John Newsom-Davis, MD (1932-2007)

John Newsom-Davis died in an automobile accident on August 24, 2007, while he was on vacation in Romania. His wife of 44 years, Rosemary, was seriously injured but survived. So ends the career of one of the most productive of all modern research neurologists.

Newsom-Davis came late to medicine; he was delayed because he was piloting jet fighters in the Royal Air Force in 1951 to 1953. In 1954 he entered Cambridge University, where he played varsity hockey; he received a BA degree in 1957. He then enrolled in the Middlesex Hospital Medical School, where he gained his MD in 1966. He was a house physician at the National Hospital for Neurology and Neurosurgery (Queen Square) from 1966 to 1968. After coming to New York for research training with Fred Plum, Newsom-Davis’ first papers were about pulmonary function in health and disease. For the next decade, he was a consultant neurologist at Queen Square and the Royal Free Hospital. In 1980, he was made MRC Clinical Research Professor of Neurology, and in 1987, he became professor of clinical neurology at Oxford. There, with Angela Vincent, he established a myasthenia gravis unit, where he worked for the next 11 years. He continued as professor and honorary consultant until his retirement in 1998.

John Newsom-Davis’ first contribution to myasthenia research, with Anthony Pinching and Keith Peters, showed the dramatic effect of plasmapheresis. Vincent was a coauthor on almost all of Newsom-Davis’ papers on autoimmune diseases, and together they documented the therapeutic value of plasmapheresis and the pathogenic effects of antibody to the acetylcholine receptor. In 1980, with Alastair Compston, they reported that combinations of HLA antigens, clinical findings, and antibody status demonstrated clinical heterogeneity. The next year, they found that the antibodies were formed in the thymus and that antibody receptors were present on muscle-like cells in the thymus. They tackled the question of “seronegative” myasthenia, the patients who lacked anti–AChR in about 15% of all cases. Ultimately they identified antibodies to voltage-gated calcium channels, detectable in more than 90% of patients. They also identified antibodies to voltage-gated potassium channels (VGKCs) in about 40% of patients with peripheral nerve hyperexcitability in the syndrome called neuromyotonia and even in some people with the more common cramp-fasciculation syndrome. The same VGKC antibodies were found in a syndrome of cognitive decline and muscle fasciculation (Morvan syndrome) and also in limbic encephalitis. These syndromes were sometimes but not always paraneoplastic.

Newsom-Davis and his colleagues were leaders in the study of nonimmune congenital forms of myasthenia, demonstrating that they were heterogeneous and not autoimmune. They identified 1 form with proximal limb muscle weakness and autosomal-recessive mutations in the gene for Dok7, resulting in a defective structure of the neuromuscular junction.


His lectures were always memorable. The president of the Australian Association of Neurologists commented, “That was the best lecture in clinical neurology I ever heard—real bonzer.” In Australia, bonzer is a slang adjective meaning extraordinarily first-rate.

To those who had the true privilege of knowing him personally, Dr Newsom-Davis was warm, considerate, thoughtful, and witty. He is survived by his wife, Rosemary, a twin sister, and 3 children.

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