New Treatment Modalities for Diabetes Mellitus – An Overview

Chair: ***Rosemarie Wright-Pascoe***

New Treatment Modalities for Type 2 Diabetes Mellitus – An Overview with Emphasis on the Role of Glucagon

Luis Mejia-Rivera

Glucagon was discovered in 1922 as a hyperglycaemic factor in the pancreas. During its early history up to 1970, glucagon was shown to increase circulating glucose through stimulating glycogenolysis in the liver. It was also shown to be a constituent of islet non-β cells and to signal through G protein coupled receptors and cyclic adenosine monophosphate (AMP). Furthermore, its chemical characteristics, including amino acid sequence, and its processing from the preproglucagon gene had been established. During the modern research during the last 40 years, glucagon has been established as a key hormone in the regulation of glucose homeostasis, including a key role for the glucose counter regulation to hypoglycaemia and for development of Type 2 diabetes mellitus and, today, glucagon is a potential target for treatment of the disease. Glucagon has also been shown to be a key factor beyond glucose control and is involved in many processes.

This review focusses on the growing body of evidence that glucagon abnormalities contribute significantly to the pathophysiology of diabetes mellitus and on recent efforts to target the glucagon axis as adjunctive therapy to insulin replacement. This lecture review summarizes the more than 90-year history of this important hormone as well as discusses potential future research regarding glucagon. We will discuss the dysregulation of glucagon action in patients with Type 2 diabetes mellitus and examine incretin effects on glucagon regulation and the effects of SGLT-2 inhibitors on glucagon.

Nerve Conduction Studies in Diabetes Mellitus

Daniel Graham

Diabetes mellitus is the most common cause of neuropathy worldwide, producing a wide variety of conditions involving different types of nerves and pathologic mechanisms (*eg* metabolic, ischaemic, immunologic, compressive).

The different forms of diabetic neuropathy can be classified in terms of their anatomical distribution (*eg* proximal/distal, symmetric/asymmetric, focal/multifocal/diffuse), clinical course (*eg* acute/subacute/chronic), characteristic features (painful/non-painful, sensory/motor/autonomic) or pathophysiology. Classification into 'typical' or 'atypical' forms is based on their occurrence, the most typical form being a chronic, distal (length dependent), symmetric polyneuropathy that accounts for about 75% of diabetic neuropathies.

In this presentation, we will briefly review the most common types of diabetic neuropathies, core principles of nerve conduction and needle electromyography (EMG) studies and their role in the electrodiagnosis of diabetic neuropathies. If time allows, a few clinical cases will be discussed.