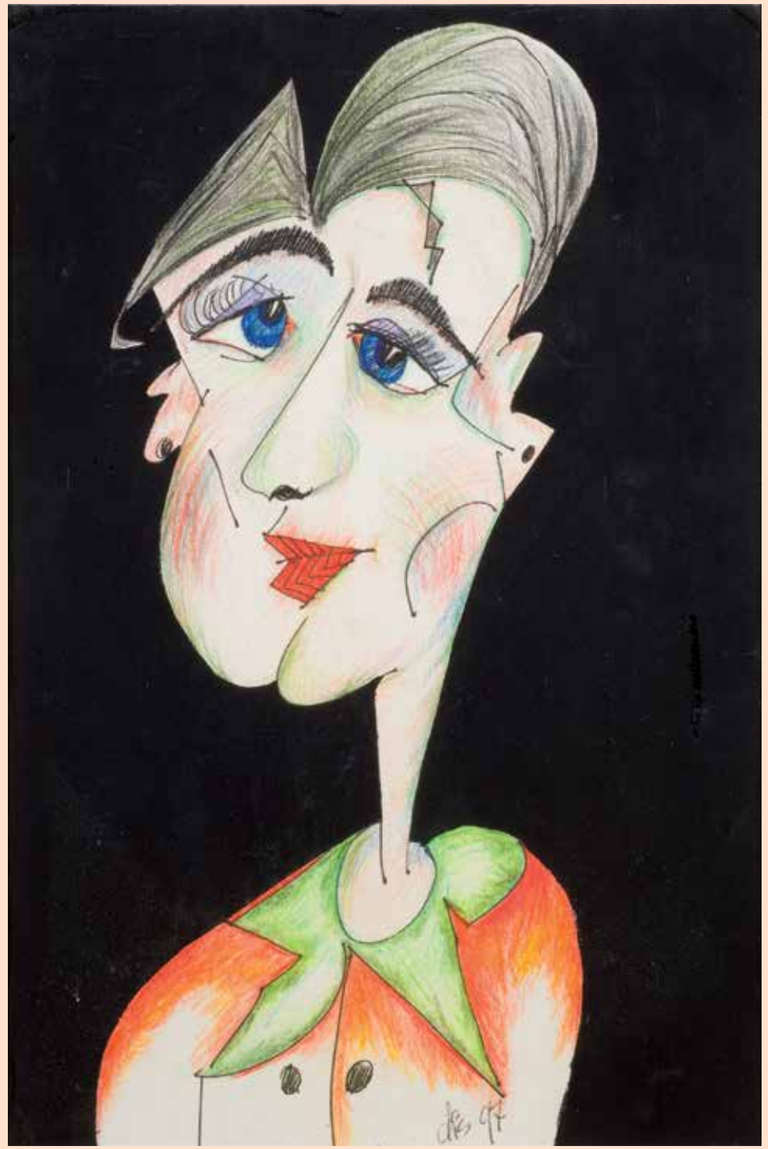
The self-portrait's relationship with epilepsy is that it was one of many pieces of art created whilst I was on a disability pension after my AEDs 'crashed' and control of my epilepsy was lost for a few years. It was a time which saw a personal fascination for art inadvertently become a therapy against the darker side of experiencing epilepsy—a darker side I consciously choose to forget. This approach, combined with a good sense of humour and a touch of faith, was able to get me through almost four years of 'disharmony' and continues, to this day, to assist.

Restricted to self-comparison, creative exploration and experience act as a focal point away from the stress associated with epilepsy and life in general and thereby is, I believe, one of the best medications. A seizure initially invokes fear, but when referred to later in a creative perspective it becomes an enhancement rather than a burden. This approach also makes the darker aspects of life easier to bypass or forget, which in turn is a good anti-depressant.

I'll end with two quotations from two people I was fortunate enough to know as close personal friends:

> '… with epilepsy you have a choice, fear or fascination.'
>
> —Ms Sue Davies, former Manager of Epilepsy Association of Western Australia

> '… the creative experience, no matter how small, is everlasting and shall touch fulfilment.'
>
> —The late Mr Iwan Iwanoff, Fellow of the Royal Australian Institute of Architects



Cat. 37 David Sharp (Australia), ***Self-portrait***, 2007, pencil on paper, 26.0 × 18.0 cm. Collection of the artist.

Alison Silva

I am a self-taught painter, having always been inspired by legends, folklore and dream worlds. The fantastic and peculiar worlds depicted in Lewis Carroll's classic novel, *Alice through the looking glass*, have been my most enduring influence.

As I learn to understand my condition every day, seizures and migraines continue to impact my visual ability and perceptions of reality. Despite this challenge, and also because of it, inspiration to paint is fuelled all the more.

Bearing witness is a collage of nightmares and revelations of life-changing events influenced by my disease and close encounters with death. It explores the connections between science and art, the process of creativity, the fragility of this disease, and the feelings of sudden sensations of fear, anger and joy. In this painting, the March Hare is racing through time and a pestering woodpecker is surgically tearing through my mind, blending my palette of emotions and memories. The crown of thorns denotes my burden, while the Shaman Bird Man connects to the unknown. The Wolf imparts learning, wisdom and access to an animal-self.

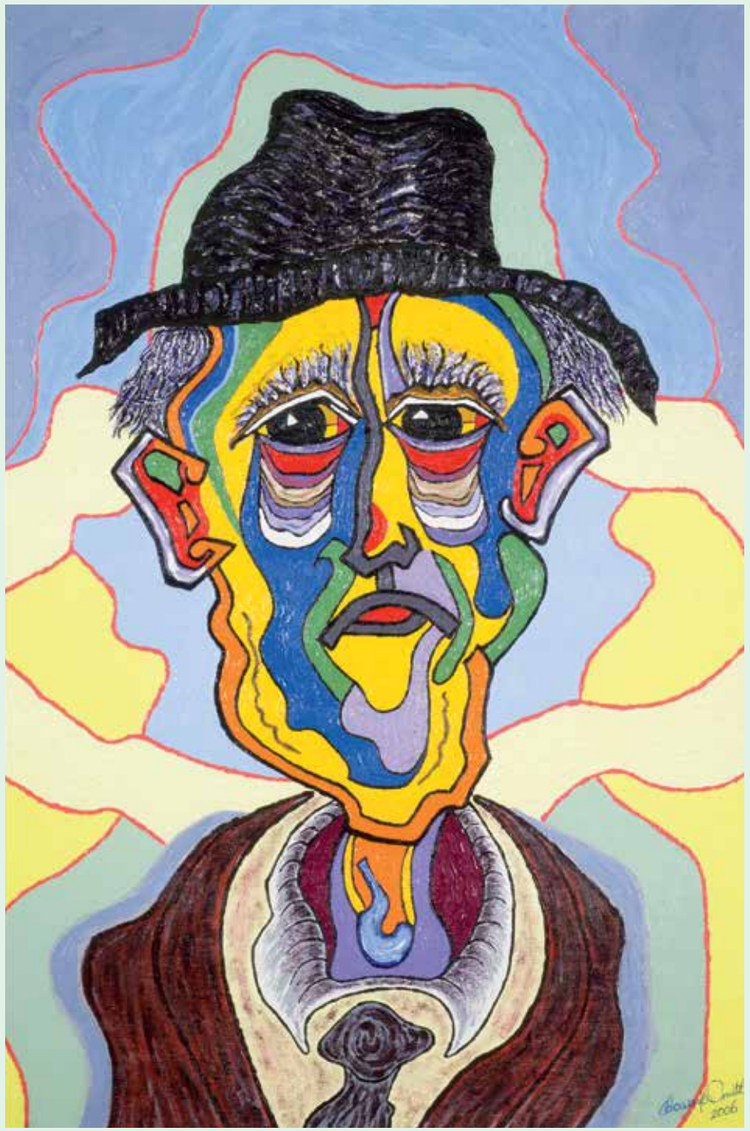
Cat. 38 Alison Silva (USA), ***Bearing witness***, 2010, oil on canvas, 53.0 × 46.0 cm. Collection of the artist.

