PUPILLARY PHENOMENA ON APPLICATION OF A STRONG CONSTANT ELECTRIC CURRENT AS USED IN ELECTRONARCOTIC TREATMENT.

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IN the usual procedure of electronarcotic treatment, the electrodes are placed bitemporally and an initial electric current of 180 m.a. is applied for 30 seconds. During this period a tonic extension of the skeletal muscles occurs, accompanied by strong contraction of the pupils, which in the majority of cases are reduced to pin-point size. Slight convergence of the eyes may accompany the contraction. The contraction relaxes somewhat when clonus ensues, but usually becomes slightly more marked in the following stage when the electric current applied is between 60 and 80 m.a.

This pupillary contraction is in contrast to the pupillary dilatation observed in the tonic-clonic stage of electric shock treatment. In electroshock, an electric current of very short duration is applied to produce an epileptiform fit, whilst in electronarcosis the skull is submitted to a strong constant electric current. The eye signs in shock treatment are part of the convulsive mechanism, whereas in electronarcosis the possibility of stimulation of central and peripheral parts must be considered.

Pupillary contraction has been elicited in animal experiments by stimulation of the occipital lobe ; this area is too far away from the site of stimulation to be considered in our observations. In the human subject no constricting effect upon the pupils could be produced by direct electric stimulation of any cortical area (Foerster, 1936 ; Penfield and Erickson, 1941). It is unlikely, considering the site of the electrodes, that the pupillary phenomena are due to stimulation of subcortical oculomotor centres ; moreover, upon placing one electrode on more occipital parts of the skull the contraction of the pupil on this side failed to take place. This finding strongly suggests that the pupillary response is produced by local stimulation of the peripheral tissue.

There are three pathways by which the pupillary size might have been influenced : reflexly through the trigeminal nerve or the optic nerve ; directly through the oculomotor nerve. As the pupillary contraction did not occur when one electrode was placed more occipitally but still in the distribution area of the trigeminus, a trigeminal reflex can be excluded. For the same reason an optic reflex is improbable. Because of the partial crossing of the optic fibres, if stimulation of the optic nerve should lead to reflex contraction, one would expect a contraction in both eyes wherever the second electrode is placed. There remains to be considered direct stimulation of the oculomotor nerve ; there is no fact in the experimental conditions which would contradict

this assumption. It is therefore interesting to note that amongst the oculomotor fibres the pupillo-constrictory are the most responsive to electric stimulation.

In further examination one eye was, before treatment, atropinized so that no contraction of the pupils to light nor to convergence could be elicited. It was then found during application of the electric current that whilst the pupil of the untreated eye contracted as usual, a dilatation of the atropinized pupil occurred. This result would be open to controversy but for the experimental findings of Kunz and Richins (1946). These authors, experimenting on cats and dogs, stimulated the oculomotor nerve with faradic current, with the result that contraction of the pupils occurred. When they had atropinized one eye and then applied the stimulus, the pupil of this eye dilated still further whilst the other pupil contracted as before. The dilatation of the pupil occurred in their experiments only when the atropinization had been complete, and the stimulus required was appreciably greater than that which elicited constriction.

They conclude from these experiments that two separate neurones reach the sphincter muscle of the eye *via* the oculomotor nerve, a cholinergic, whose stimulation causes the sphincter to contract, and an adrenergic and inhibitory, which when stimulated causes the sphincter to relax. The findings of these authors in animal experiments are therefore identical with our own observations in humans.

In the past it has been the accepted view that the pupillary reflex mechanism is regulated by the parasympathetic and sympathetic system, by an antagonistic inhibitory activity of the two systems. Recent experimental work, however, has shown that the role of the sympathetic in the variability of pupillary size is of minor, if of any significance. Recording of the pupillary reactions after sectioning of the oculomotor nerve and the extirpation of the sympathetic proved that the parasympathetic is almost exclusively responsible for the pupillary reactions to painful stimuli, to emotional stimuli, and to the withdrawal of light (Ury and Gellhorn, 1939; Hodes, 1940; Seybold and Moore, 1940; Ury and Oldberg, 1940; Langworthy and Ortega, 1943; Gellhorn and Levin, 1945; Kunz and Richins, 1946). The experiments of Kunz and Richins, and our own observations, suggest that the regulation of the pupillary size in the midbrain, mediated by the oculomotor nerve, is actuated by two separate fibre systems, one whose degree of activity determines the intensity of sphincter contraction, the other which, when active, brings the sphincter to complete relaxation.

If this is correct we should find it expressed under physiological and pathological conditions.

The following observations were made on two patients both suffering from an anxiety state. They had in ordinary daylight a pupillary diameter of 5 and 6 mm. respectively, and reacted well to light. When in the dark the pupils enlarged to 7 and 8 mm. and the reaction to light was absent. There was no serological nor neurological abnormality in either case, and the cerebrospinal fluid of one was negative. Assuming two separate fibre systems arising from the oculomotor midbrain centre, then withdrawal of light may not only reflexly lower the activity of the constrictor fibres, but may also stimulate the

sphincter relaxing adrenergic fibres; if this be so the light stimulus will find the constrictory fibres in a refractory state and no contraction of the pupils will occur. From the preceding it seems reasonable to assume that excess of adrenaline in the blood stream might be a factor which conditions the rigidity of the pupils under these circumstances. The mental state of the two patients would support this assumption. Failure of the pupils to react to light, without any organic process, has long been known in patients where emotional tension is in the foreground of the picture. This has been described in catatonic states and in the hysterical fit. In these conditions the underlying mechanism may be the same as in our two cases, with the difference that withdrawal of light is not an essential factor for the appearance of this phenomenon.

The existence of two specific neurones originating in the Westphal-Edinger nucleus would finally offer an explanation for the paradoxical enlargement of the pupils to light found occasionally in the Argyll Robertson pupil and in internal ophthalmoplegia; the mechanism may be similar to that in the atropinized eye following oculomotor stimulation by electric current. Provided that the constrictor neurone is sufficiently inactive, failure of the pupils to react to light would increase the likelihood of the antagonistic adrenergic neurone being activated by the light stimulus and producing a pupillary dilatation.

Summary.

In electronarcotic treatment when a galvanic current of 180 m.a. is used with the electrodes bitemporally placed, a strong contraction of the pupils takes place. This is found to be due to stimulation of the oculomotor nerve. On complete atropinization of one eye, the pupil of this eye dilated still further under the same conditions. The results are identical with those obtained by Kunz and Richins in animal experiments, and they suggest two separate neurones arising from the oculomotor midbrain centre conducted through the oculomotor nerve to the sphincter muscle of the iris; the cholinergic determines by its intensity of discharge the intensity of sphincter contraction, the adrenergic actively relaxes the sphincter. Clinical phenomena which can be correlated to these findings are mentioned.

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