T. hydatigena. The evidence, however, for a similarly shared immunogen between the embryos and cysticerci of the two sheep metacestodes and those of the rabbit metacestode *T. pisiformis* is less certain.

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ANATOMY

Effect of Light Deprivation on the Postnatal Development of the Optic Nerve

Gyllensten and Malmfors[1] examined *inter alia* the cross-sectional area and myelinated fibre distribution of the optic nerve in mice reared in the light or in the dark. In mice reared in complete darkness for 20 and 30 days, delay in myelination of the optic nerve fibres caused a reduction in the number of myelinated fibres of some 12 per cent. The number of larger fibres was affected to a greater extent than the number of smaller ones. At 20 days the cross-sectional area of the optic nerve was reduced by 3·4 per cent and at 30 days it was increased by 0·4 per cent; this was said to be insignificant. This latter finding prompts me to report some preliminary observations and a new experimental technique for light deprivation.

The classic experiments in this field are those of Held[2] (cited by Sattler[3]). "Bei neugeborenen Kaninchen, Katzen und Hunden öffnete er einseitig die Lidspalte und fand bei Untersuchung in polarisiertem Licht, 'einen deutlichen, wenn auch nicht sehr beträchtlichen Unterschied in der Markreife', während ein vollständig im Dunkeln gehaltener Hund, dessen eine Lidspalte vorzeitig geöffnet worden war, keinen Unterschied in der Markentwicklung zwischen beiden Sehnerven erkennen liess." In Held's experiments, since the closed eyelids of the new-born are translucent, the difference in stimulus between the two retinæ was one of degree only. It was thought that occlusion of all light from one eye from the time of birth would give a more clear-cut effect and would permit quantitative assessment of the effect of function on fibre size. Even in these experiments, however, the difference may be one of degree only, as the question of spontaneous activity[4] must be considered.

The occluding shield was made from tantalum foil (0·002–0·003 in.). Circular disks of 5 mm diameter were cut from the sheet and made ovoidal with a specially-shaped punch. Newborn mice were operated on within a few hours of birth. From a midline incision over the sagittal suture the skin was undercut over one eye and a shield was inserted. The shape of the shield kept it in position over the convexity of the eyeball and the incision was closed by holding the wound edges together with toothed forceps for a while. The mice were then returned to a foster mother of a hybrid strain who had littered a day or two earlier. The use of recently-littered or highly-strung pure-strain mice resulted in cannibalism. In experiments conducted so far the unoccluded eye opened on the 12th to 14th day but the lids of the occluded eye usually remained fused and the shield remained in position. The eyelids of control animals (operation but no shield) opened at the normal time. The animals were weaned 10 days later and anæsthetized at 75 days with intraperitoneal 'Nembutal'. The animals with occluded eyes were normal in weight and the eye beneath the shield was normal in size and structure. The calvarium was removed and the brain divided in the mid-coronal plane so that the optic nerves and chiasma were left *in situ* when the rest of the brain was removed. For light microscopy the nerves were prepared as follows: the nerve of the operated side was cut longer than the other for identification; the nerves and chiasma were then removed and processed *en bloc* so that the nerves received identical treatment; they were sandwiched between pieces of liver[5], the operated side being identified by a human hair placed alongside it, and placed in a cell; the cell was frozen in a CO_2–acetone mixture and transverse sections were cut at 7μ in a cryostat[6]. The sections were fixed and stained on a slide with potassium permanganate[5] and mounted in Farrant's medium.

The fresh-frozen section technique is particularly suitable for detecting a gross effect since it 'fixes' tissue fluid and preserves the relations of living tissues so that the shrinkage and distortion which accompany most routine methods of fixation and embedding[7] are absent. In the control animals the cross-sectional areas of the nerves of the two sides were similar. In the animals with occluded eyes the nerves of the operated side had a cross-sectional area some 10 per cent less than those of the unoperated side.

The question of which tissue component or components of the optic nerve was responsible for this gross effect could not be settled with certainty. Difficulties in quantification with this method may be related to the large number of small fibres and to the intimate membrane contacts which exist in this situation[8]. The visual impression gained was that both axon diameter and sheath thickness failed to reach normal values. Substantiation of this is being sought by electron microscopy; phase contrast microscopy is unsuitable for precise dimensional estimations of this kind[6]. Gyllensten and Malmfors[1] commented only on overall fibre diameter. Since their experiments affect the time of onset of myelination, which is an incremental process, it is likely that sheath thickness has been affected. Has axon diameter also been affected by light deprivation? Is the retardation of myelination secondary to retardation of axonal growth? One would expect, *a priori*, a primary effect on the axon, but the secondary effects of this cannot be predicted. While axons do not myelinate below a certain minimum diameter (which varies with site and species) the diameter of newly myelinated axons varies quite widely[9–11] and it may be that myelination is related more closely to neuronal type and maturity, as indicated by the pattern of Nissl substance[12], and to neuronal activity, which does not necessarily depend on connexions. Whatever the factors, it would be interesting to know whether the rate of incremental myelination (increase in sheath thickness) as well as its onset are affected by light deprivation. This is yet another situation in which analysis is incomplete without determination of both axon diameter and sheath thickness[13].

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