Howard Smith

This painting is based on a sketch I did in 1987 after coming across yet another 'old timer' on the street in Fremantle, a person who I sensed was indeed 'down and out'. The America's Cup (yacht race) had just been held in Fremantle, and this man was a contrast to the glamour and hype that accompanied the event, and there were many like him. At the time I was living in a small rented flat in the middle of the town and experiencing frequent complex partial seizures. My drawing books were my diaries in which I documented the life around me.

The sketch was put into a drawing file and stayed there for about 20 years. Looking through my files one day in Geelong in 2006, this sketch immediately brought back to me the old man and the feeling I had when seeing him and I decided to use it as the basis for a painting. I had undergone surgery for my epilepsy in between the drawing and the painting, and as part of my 'healing' had undertaken an art course. This was a turning point for me, as it gave me the confidence and opportunity to take my drawings to another stage and visually tackle my epilepsy.

With *Old timer—down and out in Fremantle*, I wanted to try to capture with strong colour and simple but unsettling line-work the fact that this man is old and has nowhere to go. I wanted to create an image that would make viewers search within themselves, and both sense and feel the life of this individual. I felt a connectedness to him, to his inward gaze and to his sense of loss, while being highly aware of the fact that I had support and security, while in my mind he had neither of these.



Cat. 41 Howard Smith (Australia), ***Old timer—down and out in Fremantle***, 2006, acrylic on canvas, 105.0 × 79.0 cm. Collection of the artist.

Peter Walker

I first encountered epilepsy in 1970 whilst working as an artist in London. I was on the phone to my flatmate when I felt decidedly unwell, thinking I was having a brain aneurysm. I was 21. My mind seemed to tune in and out when talking to people, sometimes forcing me to concentrate so hard to take in what they are saying.

One constant throughout my life is that I appear vague to some people, along with a tendency to stare. During a period of about 18 months I suffered from panic attacks, in parallel with epilepsy, almost freezing at times—especially when walking—thinking people were looking at me. With my epilepsy I tend to get an aura followed by a seizure, then feel heavy and despondent for a couple of days. I was anxious in those two days of recovery not to get what I call 'aftershocks', metaphorically similar to that from an earthquake.

Because my epilepsy started at a similar time as my art career, I paint what I call the 'human condition'. For me I can't think of any other subject that is so interesting as the human being to investigate in my practice as an artist. Art is my work, my knowing, my confidence, my passion and my way of communicating. Together my art and epilepsy dance side by side.

Like birds dance as an expression of their courtship love, so human beings court each other through dance too. Bringing together bird and human in a common theme of dance is what this picture is about. It was inspired, in part, by 'Dance me to the end of love' by Leonard Cohen, on his live album *Live in London*. Another line in the same song is 'Dance me to the children who are asking to be born'.

Cat. 46 Peter Walker (Australia), ***The dance***, 2007, mixed media on canvas, 80.0 × 100.00 cm. Collection of the artist.

Works in the exhibition

ARTWORKS
Collection of the artist, unless otherwise stated. All publication rights reserved by the artists.

1 Sharon Anderson (Australia)
All seeing eye, 2008
pastel on paper
27.5 × 38.0 cm (image)
(see p. 105)

2 Sharon Anderson (Australia)
Created mind, 2009
pastel on paper
50.0 × 52.0 cm
(see p. 107)

3 Sharon Anderson (Australia)
Test for Creative Thinking—Drawing Production (TCT—DP)
2009
pen on paper
15.0 × 14.5 cm (image)
(see p. 105)

4 Patricia Bernard (USA)
Just pulling me in, 2012
acrylic and ink on canvas
50.0 × 40.0 cm
Jim Chambliss Collection
(see p. 109)

5 Nadine Binder (Australia)
FACET, 2006
pen on paper
21.0 × 14.8 cm
JTA Australia Collection
(see p. 111)

6 Marea Breisch (Australia)
Orange nude, 2005
oil on canvas
112.0 × 87.0 cm
(see p. 113)

7 Emma Brockett (Australia)
Linear confusion, 2003
mixed media on paper
101.0 × 63.0 cm
(see p. 115)

8 Vincent Buchinsky (USA)
Windy, 2013
mixed media with bronze patina
55.9 × 20.3 × 12.7 cm
Jim Chambliss Collection

9 Dianne Burnett (Australia)
A click to music, 2008
mixed media
51.0 × 41.0 cm

10 Jim Chambliss (USA/Australia)
Discovering the source, 2005
ceramic
38.5 × 45.0 × 45.0 cm
Dr Steven Schachter Collection, Harvard Medical School
(see p. 117)

11 Stacey Crawford (Australia)
Broken bus stop, 2003
glass on paper
12.0 × 8.0 cm

12 Vicki Deutsch (USA)
Fear of fear
reproduced on cover of *International Epilepsy News*, issue 1, 2009
print on paper
22.3 × 18.5 cm (image)
29.5 × 20.7 cm (cover)
Jim Chambliss Collection
(see p. 119)

13 Myron Dyal (USA)
Face out of the wall, 2006
oil on canvas
97.0 × 66.0 cm
(see p. 121)

14 Tremain Farrar (USA)
Ascension no. 3, 2010
charcoal on paper
73.0 × 110.0 cm
(see p. 123)

15 Ryan Fletcher (Australia)
Sacrificing service, 2011
acrylic on canvas
60.0 × 45.0 cm
(see p. 125)

16 Craig Getzlaff (USA)
Dandelion drowning in the rain
2006
oil on canvas
60.0 × 62.0 cm
(see p. 163)

17 Peter Goodman (Australia)
Epilepsy, 2000
pen on paper
43.0 × 31.0 cm
(see p. 127)

18 Peter Goodman (Australia)
Blood Bank, 2001
pen on paper
43.0 × 31.0 cm
(see p. 127)

19 Cinders Gott (USA)
Primordial womb, 2007
ink and watercolour on paper
24.0 × 14.0 cm
(see p. 129)

20 Cinders Gott (USA)
Test for Creative Thinking—Drawing Production (TCT—DP)
2008
pen on paper
15.0 × 14.5 cm (image)
(see p. 101)

21 Sylvia Heuge de Seville (New Zealand)
For those we loved, 2010
mixed media on canvas
110.0 × 152.0 cm
(see p. 131)

22 Cheryl Heuston (USA)
Drawing #435, 2009
pen on paper
42.0 × 49.0 cm

23 Cathy Hozack (USA)
Strong starlight, 2008
acrylic and watercolour on canvas
60.0 × 74.0 cm

24 Sherion Jones (USA)
Mystified, 2008
mixed media
57.0 × 47.0 cm
(see p. 133)

25 Maggie Keegan (Australia)
Temporal zone 1, 2008
ink and watercolour on paper
60.0 × 48.0 cm
(see p. 135)

26 Serene Low (Malaysia)
Epilepsy and migraine, 2008
acrylic on canvas
74.0 × 56.0 cm
(see p. 137)

27 Paul McCall (USA)
Aura, 2006
pen and coloured pencil on paper
17.0 × 27.5 cm

28 Jessica Merrell (USA)
Untitled, 2006
lithograph on paper
87.0 × 76.0 cm
(see front cover and p. 58)

29 Debbie Motsinger (USA)
Ms. Hatchet, 2008
petals on ceramic tile
10.0 × 10.0 cm (irregular)

30 Shea O'Keefe (Australia)
A time for rest, 2004
pen and pencil on monoprint
48.5 × 48.0 cm
UCB Collection
(see p. viii)

