



**XIV<sup>th</sup> FEDERATIVE INTERNATIONAL  
CONGRESS OF ANATOMY**

**76e CONGRES DE L'ASSOCIATION  
DES ANATOMISTES**

**NEW UNIVERSITY OF LISBON  
FACULTY OF MEDICAL SCIENCES**

Departments of Anatomy and of  
Histology and Embriology

24<sup>th</sup> - 30<sup>th</sup> JULY 1994

LISBON - PORTUGAL

**ABSTRACTS BOOK**

## EXPERIMENTAL DIABETIC VENOPATHIE.

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Elevated glucæmia levels over long periods of time tend to produce premature ageing and modifications in certain tissues above all in the vascular system. Special attention has been given to the study of the modifications produced by this pathological process in the capillary and artery walls. However, the studies of the effects of hyperglucæmia on the vein walls are scarce despite the fact that the physiological behaviour of the vein structural elements differ from those presented by the artery or capillary wall.

Given the scarce data available with respect to the behaviour of the vein wall in diabetes, our aim in this study was to observe the possible existense of ultrastructural modifications in the femoral vein in experimental diabetes.

A total number of 32 two-month old Sprague Dawley rats of both sexes in equal proportions and equivalent weights was used to carry out the study. Diabetes was induced in half of these animals via an intraperitoneal injection of Streptozotocin in doses of 65mg/kg with a 4.5 ph citrate buffer. The rest were injected with the buffer alone and were therefore used as the control group.

Half of the rats were sacrificed six weeks later and the rest twelve weeks later. A section of the common femoral vein was then extracted under stereoscopic microscopy, and prepared for MET. Semi-thin sections were used for the morphometrical studies. Using image analysis (Imagepro) the thickness of wall was calculated, the results were expressed as mean and standard deviations and analysed via a T-Student. Ultra-thin sections were used to analyse the ultrastructural vein wall under a Philips 301 microscope.

The results showed that the three levels of the vein wall had been affected principally in an increase in the deposition of extracellular tissue without any thickening of the wall. The increased deposition was also accompanied by morphofunctional changes in the endothelial, muscular and fibroblast cells which would tend to indicate their contribution towards the production of the aforementioned extracellular tissue.