$$\text{R. vision } \bar{c} \frac{+10.50 \text{ D.Sph.}}{+1.50 \text{ D.Cyl. Ax. } 60^\circ} = \frac{6}{36}$$

$$\text{L. vision } \bar{c} \frac{+12.00 \text{ D.Sph.}}{+1.00 \text{ D.Cyl. Ax. } 90^\circ} = < \frac{6}{60}$$

Lens and vitreous were clear R. and L. Fundi normal. The tension was normal in each eye.

Photographs were taken of a normal eye, to act as control, and of the abnormal in as nearly the same relative positions as possible. An attempt was made also to secure the same degree of magnification in all photographs. From a comparison of the two sets of photographs the following points relative to cornea plana may be noted:—

1. The globe is approximately of normal size.
2. The cornea is definitely smaller than normal.
3. The line of corneo-scleral junction has lost its sharp definition.
4. The curvature of the cornea is the same as that of the sclera.

The condition of cornea plana is regarded as a familial one being probably transmitted as a Mendelian recessive character (Duke-Elder). In this case, nil of importance could be elicited from the Family History.

I wish to thank Brigadier G. I. Scott, Consultant Ophthalmologist, Middle East Forces, for kind permission to publish these notes.

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THE EFFECTS OF FARADICALLY INDUCED CURRENTS UPON THE EXTRINSIC AND INTRINSIC OCULAR MUSCULATURE*

A Clinical Self Experiment

BY

Major J. HORTON YOUNG, R.A.M.C.

ALTHOUGH the effects of faradism upon the extra-ocular muscles have been noted by previous observers, the information at my disposal was not sufficiently detailed for my own satisfaction. In fact, this subject is rather avoided in text-books, whether they are ophthalmic, electrical, therapeutic or physiological manuals. In the circumstances two simple clinical experiments were

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designed to clear up this matter, the results being incorporated in this article.

The aim of these experiments was straightforward enough and limited in scope, but the ensuing phenomena, being somewhat widespread and complex considering the nature of the tests, proved less easy of interpretation than was expected. This latter fact has induced me to publish these experiments, as it was felt that other ophthalmologists may find interest in placing their own interpretation upon the clinical signs and symptoms elicited. The information may also be of use to physiologists.

In analysing the results an attempt has been made to explain the clinical data in terms of applied physiology. Some of the opinions expressed are at variance with acknowledged opinion on this subject, and are given with all due diffidence. Where possible such expressions of opinion are supported by argument which, being of a clinical nature, cannot be conclusive. It is hoped, however, that this article may revive interest in faradism and the ocular musculature.

Being abroad on active service at the time of writing, the number of references at my disposal is limited, and in consequence it has been impossible to uphold fully the golden rule of scientific precedent. The design of the experiments and the experimental data embodied in this paper are therefore self-limited, and as such form an original work.

In undertaking the experiments outlined below, I did so with the object of studying the clinical effect of induced faradic currents upon the extrinsic muscles of the eye in an effort to discover whether it was possible to exploit this electrical medium on a therapeutic basis for the cure of ocular anomalies. The effects of faradism upon the intrinsic ocular muscles and the plain muscle of the orbit were complementary phenomena of unexpected severity, which proved most interesting, if somewhat painful.

Faradic stimulation of the left internal rectus muscle was decided upon, using the Smart-Bristow coil. This particular instrument was chosen because it permitted the use of minimal currents which could be easily controlled by the operator.

The Smart-Bristow coil consists of a dry cell operated electrical induction circuit in which a solenoid has been inserted in the primary circuit in association with a controlled "make and break" oscillator. The secondary unit is tapped in four places. Both electrodes arise from the secondary circuit, which carries the induced currents to the side elected for stimulation. The electrode applied to the globe is called the active electrode. In order to increase the strength of the induced currents an iron core is inserted in the primary circuit. Half the iron core was used in these experiments. The "make and break" of the primary circuit

was set at about 70-80 oscillations per second. One layer of wire from the secondary coil was employed to obtain the very minute currents for the experiments. A fine circular metal disc electrode, five mm. in diameter, covered in chamois and moistened in saline, was used for the active electrode, while the non-active electrode was placed on a saline pad and attached to the left arm. By a makeshift arrangement the ocular electrode was made to point at right angles to the insulated hand electrode, on which there was an additional safeguard in the form of a thumb-operated contact breaker.

As this was a self-experiment it was imperative to note as quickly as possible, on an available standard clinical instrument, any resulting phenomena. For this reason the Maddox Wing test was chosen for registering the amount of contracture of the rectus internus, while the degree of spasm of the ciliary muscle was estimated by the strength of minus lens required to render the left eye emmetropic for distance.

In order to simplify the tabulation of results all visual estimations in these experiments were made with the refractive error corrected by the appropriate lenses. In the case of the Maddox Wing test, where the letters were not clearly seen owing to ciliary spasm or paralysis, a white arrow was made to slide above the numbers until it coincided with the white arrow visible to the right eye. With correction the author registered four to six prism dioptres of exophoria on the above instrument. Vision with correction in both eyes was 6/6 and J.1, the right eye being the master eye.

At the first experiment five applications of induced current were made of approximately three seconds duration and at one and a half minute intervals. The site of election for stimulation was over the left internal rectus muscle, the centre of the disc electrode being 10 mm., posterior to the medial limbus of the cornea.