31 Dotty Pedi (USA)
Wandering thoughts, 1998
ink and watercolour on paper
65.0 × 53.0 cm
Jim Chambliss Collection
(see p. 139)

32 Terry Porter (USA)
Adirondack chair, 2010
ink on paper
52.0 × 62.0 cm

33 Fiona Pringle (Australia)
Contemplation, 2001
ink and watercolour on paper
44.0 × 65.0 cm
(see p. 141)

34 Matt Rees (Australia)
Forgotten lands, 1997
pencil and pastel on paper
76.0 × 96.0 cm
(see p. 143)

35 Jude Rouslin (USA)
Partially where? 2005
oil on canvas
50.0 × 40.0 cm
Jim Chambliss Collection
(see p. 145)

36 Janet Y Shagam (USA)
Loon Lake, 2006
etching on paper
92.0 × 74.0 cm
(see p. 147)

37 David Sharp (Australia)
Self-portrait, 2007
pencil on paper
26.0 × 18.0 cm
(see p. 149)

38 Alison Silva (USA)
Bearing witness, 2010
oil on canvas
53.0 × 46.0 cm
(see p. 151)

39 Howard Smith (Australia)
'Big Boom' McGann, 2000
pastel, pencil on paper
30.0 × 42.0 cm

40 Howard Smith (Australia)
Blues man, 2000
pastel, pencil on paper
30.0 × 42.0 cm

41 Howard Smith (Australia)
Old timer—down and out in Fremantle, 2006
acrylic on canvas
105.0 × 79.0 cm
(see p. 153)

42 Howard Smith (Australia)
Experimental drawing 1, 2010
pen on paper
30.0 × 21.0 cm
(see p. 93)

43 Howard Smith (Australia)
Experimental drawing 2, 2010
pen on paper
30.0 × 21.0 cm
(see p. 93)

44 Howard Smith (Australia)
Experimental drawing 3, 2010
pen on paper
30.0 × 21.0 cm
(see p. 93)

45 David Thinger (USA)
Emotional model, 1997
oil on board
52.0 × 44.0 cm

46 Peter Walker (Australia)
The dance, 2007
mixed media on canvas
80.0 × 100.00 cm
(see p. 155)

BUNDANON TRUST COLLECTION
All works by Merric Boyd
(Australia, 1888–1959)

47 ***Tree***, n.d.
pencil on paper
25.5 × 20.0 cm
95-0687-001-01

48 ***Tree***, n.d.
pencil on paper
25.5 × 19.7 cm
95-0588-001-01
(see p. 75)

49 ***Vase with tree design***, n.d.
pencil on paper
25.0 × 17.7 cm
95-0242-001-01

50 ***Blue tree***, n.d.
pencil on paper
23.1 × 17.6 cm
95-0349-001-01

51 ***A blue gum tree***, n.d.
pencil on paper
24.7 × 18.0 cm
95-0908-001-01

52 ***A kelpie dog and a boundary rider***, n.d.
pencil on paper
24.3 × 18.0 cm
95-0295-001-01

53 ***Sheep and lambmm***, ***sheep dog***
1949
pencil on paper
15.8 × 20.8 cm
95-0041-001-01

54 ***A local farm milk moo cow***, n.d.
pencil on paper
24.9 × 19.5 cm
95-0419-001-01

55 ***At a sheep station, morning tea, jackerooh, Conscious memory***, n.d.
pencil on paper
24.5 × 17.8 cm
95-0275-001-01

56 ***Shearer***, n.d.
pencil on paper
24.5 × 17.8 cm
95-0337-001-01

57 ***Pussy cat***, 1948
pencil on paper
27.3 × 18.0 cm
95-0438-001-01

58 ***A red parrot in Australia, a tourists bird country***, n.d.
pencil on paper
24.8 × 19.8 cm
95-0420-001-01

59 ***Peter our doggie***, n.d.
pencil on paper
25.8 × 20.8 cm
95-0733-001-01

60 ***A kanGaroough in Australia, a genuine moveing attitaude***, n.d.
pencil on paper
24.8 × 19.4 cm
95-0421-001-01

61 ***Kookaburrah***, n.d.
pencil on paper
18.4 × 25.3 cm
95-0844-001-01

62 ***Truth holds me***, n.d.
pencil on paper
31.3 × 24.5 cm
95-0509-001-01
(see p. v)

63 ***Mummy***, 1951
pencil on paper
17.8 × 24.8 cm
95-0286-001-01

64 ***Yvonne, takes love the present life's revealed***, 1951
pencil on paper
24.7 × 18.2 cm
95-0008-001-01

65 ***Arthur our eldest potter, by Doris and Merric Boyd***, n.d.
pencil on paper
29.3 × 22.5 cm
95-0487-001-01

EPILEPSY FOUNDATION COLLECTION

66 **Talbot Colony for Epileptics, Clayton**, 1907
photograph
9.0 × 22.0 cm
(see p. 29)

67 **Talbot Colony for Epileptics, billiard room**, 1954
photograph
15.0 × 21.0 cm
(see p. 47)

68 **Talbot Colony for Epileptics, dormitory**, 1907
photograph
17.0 × 21.0 cm
(see p. 29)

69 **Talbot Colony for Epileptics, farm workers**, 1954
photograph
17.0 × 21.0 cm
(see p. 31)

70 **Talbot Colony for Epileptics, school room**, 1907
photograph
18.0 × 23.0 cm
(see p. 44)

71 **Talbot Colony for Epileptics, school room**, 1954
photograph
20.0 × 24.0 cm

72 Epilepsy Victoria
Journal of the Epilepsy Victoria
issue no. 1, June 1980
print on paper
21.0 × 15.0 cm

73 The Talbot Colony for Epileptics, Masonmeadows, Clayton
Thirty-seventh annual report for year ended June 30th 1944
print on paper
21.0 × 14.0 cm
(see p. 169)

74 ***Grapevine***
newsletter of Epilepsy Group, October 1988
print on paper
30.0 × 21.0 cm
(see p. 83)

75 Victorian Bureau for Epilepsy
Expectation
quarterly magazine: issue no. 2, May 1966
print on paper
30.5 × 23.0 cm
(see p. 81)

76 Victorian Bureau for Epilepsy
Expectation
quarterly magazine: issue no. 5, March–April 1967
print on paper
25.0 × 18.0 cm

77 The Rotary Club of Hawthorn
Bulletin
vol. 13. no. 33, 8 March 1966
print on paper
22.0 × 17.0 cm

78 Victorian Bureau for Epilepsy
Minutes of Executive Committee meeting
27 May 1964
print on paper
26.0 × 21.0 cm

79 Victorian Bureau for Epilepsy
Minutes of management meeting
5 September 1966
print on paper
33.5 × 21.0 cm

80 **Scrapbooks of newspaper cuttings on epilepsy**, 1964–68
print on paper (two albums)
24.0 × 26.0 cm

Cat. 124 ***The Yarra Bend & New Lunatic Asylum***, c. 1870–80, albumen silver photograph in leather- and cloth-bound album embossed with gold, 14.5 × 19.0 cm. H31510/32, courtesy State Library of Victoria.

