

John Newsom-Davis, 1932–2007

Angela Vincent

John Newsom-Davis, who died in August 2007 aged 74, was an exceptional person, a renowned clinician scientist and a shining example to many neurologists and trainees around the world.

After training as a pilot in the Royal Air Force during his National Service, John studied at Cambridge University, and the Middlesex Hospital, London, UK. He then joined Tom Sears at the National Hospital for Neurology and Neurosurgery, Queen Square, London, to investigate respiratory sensation and control. Later, he studied muscle spindles in human muscle biopsy tissue. After completing his clinical training, John directed the Intensive Therapy Unit at Queen Square for several years, gaining experience of caring for patients with myasthenia gravis (MG). The combination of looking after these patients and experience with muscle biopsy tissue was crucial in determining his future scientific and clinical career.

During the 1970s, Toyka and Drachman showed that MG was caused by loss of muscle acetylcholine receptors (AChRs), and was associated with pathogenic anti-AChR antibodies. Around the same time, John collaborated with Ricardo Miledi at University College London to examine AChR numbers in the muscles of patients with MG. John's first major contribution to this exciting area of research was to show that plasma exchange, which was already being used in Goodpasture's syndrome, also showed efficacy in MG. With my help, he subsequently demonstrated the relationship between AChR antibody levels and clinical severity of MG after treatment.

Grasping the available opportunities with both hands, John convinced me to help establish an MG research group at the Royal Free Hospital, London, where he was also a consultant. We went on to study many aspects of thymic involvement in MG, principally with Nick Willcox who joined us in 1979. Another recruit, Bethan Lang, provided the first definitive evidence that Lambert–Eaton myasthenic syndrome had an

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autoimmune basis, with calcium channels being the autoantibody target. Using similar approaches, Ian Hart identified potassium channel antibodies in acquired neuromyotonia. In 1985, David Beeson joined us to clone human AChR genes and to identify mutations in congenital forms of myasthenia.

In 1987, John was appointed to the Chair of Clinical Neurology at Oxford University, taking most of the Royal Free team with him. The Neurosciences Group in the Weatherall Institute of Molecular Medicine became—and remains—a major UK and European center for neuroimmunology and for the congenital myasthenic syndromes. John twice chaired the Medical Research Council Neurosciences Board, and, as retirement beckoned, he began to take up other activities, first as President of the Association of British Neurologists and then, in 1997, as Editor of *Brain*. He was awarded many prizes and honors, including Fellow of the Royal Society in 1991, and Foreign Membership of the Institute of Medicine of the US National Academy of Sciences in 2001.

As his editorship of *Brain* came to an end, John began to seek new challenges, and along with US myasthenia experts he took on the huge task of organizing and obtaining funding for a multicenter trial of thymectomy involving over 80 participating centers. This trial dominated his working life over the past few years, and it was following a morning visit to a hospital in Bucharest in August 2007 that he and his wife, Rosemary, were involved in a tragic car accident. She is recovering slowly back in the UK.

In many ways, John's clinical and scientific career illustrates how grasping opportunities, as well as ability and hard work, can determine success. He was distinguished by these attributes, combined with youthful enthusiasm, charm, humor, tolerance and capacity for friendship. He will be greatly missed not only by his family, many close friends and UK colleagues, but also by countless neurologists and patients worldwide.

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Competing interests

The author declared no competing interests.

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