6.1. Introduction

Recording the electrical activity of the nervous system in both health and disease has a long and distinguished pedigree going back to Luigi Galvani in 1791\(^1\), whose bold and contentious experiments on animal electricity led to the modern science of electrophysiology. The discovery that electrical activity could be recorded from the animal brain was made a century later in 1875 by Richard Caton, whose feeble currents of the brain were incredibly, discovered at least half a century before the advent of electrical amplification\(^2\). This electrical activity is what we now know as the electroencephalogram (EEG), a term coined by Hans Berger while working on human heads in Jena in the 1920s. Berger was also the first to suggest that the EEG could be used for clinical diagnosis and was the first to record the high amplitude synchronous electrical activity associated with epileptic seizures in the human brain \(^3\). Even at this early state of the art, distinctions could be seen between focal damage to the brain surface and more widespread activity inferred to be projected from subcortical, diencephalic structures. Today, analysis of the EEG is routinely used in the diagnosis of many neurological conditions, often in conjunction with a variety of brain imaging methodologies. In a few cases of focal damage, where the lesion is invisible to current imaging methodologies, detailed EEG analysis is still the only way of securing an initial diagnosis. An excellent account of early electrophysiological
history can be found in Mary Brazier's delightful book, *The Electrical Activity of the Nervous System*[^1].

One area where the EEG plays an essential part of the diagnosis is in cases of sporadic Creutzfeldt-Jakob disease. Classically, the normal EEG patterns gradually disappear and are replaced with widespread bilateral synchronous spike-wave discharges with a periodicity of 1 to 2 second as shown in Figure 6.1. This activity does not constitute a definitive diagnosis, nor does the absence of these periodic discharges necessarily rule out a diagnosis of sCJD, so the use of the EEG must be used in conjunction with other methods of assessment, including an MRI scan and currently, the abnormal appearance of 14-3-3 proteins in the CSF. Interestingly, the appearance of synchronized discharges in the EEG is not a diagnostic feature of new variant CJD and just to complicate matters, the following conditions sometimes also present with similar EEG abnormalities: Alzheimer's disease, multiple cerebral abscesses, metabolic encephalopathy, certain toxic encephalopathies (e.g. lithium), anoxic encephalopathy, progressive multifocal leucoencephalopathy, Lewy body disease[^5].

Moving away from the EEG, many other, often much more sophisticated electrophysiological methods have been used in the study of neurodegeneration. In the field of basal ganglia disease for example, electrophysiology has been crucial in laying a foundation for understanding the basic anatomy and physiology of a complex set of inter-related brain areas with complex and often unexpected interactions. The most important of these discoveries was that the circuits linking the various

[^1]: MacLeod, Johnston and Curtis, *Electrical Activity of the Nervous System*.

[^5]: MacLeod, Johnston and Curtis, *Multi-electrode EEG recorded from a patient with sporadic CJD showing periodic synchronous wave discharges across a wide area of cortex.*

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*Figure 6.1.* Multi-electrode EEG recorded from a patient with sporadic CJD showing periodic synchronous wave discharges across a wide area of cortex.