81 Reliance
Football protective headgear
2011
synthetic materials
8.0 × 18.0 × 22.0 cm

82 A & D Company Ltd
Scales SJ-1000H, 2009
metal and plastic
11.0 × 23.0 × 24.0 cm

83 Epilepsy Foundation
The epilepsy report
December 2013
print on paper
30.0 × 21.0 cm

84 Epilepsy Foundation
Epilepsy emergency medication administration workshop, 2014
leaflet, print on paper
30.0 × 21.0 cm

85 Epilepsy Foundation
Seizure first aid, 2014
leaflet, print on paper
30.0 × 21.0 cm

86 Epilepsy Foundation
Purple day for epilepsy, March 26, 2014
poster, print on paper
30.0 × 21.0 cm
(see p. xii)

87 Epilepsy Foundation
In touch with epilepsy
newsletter: Autumn 2014
print on paper
30.0 × 21.0 cm

88 Epilepsy Foundation
How epilepsy smart are you? 2012
leaflet, print on paper
15.0 × 21.0 cm

89 Barry Brailsford (text) and Kim Gamble (illustrations)
Our Mummy has epilepsy
Australia: Marrion Merrell Dow, 1994

90 Manning Clark
The quest for grace
Melbourne: Viking, 1990
signed in ink *Manning Clark*

91 Manning Clark
The puzzles of childhood
Melbourne: Penguin Books Australia, 1990
signed in ink *Manning Clark*

92 Gordon Coleman (text) and Kargun Fogarty (Mooj) (illustrations)
Epilepsy with Indigenous Australians
Canberra: Epilepsy Australia, 2002

93 Adele Jackson (illustrations)
Because you are my friend
Epilepsy Association NZ (Inc.), 2001

94 Kate Lambert (text) and Scott Hellier (illustrations)
Can I tell you about my epilepsy? A guide for friends, family and professionals
London: Jessica Kingsley Publishers, 2012

95 Anne Little (text) and Denise McMahon (illustrations)
Poss's school days: An activity based story book for young children
Brisbane: Epilepsy Queensland, 1995

96 Judy Nation, J Helen Cross and Ingrid E Scheffer
Ketocooking: A practical guide to the ketogenic diet
St Albans, Hertfordshire: Homewood Press, 2012

97 CR Yemula and F Besag
My doctor says I have epilepsy: A child's journey
Bedford: Health Insights 4U, 2010

HARRY BROOKES ALLEN MUSEUM OF ANATOMY AND PATHOLOGY, UNIVERSITY OF MELBOURNE

98 **Human brain specimen**
18.5 × 14.0 × 9.0 cm
516-100713
(see p. 55)

MEDICAL HISTORY MUSEUM, UNIVERSITY OF MELBOURNE

99a Chief Secretary's Office
Letter to [Dr] AS Joske Esq, MB JP, appointing him as an Official Visitor of Yarra Bend and Kew lunatic asylums
5 May 1893
ink on paper (handwritten and printed)
33.0 × 21.0 cm
Gift of Dr William Joske
MHM03710.6

99b Chief Secretary's Office
Letter to Dr Joske, to thank him for the report written with Dr Jamieson, which commented on charges of ill-treatment made by WH Wilcock against certain attendants at the Yarra Bend Lunatic Asylum
3 October 1903
ink on paper (handwritten and printed)
33.0 × 21.0 cm
Gift of Dr William Joske
MHM03711.2
(see p. 35)

100 **Phrenology bust**, c. 1850–1914
porcelain, ink, brass, wood
9.5 × 5.0 × 5.0 cm
Gift of Alan Attwood
MHM04392
(see p. ii)

101a **Post-mortem instruments set, in case**, c. 1880
wood, brass, metal
27.4 × 16.4 × 4.6 cm
inscribed on brass plate *Yarra Bend Asylum*
Gift of Alan Kilgour, 2005
MHM04523
(see p. 16)

101b Savigny & Co.
Trephining instrument set, in fitted case, c. 1840
brass, steel, ebony, bristles, wood, paint, varnish and velvet
5.2 × 20.6 × 10.2 cm (case)
MHM00058
(see p. 50)

101c Evans & Wormull
Surgeon's instruments: trephine and trephine handle, c. 1896
brass, silver, nickel-plated wire, other metals, mahogany
10.8 × 2.3 cm (trephine)
10.3 × 2.2 cm (handle)
MHM00072.37 and .38

102 Dr John William Springthorpe
Notes taken at Professor George B Halford's lectures on anatomy, physiology and pathology, 1877
ink, pencil on paper, leather and cardboard
19.5 × 23.4 × 3.0 cm
MHM01047

103a **Dr JW Springthorpe**, c. 1900
photograph
12.0 × 9.8 cm
Courtesy Dr Guy Springthorpe
MHM00674
(see p. 27)

103b **Dr George Adlington Syme** MS FRCS, c. 1900
photographic reproduction on cardboard
14.4 × 10.5 cm
MHM00342

104 Johnstone, O'Shannessy & Co. Limited
Dr James Jamieson, c. 1900
sepia photograph and ink, mounted
16.6 × 11.0 cm
MHM00390

105 Talma Studios (Melbourne)
5th Year Medical Students 1893, 1893
photograph and ink on card
54.2 × 64.6 cm
MHM00511
(see p. x)

106 English
Pharmacy bottle for digitalis, c. 1900
painted glass
20.0 × 7.8 cm diameter
labelled *TR:DIGITAL:*
MHM01155
(see p. 25)

107 English
Pharmacy bottle for chloral hydrate, c. 1900
moulded painted glass and printed paper
17.5 × 6.5 cm diameter
labelled *Chloral Hydrat*
MHM01164
(see p. 25)

108 English
Pharmacy bottle for opium, c. 1900
moulded glass and printed paper
18.3 × 55.8 cm diameter
labelled *LIQ.OPII SED.*
MHM01166
(see p. 25)

109 English
Pharmacy bottle for zinc and valerian, c. 1900
painted glass
14.0 × 5.0 cm diameter
labelled *Zinci Valerian*
MHM01189.15
(see p. 25)

110 English
Topical medication applicator and container, c. 1900
glass, paper (printed), wood, metal and bristle
6.5 × 3.6 cm diameter (jar and stopper); 8.0 × 4.5 cm diameter (container)
labelled *Liquor vesicatorius*
MHM01213
(see p. 25)

111 English
Canister, c. 1900
tin (pressed and painted), and brass
17.7 × 13.5 × 15.0 cm
labelled *Rad. Aconitii*
MHM01250

112 English
Pharmacy bottle for chloroform, c. 1890
painted glass
25.3 × 10.3 cm diameter
labelled *Lin Chlorof*
MHM01582.7
(see p. 25)

113 German
Lidded jar, early 19th century
glazed earthenware with underglaze painting
21.5 × 11.9 cm diameter
labelled *S Nitr: Dep:*
MHM01724
(see p. 42)

114 French
Jar for cassia pulp, 19th century
porcelain
26.0 × 13.2 cm diameter (incl. lid)
inscribed *PULPA / CASSIAE*; inscribed on base *GL* (monogram)
Gift of the estate of Graham Roseby, 2009
MHM2009.5
(see p. 69)

