aldehyde-blockade produced by the use of bisulphite after periodate oxidation.

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Effect of Carbon Dioxide Inhalation on the **Blood-Brain Barrier**

THE trypan blue test has been used for studies of damage to the blood-brain barrier caused, for example, by carbon monoxide¹; but so far as can be seen from the literature no investigation has been carried out on the effect of carbon dioxide on the barrier. Histopathological changes after prolonged exposure to carbon dioxide have been reported by Stephens². They consisted mainly of swelling, chromatolysis and vacuolation of nerve cells.

The present experiments were performed on rabbits which were exposed to various concentrations (10, 15, 20 and 30 per cent) of carbon dioxide in oxygen in a gas chamber. The exposure times varied from 2 to 60 min. Ten minutes before the exposure a trypan blue solution was injected intravenously. Ten minutes after the exposure the animals were killed and their vessels were washed out with normal saline followed by a 20 per cent formalin solution for fixation. On serial frozen sections the whole brains were examined for blue stainings and hæmorrhages.

30 per cent carbon dioxide for 2 min. scarcely caused any obvious changes ; but already after 3 min. exposure more consistent changes were seen. After 4, 5 and 10 min. exposure the changes were more pronounced, consisting of marked multiple stainings as well as multiple hæmorrhages. After 15 min. or more the changes often were maximal. Some of these brains were almost totally blue, and there were rich hæmorrhages in the brain substance and the meninges.

In animals exposed to the lower concentrations of carbon dioxide the changes were proportionally less pronounced and occurred only after a somewhat longer exposure. However, still with 10 per cent carbon dioxide and 15 min. exposure clear changes were seen.

The damage to the walls of the blood vessels obviously was reversible within a short time, which was shown by the fact that when trypan blue was injected a few minutes after the gas exposure, the stainings were only slight and the hæmorrhages were unstained.

It may be questioned whether the cause of the barrier damage was a vigorous dilatation of thinwalled vessels or maybe a direct toxic effect on the endothelial membranes. The results indicate that hypercapnia is a factor that should not be disregarded as a cause of cerebral damage, for example, in asphyxia, where the component of anoxia sometimes seems to have been over-emphasized.

A detailed account of this work is to be published elsewhere.

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Nature of Reticulin

FOR a long time there has been confusion in the use of the name 'reticulin' to describe the argyrophilic fibrous structures present as basement membrane in the parenchymatous organs and also to describe fibres which are histologically similar but which are present in developing connective tissue. Recently, however, a number of workers¹⁻³ have shown that the basement membrane type of reticulin is a complex composed of collagen, carbohydrate and lipid which are released only after acid hydrolysis. Windrum et al.³ have questioned the assumption that the two types of reticulin are identical. now report evidence which suggests that they are indeed different structures.

In the early stages of development of a granuloma produced by subcutaneous injection of carrageenin⁴. the fibres present are largely of the reticulin type. Such tissue has been subjected to a differential extraction procedure⁴ and examined histologically after each extraction. The histological examination showed that : (a) after extraction with 0.2 M sodium

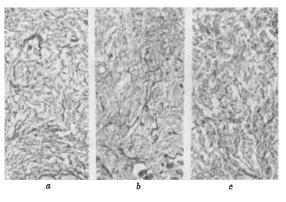


Fig. 1. Carrageenin granuloma after six days. a, Normal control showing abundant silver staining reticulin fibres; b, after neutral salt extraction the fibres lose their argyrophilia and stain red with Yan Gieson; c, extraction with citrate buffer after neutral salt gives almost complete removal of fibres. All stained under identical conditions by silver impregnation/Van Gieson. (× c. 120)