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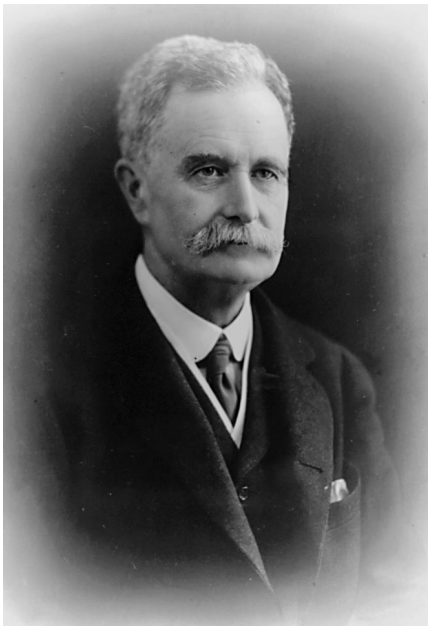
Howard Henry Tooth (1856–1925)



Howard Henry Tooth

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Howard Tooth was born in Hove, Sussex, educated at Rugby School and at St. John's College, Cambridge, where he qualified in 1880 [1–3]. He had his clinical training at St. Bartholomew's Hospital, London, where he started his work on hereditary peroneal muscular atrophy. After further medical appointments he became physician to the London Metropolitan Hospital in 1887. His neurological interest led to his appointments at the Hospital for the Paralysed and Epileptic, Queen Square, as assistant physician in 1887 and physician in 1907, and at St. Bartholomew's Hospital as assistant physician in 1895 and full physician in 1906. Tooth lived at 34 Harley Street until his retirement. His work showed extraordinary patience, sympathy and thoroughness. His work on 'The Growth and Survival Period of Intracranial Tumour' [9] was regarded as the definitive study at the time. He was an expert on the microscopic anatomy of the nervous system.

During the Boer War he served in South Africa, and in the First World War he served with great distinction in London, Malta and Italy, reaching the rank of colonel. He was created CMG in 1901 and CB in 1918. His early success was signalled by the highly prized Goulstonian lectures on 'Secondary Degeneration of the Spinal Cord' at the Royal College of Physicians of London in 1889; he later became Censor to the College. Contemporaries admired his 'never failing cheeriness and good temper'. He was an enthusiastic teacher although despite a love for outdoor activities he never regained his full health after the anxieties of the Great War. Thus, his colleagues believed that his early promise was not wholly fulfilled. As with many physicians in the Victorian age, he was a man of catholic interests. A talented musician and worker with wood and metal, Tooth was also a keen gardener and cyclist. He was married twice, first to Mary Beatrice Price who bore a daughter, then to Helen Katherine Chilver by whom he had a daughter and two sons. He suffered a cerebral haemorrhage whilst driving his car, and died 3 months later at home in Hadleigh, Suffolk.

Tooth is remembered for his doctoral thesis presented to the University of Cambridge in 1886, the text of which was published by HK Lewis [8]. In the same year, Charcot and Pierre Marie in Paris published similar clinical accounts [4], so that the syndrome is justly remembered eponymously as Charcot-Marie-Tooth (CMT) disease. They described a disorder mainly of adolescents and early adult life, characterised by slowly progressive wasting and weakness of the lower legs resulting in 'pied en griffe', with the classical champagne bottle configuration of the legs. Sensory loss was absent or mild, and progression to the hands was a late feature of the disorder. Enlargement or

hypertrophy of the peripheral nerves was apparent in some cases, and pes cavus and many other skeletal deformities were common. Charcot and Marie thought it was a German muscle disease, whereas Tooth correctly suggested a peripheral neuropathy. Later writers recorded variants with benign tremor, pupillary abnormalities, deafness; the cerebrospinal fluid was usually normal. Charcot, Marie, and Tooth acknowledged earlier descriptions of the illness by Eulenburg (1856), Friedreich (1873), Ormerod (1884) and Osler (1880) that were later collected by Schultze who Kinnier Wilson said had described the condition in 1884 [7].

More recently, many variants have been dissected from this pleomorphic syndrome. Its hereditary nature led to the term hereditary motor sensory Neuropathy (HMSN). CMT, or HMSN, is a clinically and genetically heterogeneous condition [5, 6]. The demyelinating form, autosomal dominant HMNS I (CMT 1), has been associated with duplication of, or point mutations in the PMP-22 gene on chromosome 17p11.2 (*CMTIA*) and, less frequently, with mutations of peripheral myelin protein zero (*PMP0*) on chromosome 1q22–23 (*CMTIB*). The axonal or neuronal type (HMSN II or CMT2) is most often linked to loci on chromosome 1p35–36, 3q13–q22 and 7p14, although no mutated gene has yet been identified. Finally, mutations in the *PMP0* gene have been associated with an intermediate form between HMSN I and II, a rare X-linked form, isolated instances of Dejerine-Sottas syndrome (HMSN III), and congenital hypomyelination.

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