115 French
Jar for benzoin, 19th century
porcelain
25.8 × 13.3 cm diameter (incl. lid)
inscribed *BENZOIN*; inscribed on base *MADE IN FRANCE / GW* (monogram)
Gift of the estate of Graham Roseby, 2009
MHM2009.41
(see p. 69)

116 French
Jar for extract of valerian, 19th century
porcelain
25.8 × 13.2 cm diameter (incl. lid)
inscribed *EXT / VALEA*; inscribed on base *GW* (monogram)
Gift of the estate of Graham Roseby, 2009
MHM2009.40
(see p. 69 and back cover)

117 Italian
Spouted jar for elderflower water, late 16th – early 17th century
glazed earthenware with underglaze painting
24.0 × 17.5 × 22.8 cm
inscribed *AQa.D.FIOR.D.SANBVCO*
Gift of the estate of Graham Roseby, 2009

118 Italian
Majolica drug jar for essence of *Aconitum napellus* (monk's hood or wolf's bane), late 18th – 19th century
earthenware
14.3 × 11.8 cm diameter
inscribed *ES. ACCONito NAPElli*
Gift of the estate of Graham Roseby, 2009
MHM2009.42
(see p. vi)

OAKLEIGH AND DISTRICT HISTORICAL SOCIETY

119 **Talbot Colony for Epileptics**, 1960
photograph
20.5 × 30.0 cm

120 **Construction of Monash University** (buildings of Talbot Colony for Epileptics in the background), 1960
photograph
20.5 × 30.0 cm
(see p. 33)

STATE LIBRARY OF VICTORIA

121 ***Awfully sudden death in a railway train***
wood engraving in *Police News*
30 June 1877
PN30/06/77/00
(see p. 71)

122 ***Lady Margaret Talbot***, 1908
photograph mounted on card
25.0 × 15.0 cm
H33057
(see p. 167)

123 Frederick Grosse (engraver)
The Yarra Bend Asylum for the Insane
wood engraving in *The Illustrated Australian News*, 23 May 1868
IAN23/05/68/12
(see p. 19)

124 ***The Yarra Bend & New Lunatic Asylum***, c. 1870–80
albumen silver photograph in leather- and cloth-bound album embossed with gold
14.5 × 19.0 cm
H31510/32
(see p. 159)

ST VINCENT'S HOSPITAL ARCHIVES AND HERITAGE CENTRE

125 **Casebook of George Adlington Syme**, 1893–96
leather-bound register
31.7 × 14.0 cm
(see p. 73)

126 **EEG machine in use**, 1955
photograph
18.5 × 24.7 cm
Clinical Photography Department Collection
(see p. 77)

127 **CT machine**, 1976
photograph
15.5 × 20.5 cm
Clinical Photography Department Collection
(see p. 79)

128 Both Equipment Pty Ltd
Type BST electroconvulsive therapy machine, c. 1955
ECT machine, in vinyl-covered case with leather handle and fittings of metal, glass and plastic
20.6 × 33.0 × 27.0 cm
Department of Psychiatry Collection

129 Siemens
Konvulsator 2077S electroconvulsive therapy machine, c. 1978
ECT machine, in metal case with flexible rubberised handle and fittings of metal and plastic
19.5 × 22.5 × 33.0 cm
Department of Psychiatry Collection

130 Simon Vogrin
Current density source reconstruction of epileptiform discharges, 2010
print on paper
11.0 × 19.0 cm (three items)

WALTER AND ELIZA HALL INSTITUTE ARCHIVES

131 FW FitzSimons
Snake venoms: Their therapeutic uses and possibilities, 24 July 1929
book: print on paper
22.0 × 14.0 cm
inscribed in ink on cover *14/10/29*
WEHA00048

132 SH Minogue (Medical Superintendent, Mental Hospital, Stockton)
Letter to Dr Kellaway (Director, Walter and Eliza Hall Institute)
31 January 1935
ink on paper
20.0 × 13.0 cm (two pages)
(see p. 87)

133 SH Minogue (Medical Superintendent, Mental Hospital, Stockton)
Letter to Dr Kellaway (Director, Walter and Eliza Hall Institute) seeking advice on treatment of epilepsy by snake venom
23 January 1935
print on paper
34.0 × 20.0 cm (two pages)
inscribed by hand in ink *Referred by Dr Kellaway to Dr H. Maudsley for comment*

134 TG Gray (Director-General, Mental Hospitals Department, Wellington)
Letter to Dr Dunston (Inspector-General Mental Hospitals Dept. Cape Town, South Africa), concerning Mr FitzSimons' claim for the curative properties of snake venom for treatment of epilepsy
9 May 1930
print on paper
34.0 × 20.0 cm (two pages)

PRIVATE COLLECTIONS

135 Merric Boyd (Australian, 1888–1959)
Vase, 1927
earthenware and glaze
height: 11.0 × 12.0 × 9.5 cm
incised on base: *M.Boyd 1927*
Private collection

136 Merric Boyd (Australian, 1888–1959)
Jug, 1947
earthenware and glaze
height: 12.0 × 15.0 × 10.0 cm
incised on base: *Truth / Love from all here / You both / love Merric Boyd 1947*
Private collection

137 St John Ambulance Association
First aid to the injured, 1951
booklet: print on paper
13.0 × 10.0 cm
Pauline Brockett Collection

Cat. 16 Craig Getzlaff (USA), ***Dandelion drowning in the rain***, 2006, oil on canvas, 60.0 × 62.0 cm. Collection of the artist.

Authors

Professor Sam Berkovic, AC, MD, FAA, FRACP, FRS, is Laureate Professor in the Department of Medicine, University of Melbourne, and Director of the Epilepsy Research Centre at Austin Health. He is an adult neurologist and clinical researcher and heads a large Australian Program Grant integrating clinical, genetic, imaging and physiological studies in epilepsy. His main focus is genetics of epilepsy but he also works on new-onset epilepsy, surgical treatment of epilepsy and neuroimaging in epilepsy. He was elected a Fellow of the Royal Society in 2007.

Professor James D Best, MBBS, MD, FRACP, FRCPath, FRC, HonMD *St Andrews*, is Head of the Melbourne Medical School. After graduating from the University of Melbourne he trained in endocrinology and diabetes research, and worked as an endocrinologist at St Vincent's Hospital. He joined the university staff as Deputy Head of the Department of Medicine (St Vincent's Hospital) and in 1999 was appointed Professor of Medicine and Head of Department. He is on the National Health and Medical Research Council and chaired its Research Committee (2006–12). In 2015 he will take up the position of Dean of the Lee Kong Chian School of Medicine in Singapore.

Professor Peter F Bladin, AO, MD, BS, BSc, FRACP, FRCPEd, was Director of Neurology at the Austin Hospital's Comprehensive Epilepsy Program. He is a Professorial Fellow in the Department of Medicine at the University of Melbourne, former President of the Epilepsy Society of Australia and of the National Epilepsy Association Australia, and Ambassador for Epilepsy with the International League Against Epilepsy. He has published extensively on the history of epilepsy.

Professor Edward Byrne, AC, DSc, MD, FRACP, FRCPE, FRCP, FTSE, trained in medicine in Tasmania and in Adelaide, then as a clinical neurologist at the Institute of Neurology in London. He became Professor of Neurology and of Experimental Neurology at the University of Melbourne; Dean of Medicine, Nursing and Health Sciences at Monash University; Vice-Provost Health and Executive Dean of Medicine at University College London; Vice-Chancellor at Monash University and will soon commence as President of King's College London. He has published widely in medical research and has authored three books of poetry.

