THE EFFECTS OF ISCHAEMIA

ON

PERIPHERAL MYELINATED NERVE

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SUMMARY

The abnormal resistance of peripheral nerve to ischaemia observed in certain disease conditions - diabetes mellitus, chronic liver disease, motor neurone disease and alloxan-diabetes, has been explained by Seneviratne and Peiris on the basis of a defective periaxonal diffusion barrier. It is thought that in the healthy nerves during ischaemia, the intact barrier serves to accumulate the K^+ leaking from the axon against the axonal surface thus causing a depolarization conduction block. In this study the effect of increasing the K^+ content within the periaxonal space, on nerve excitability have been investigated. The effect of ischaemia on the peripheral nerve of these hyperkalaemic cats has also been studied to ascertain the role of K^+ dynamics in the periaxonal space in the ischaemic inactivation process. In the second part of the study an attempt has been made to identify this barrier structurally and to demonstrate histological changes of this barrier in the diabetic condition. The results of these experiments complement the results obtained from physiological studies on the periaxonal diffusion barrier of diabetic nerve.

From the results of this study it has been concluded that the paranodal gap substance at the nodes of Ranvier constitute the postulated periaxonal diffusion barrier. Further, a reduction in the cation binding capacity of this gap substance in

the nerves of diabetic subjects and alloxan-diabetic rats has been demonstrated. The possible antecedent biochemical changes which lead to the altered staining properties of the gap substance are examined. A correlation between Schwann cell metabolism in segmental demyelination and changes of the gap substance is suggested.