Professor Gregory D Cascino, MD, FAAN, is the Whitney MacMillan Jr Professor of Neuroscience at the Mayo Clinic College of Medicine, and Chair of the Division of Epilepsy at the Mayo Clinic in Rochester, Minnesota.

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Dr Jim Chambliss, BA, JD, MA, PhD, completed his PhD in creative arts and medicine at the University of Melbourne. He is currently lecturing in arts and medicine at Melbourne Medical School. He is also doing a pilot study on the value of art in educational programs to improve understanding of medical conditions and increase human empathy. His website www.artandepilepsy.com features the visual art of more than 100 talented people who have epilepsy or experience seizures. He is currently promoting what he hopes will be an international tour of the art of people with epilepsy, and is writing a book on the topic.

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Professor Patricia Desmond, BSc, MSc, MBBS, MD, is Edgar Rouse Professor and Head of the University of Melbourne Department of Radiology, and Director of the Department of Imaging at the Royal Melbourne Hospital. She qualified MBBS at the University of Melbourne in 1984, after obtaining her BSc and MSc in physics. In 2005 she received her Doctor of Medicine for studying MRI in acute cerebral ischaemia. Desmond is a well-established neuroradiologist specialising in neuroimaging, a field in which she has more than 100 publications and two book chapters. Her areas of research interest are neuroimaging in stroke, brain tumours, dementia and epilepsy.

Associate Professor Wendyl D'Souza, MBChB, MPH, FRACP, PhD, has an appointment in neuro-epidemiology and health services research in the Department of Medicine, St Vincent's Hospital, University of Melbourne. He is also a consultant neurologist at St Vincent's with a role in epilepsy services and teaching. He is a current member of the International League Against Epilepsy Epidemiology Commission.

Dr Jacqueline Healy, BA(Hons), MBA, PhD, is the Curator of the Medical History Museum, University of Melbourne. She was the inaugural Director of Bundoora Homestead Art Centre, the public art gallery of the City of Darebin, from 2002 to 2011. Previous positions include Director of the Museum and Art Gallery of the Northern Territory and Director, Public Programs, National Gallery of Victoria.

Dr Peter Hobbins, BA, BSc(Hons), MMedicalHum, PhD, is a historian of science and medicine at the University of Sydney. Having published on the growth of biomedical research, he has recently completed his PhD on venomous creatures in colonial Australia. Peter's current project focuses on the history and archaeology of quarantine.

Professor Patrick Kwan, BMedSci(Hons), MB, BChir, PhD, FRCP, is Professor of Neurology at the Department of Medicine of the University of Melbourne, and Head of the Comprehensive Epilepsy Program and a consultant neurologist at the Royal Melbourne Hospital. He chairs the Commission of Medical Therapies of the International League Against Epilepsy.

Jeremy Maxwell, FFIA, CFRE, is General Manager of Fundraising at the Epilepsy Foundation, where he has worked for over 18 years. He has a family connection to epilepsy and over the years has known many of the people who have been influential in the epilepsy sector, including Mary Davis, one of the founders of the Epilepsy Foundation of Victoria.

Professor Terence J O'Brien, MBBS, MD, FRACP, is the James Stewart Chair of Medicine and Head of the Department of Medicine, Royal Melbourne Hospital, University of Melbourne, and a consultant neurologist. He has expertise in epilepsy, neurotrauma and related conditions, neuropharmacology and *in vivo* imaging. He leads a large translational research team undertaking basic studies involving animal models as well as a wide range of clinical research related to epilepsy, neurotrauma and neuropsychiatric disorders.

Dr Christopher Plummer, BMedSci, MBBS, PhD, FRACP, is Consultant Neurologist at St Vincent's Hospital, Melbourne, with clinical interests in epilepsy and multiple sclerosis. He holds a National Health and Medical Research Council post-doctoral fellowship position at Swinburne University of Technology, researching the role of combined EEG/MEG (electroencephalography and magnetoencephalography) for patients with medically refractory focal epilepsy.

Professor Ingrid Scheffer, AO, MBBS, PhD, FRACP, FAA, is a paediatric neurologist at the University of Melbourne, the Austin Hospital, Royal Children's Hospital and Florey Institute. She is Director of Paediatrics at Austin Health. She recently led the first

major reclassification of the epilepsies in two decades as Chair of the International League Against Epilepsy Commission for Classification. Her interests also include the genetics of autism and speech disorders. She has received many awards including the L'Oréal-UNESCO Women in Science Laureate for the Asia-Pacific region for 2012.

Colin Smith, DipT(Prim), GDSE, has, since 1988, been researching the history of Merric Boyd and his family in Murrumbeena and their relationship to their local community. He shares his extensive knowledge through talks to community organisations and undertakes regular art-trail tours in Murrumbeena. His book *Merric Boyd: The life of an artist in a time and a place* was published in 2013.

Professor Stephen K Smith, DSc, FRCOG, FmedSci, is Dean of the Faculty of Medicine, Dentistry and Health Sciences at the University of Melbourne. Prior to this, he was Vice-President (Research) at the Nanyang Technological University, which led the review of a Singapore Stratified Medicine effort focused on diabetes and oncology. A gynaecologist by training, he has published over 230 papers on reproductive medicine and cancer. He was awarded his Doctor of Science in 2001 for his work in Cambridge on the complex gene pathways that regulate the growth of blood vessels in reproductive tissue. Professor Smith led the formation of the United Kingdom's first academic health science centre and its integration with Imperial College London (ICL). He was Principal of the Faculty of Medicine at ICL and had been Chief Executive of Imperial College Healthcare National Health Service Trust since its inception, the largest such trust in the UK.

Professor Chong-Tin Tan, MBBS, FRCP, MD, FASc, is Head of the Neurology Division, University of Malaya. He has initiated, and builds, many neurology institutions in Asia. He is past Vice-President of the International League Against Epilepsy, and is founder and Editor-in-Chief of the journal *Neurology Asia*.

Dr E Bruce Tomlinson, MBBS, FRACP, trained in neurology at St Vincent's Hospital in Melbourne, Harvard University, Boston University and Queen Square, London. He is currently a neurologist at St Vincent's Hospital and the Northern Hospital.

Professor Frank Vajda, AM, Officer Polar Star (Sweden), MBBS, MD, FRCP, FRACP, is a consultant neurologist, neuropharmacologist and Professorial Fellow at the University of Melbourne, Director of the Australian Pregnancy Register, Member of Central Project Commission of EURAP (International Registry of Antiepileptic Drugs and Pregnancy), and former President of the Epilepsy Society of Australia. He is co-editor of *Textbook on clinical pharmacology of antiepileptic drugs*.

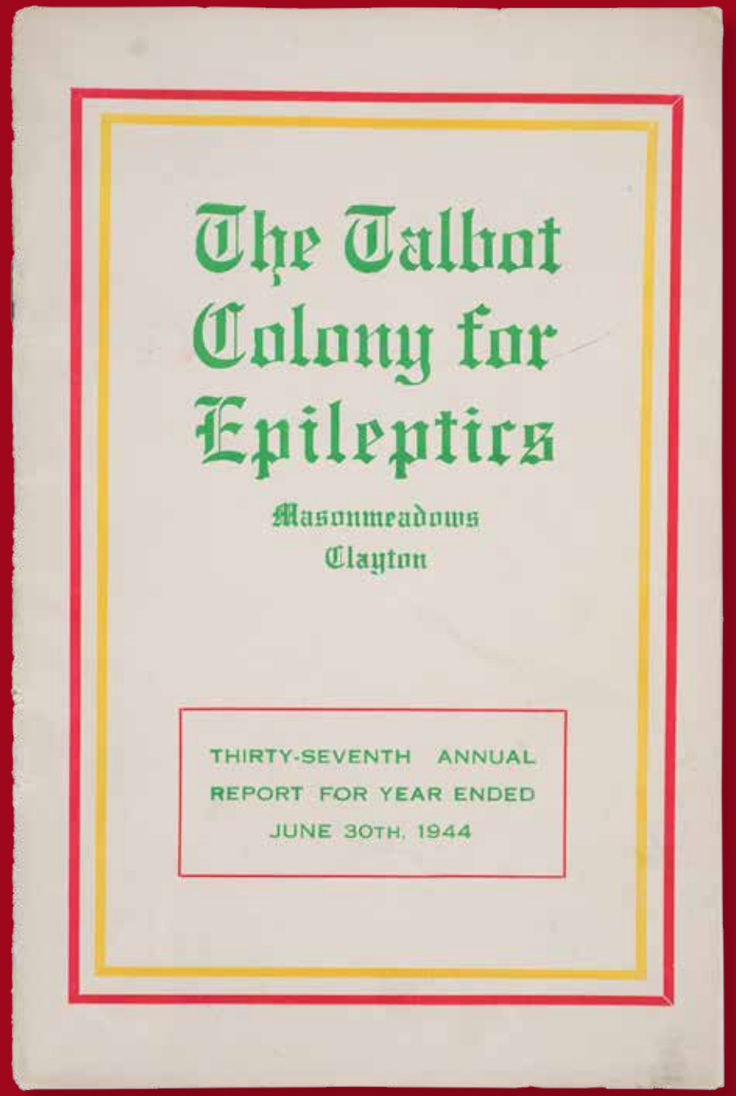
Dr Christine Walker, PhD, is a sociologist and the Executive Officer of the Chronic Illness Alliance, which aims to build a better focus in health policy and health services for all people with chronic illnesses. She is a board member of the Epilepsy Foundation and Treasurer of Epilepsy Australia, a director of NPS Medicinewise and Consumers Health Forum. She manages the Longitudinal Study on the Social Impact of Epilepsy on behalf of the Epilepsy Foundation and has published papers and reports from this research, as well as editing several books on social aspects of epilepsy.

Cat. 122 ***Lady Margaret Talbot***, 1908, photograph on card, 25.0 × 15.0 cm. H33057, courtesy State Library of Victoria.

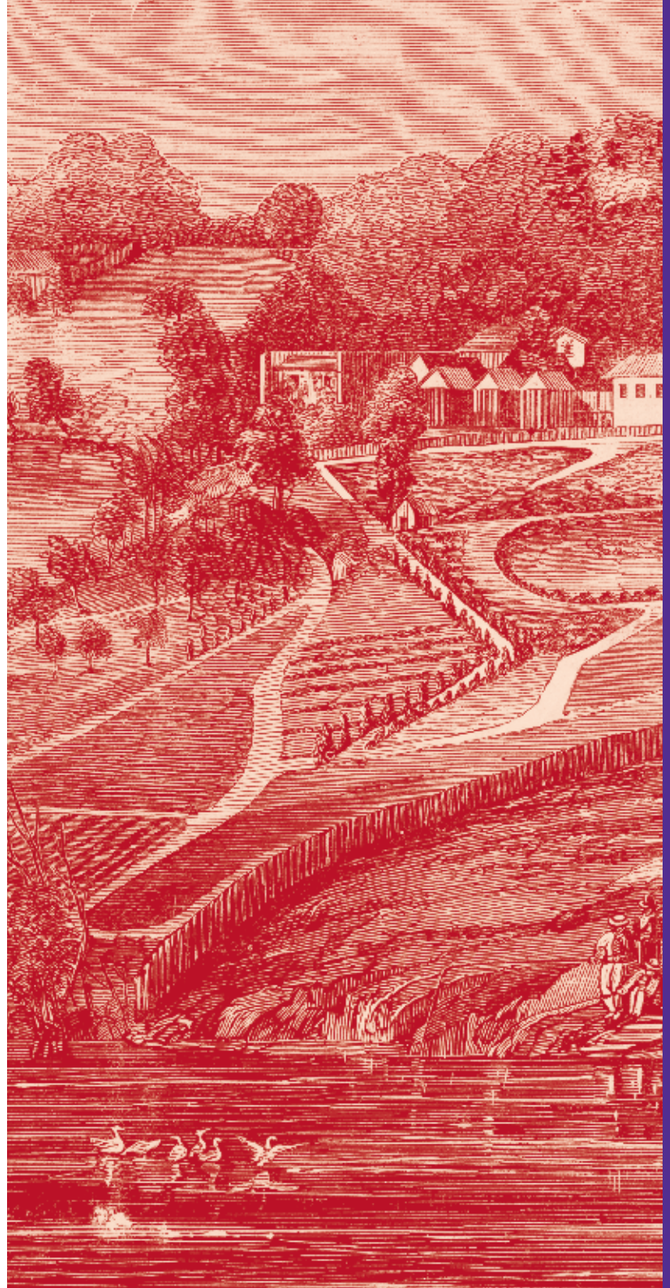
Glossary

absence seizure—a seizure that causes a brief loss of awareness. The person stops any activity and stares blankly, sometimes blinking occasionally. They are more common in children and can be so subtle they sometimes go undetected. Previously called a petit mal seizure.
AED—anti-epileptic drug.
aetiology—the various factors that come together to cause an illness.
amygdala—an almond-shaped group of nuclei deep in the brain. It is important for memory processing, decision making and emotional reactions.
anticonvulsant—an anti-epileptic drug.
atonic seizure—a seizure that causes a sudden loss of muscle tone or strength, particularly in the arms and legs. The person's eyelids might droop, their head may nod, they might drop things or suddenly fall down.
aura—a warning or initial symptom at the beginning of a seizure, experienced by the person with epilepsy, but not visible to observers. Auras might progress to become focal or even generalised seizures, or they might exist alone. Not all people with epilepsy experience auras.
cerebral cortex—the outermost layer of the brain.
clonic seizure—a seizure with repetitive, rhythmic jerks that involve all or part of the body.
complex partial seizure—obsolete term for *focal dyscognitive seizure*.
corpus callosotomy—an operation that cuts the corpus callosum (a band of nerve fibres located deep in the brain, connecting the two hemispheres of the brain), to interrupt the spread of seizures from one hemisphere to the other. Callosotomies might be complete or might involve only a portion of the corpus callosum. Although seizures generally do not completely stop after this procedure, they usually become less severe.
craniotomy—temporary removal of part of the skull.
EEG-video monitoring—continuous simultaneous recording of brain waves using *electroencephalogram (EEG)* and video observation of the person's behaviour. This technique, carried out at comprehensive epilepsy centres, is used to diagnose epilepsy and understand where the seizures originate. Results help physicians determine the best therapy, whether medical or surgical, for that person.
electrode—a conductive disc (usually metal) attached to the scalp to convey the brain's electrical activity through a wire to an EEG machine. Typically, 21 electrodes are temporarily pasted to the person's scalp during an *electroencephalogram*.
electroencephalogram (EEG)—a diagnostic test that measures brain waves, the electrical impulses in the *cerebral cortex*. This test helps a physician to diagnose epilepsy.
eloquent cortex—areas of the brain controlling important functions such as vision, movement and speech.
epilepsy—a medical condition marked by recurrent epileptic *seizures*. A person who has a single seizure as a result of fever, medicine withdrawal, or other cause, is not said to have epilepsy if seizures do not recur.
epilepsy surgery—a neurosurgical procedure to prevent further seizures, usually done by removing the *epileptogenic zone*. This is successful in eliminating seizures in a large majority of people with epilepsy, depending on the type of epilepsy identified during *EEG-video monitoring*.
epileptogenic zone—the region of a person's brain responsible for the abnormal electrical signals that cause seizures.
fit—obsolete term for *seizure*.
focal dyscognitive seizure—also called *focal seizure* with impaired consciousness. A seizure that includes loss of awareness: for example, the person seems to be 'out of it' or 'staring into space'. Other movements such as fidgeting, and chewing or swallowing, may occur as part of the seizure, and are caused automatisms.
focal seizure— a seizure that occurs in a limited area in only one hemisphere of the brain. These seizures respond better to surgery than do *generalised seizures*. Previously known as a partial seizure.
gelastic epilepsy—a rare type of epilepsy, usually occurring in children, in which the seizures include laughter, followed by the usual signs of a focal seizure.

Cat. 73 The Talbot Colony for Epileptics, Masonmeadows, Clayton, ***Thirty-seventh annual report for year ended June 30th 1944***, print on paper, 21.0 × 14.0 cm. Epilepsy Foundation Collection.



generalised seizure—a seizure that occurs throughout the brain. The person's body stiffens and jerks, and they may collapse, lose consciousness or become confused. Previously known as grand mal seizure.
glial tumour—a type of tumour of the central nervous system (which includes the brain). Also called a glioma.
grand mal seizure—obsolete term for *generalised seizure* or *tonic-clonic seizure*.
hemianopia—visual field loss.
hemiparesis—weakness on one side of the body.
hemisphere—one half of the brain.
hemispherectomy or **hemispherotomy**—surgery to disconnect all connections between a person's left and right brain, to prevent seizures spreading.
hippocampus—a part of the human brain that plays an important role in memory and spatial navigation. It is often the focus of epileptic seizures.
ictal—occurring during a seizure.
interictal—the periods between seizures. Most people with epilepsy are in an interical state more than 99 per cent of the time.
Jacksonian—based on the discoveries of the English neurologist John Hughlings Jackson (1835–1911), who made seminal contributions to the understanding of epilepsy.
ketogenic diet—a treatment for epilepsy intended to maintain fasting metabolism for a long period in order to create ketones, by-products of fat-burning metabolism. Seizures often lessen or disappear during periods of fasting. The ketogenic diet is very high in fat and low in carbohydrates. It is most often recommended for children aged two through 12 who have been diagnosed with generalised epilepsy that has not responded to a variety of medicines.
lesionectomy—surgery to remove isolated brain lesions that are responsible for seizure activity.
lobe—one of the sections of the cerebrum, the largest part of the brain. The lobes are divided into four paired sections: frontal, parietal, occipital and temporal. The *seizure focus* may be located in one of the lobes.
localisation—in epilepsy, to identify the *seizure focus*.
multiple subpial transection—a surgical procedure to help control seizures that begin in areas of the brain that cannot be safely removed because they control movements or speech. The surgeon makes a series of shallow cuts or 'transections' in the brain tissue to interrupt the movement of seizure impulses.
neurologist—a doctor who specialises in the treatment of disorders of the brain and nervous system, such as epilepsy.
neuron—a single nerve cell. The brain is made up of billions of neurons. Many neurons malfunctioning together can produce a seizure.
parahippocampal gyrus—a grey matter region of the brain surrounding the *hippocampus*.
partial seizure—obsolete term for *focal seizure*.
petit mal seizure—obsolete term for *absence seizure*.
seizure—an event of altered brain function caused by abnormal or excessive electrical discharges in the *cerebral cortex*. Most seizures cause sudden changes in the person's behaviour or motor function. Some non-epileptic events, such as those produced by Tourette's syndrome or heart arrhythmias, resemble a seizure.
seizure focus—the part of the brain where a person's seizures begin.
status epilepticus—a prolonged seizure or a series of repeated seizures without regaining consciousness. Status epilepticus is a medical emergency, and medical help should be obtained immediately.
temporal lobe resection—a surgical procedure in which brain tissue in the temporal *lobe* is cut away (resected) to remove the *seizure focus*.
temporal neocortex—the part of the *cerebral cortex* located near the temporal *lobe*.
tonic-clonic seizure—a seizure combining characteristics of *tonic seizures* and *clonic seizures*. The person's muscles usually stiffen, the person might make a cry or groan, lose consciousness and fall to the floor. Their limbs jerk rapidly and they may lose bowel or bladder control. After a few minutes the person gradually returns to consciousness and may feel drowsy, confused, agitated or depressed. This is the hallmark of a generalised motor seizure, which used to be called a grand mal seizure.
tonic seizure—a seizure characterised by stiffening of the person's muscles, sustained for more than a few seconds.
vagus nerve—a small nerve that passes through the neck and is connected to various areas of the brain.
vagus nerve stimulation (VNS)—a surgical treatment for epilepsy that involves implanting an electrode on the vagus nerve, in the person's neck. The electrode is connected to a pacemaker placed under the skin in the chest. The VNS is programmed to cycle continuously. However, if the person feels a seizure coming on, additional therapy can be given by placing a small magnet over the device.



The Medical History Museum in the Faculty of Medicine, Dentistry and Health Sciences at the University of Melbourne is the oldest and finest collection of its type associated with a medical school in Australia. Established in 1967 by Kenneth Russell, a professor of anatomy, with support from the Wellcome Trust, London, the museum covers the history of the Melbourne Medical School and the broader history of medicine in Australia and overseas.

The purpose of the museum is to encourage, through direct engagement with its collections, appreciation and understanding of the history of medicine and its role in society. The museum stimulates active learning through research, teaching and dialogue among communities of students, faculty, scholars, alumni and the wider public.

Further information on the museum can be found at museum.medicine.unimelb.edu.au.

Front cover: Cat. 28: Jessica Merrell (USA), **Untitled**, 2006 (colour altered), lithograph on paper, 87.0 × 76.0 cm. Collection of the artist.

Back cover: Cat. 116: French, **Jar for extract of valerian**, 19th century, porcelain, 25.8 × 13.2 cm diameter (incl. lid), inscribed *EXT* / *VALEA*; inscribed on base *GW* (monogram). Gift of the estate of Graham Roseby, 2009. MHM2009.40, Medical History Museum, University of Melbourne.

Inside front cover: Cat. 75: Victorian Bureau for Epilepsy, ***Expectation*** (detail, colour altered), quarterly magazine: issue no. 2, May 1966, print on paper, 30.5 × 23.0 cm. Epilepsy Foundation Collection.

Inside back cover: Cat. 123: Frederick Grosse (engraver), ***The Yarra Bend Asylum for the Insane*** (detail, colour altered), wood engraving in *The Illustrated Australian News*, 23 May 1868. IAN23/05/68/12, courtesy State Library of Victoria.


EXT
VALEA