The primary application was performed under 5 per cent. cocaine anaesthesia as a local instillation of the conjunctival sac. As the result of the experience gained in the first test the second trial was conducted under cocaine anaesthesia and *homatropine mydriasis* and paralysis of ciliary body.

Experiment 1

Phenomena observed.—Despite conjunctival anaesthesia the presence of the electrode was felt as a diffuse pressure on the globe. The induced currents were of such a nature that they could not be tolerated for any long period of time, although the impulses were not strictly painful in the true sense. Electrical stimulation of the supraorbital nerve occurred, which was felt

along the main branch only. This may have been due to the fact that the upper lid was difficult to avoid in the placing of the electrode. No unusual subjective phenomena were experienced in the form of light flashes or such like, but an increasing dimness of vision began to set in about four minutes after the commencement of the experiment. This dimness was relative and affected the distance vision only; close print was read at increasingly shorter distances. This reading distance became stationary later and coincided with the complete non-transitory tetanisation of the ciliary muscle. Visual readings were then R.V. 6/6, L.V. less than 6/60; J.1 was read with both eyes at different reading distances. The amount of ciliary spasm, registered in dioptries, was 7.00, and with this correction it was possible to read 6/6 with the left eye on the Snellen's chart. At the peak of the spasm headache developed, which was most severe and constant and resembled migraine in its intensity. This continued for an hour and a half until the ciliary spasm was released by a mydriatic, when the headache faded as quickly as it came.

During the application of the current it was possible to watch its effect on the oculomotor activity, and nystagmoid movements were observed in left eye which were slow in relation to the rate of "make and break," about 1:30.

Coincidentally with the onset of headache there was a series of remote phenomena associated with vomiting. These were nausea, excessive salivation, altered gustatory sensibility whereby there was a saline metallic taste in the mouth such as is experienced by placing the terminals of an electric battery on one's tongue. The excessive reverse peristalsis followed nausea. The vomiting was not projectile in type. It would seem that this train of symptoms was the direct result of the severe cephalalgia, the pain and/or proprioceptive impulses from the ciliary body assailing the vagal nucleus through reflex channels. It will be noted that all these symptoms are of parasympathetic origin, and as such must have been due to the sensory impulses affecting the *dorsal* nuclei of the seventh, ninth and tenth cranial nuclei. During the course of the experiment other signs developed. The most dramatic of these was the exophthalmos (unilateral) of a non-transitory type which lasted for six hours, disappearing only under the influence of sleep. All the eye signs of true exophthalmos were present—wide palpebral fissure, lid lag on downward movement of the globe, etc., but there did not appear to be any true proptosis. This last factor was difficult of self determination. The exophthalmos was due to the stimulation of the plain muscle of the orbit through the mediation of the sympathetic system, and was tetanic in type. The pupil also dilated, but this was sub-maximal in extent, and contracted under the action of the direct light reflex.

The conjunctival vessels at the point of stimulation were dilated ("Triple response of Lewis"), while the rest of the conjunctival vessels were constricted.

Repeated tests on the Maddox Wing test showed that there was an increasing toning up of the left internal rectus muscle until the figure 2 prism dioptres of exophoria were registered. This muscle spasm was also of the tetanic type and of a transitory nature, being resolved by the conjugate movement of the eyeballs in the direction opposite to that of the contraction of the left internal rectus muscle. It is to be particularly noted that no other extrinsic ocular muscle was induced to contract. A general tetanus of all these muscles would not have altered the Maddox Wing figure. If the third somatic nucleus had been stimulated the left globe would have moved up and in, with a corresponding deviation being registered in the vertical meridian on the Maddox Wing test. There is, therefore, some local regional mechanism which controls the tone of these extrinsic muscles, since faradism does not act by local stimulation of muscle. In the circumstances the effects must have been brought about by the mediation of the autonomic system, in all probability the sympathetic supply, since apart from the parasympathetic response given by the ciliary muscle, the remaining primary phenomena were due to sympathetic stimulation. It may be argued that this increase in tone was due to the coincident increase in tone of the ciliary body, but in the second experiment it will be seen that, with accommodation paralysed, the Maddox Wing figure was "0." Unilateral spasm of the ciliary body, therefore, in no way affects the accommodation-convergence reaction under these experimental conditions.

The salivary secretions were definitely in excess of the normal, and the nausea experienced was probably due to the stimulation of the parietal cells of the gastric mucosa, with an excessive production of HCl.

At no time was vertigo experienced and there was no hyperacusis. The pulse rate was not taken.

Convergent strabismus of the accommodative type did not occur, even when the right eye was focused for close work, but, if the accommodation of the right eye was made to reach an excessive figure by placing a six dioptre minus lens in front of this eye and then voluntarily contracting the ciliary muscle to the utmost, 6/6 vision could only be achieved at the expense of an internal strabismus, the action of the internal rectus muscle under these conditions being necessary before full accommodation could be obtained from the right eye. Thus the relationship of the accommodation and convergence was seen in all its peculiarity, one eye in a state of ciliary spasm without any effect upon the internal

rectus of the same side, while the other non-paralysed ciliary body was able to function independently, but under conditions of maximum *voluntary* accommodation strabismus resulted. This strabismus was a necessary concomitant to the complete effectivity of the ciliary muscle; without this, maximum accommodation could not be achieved.

Experiment 2

Phenomena observed.—In this second experiment the ciliary muscle was paralysed beforehand with homatropine solution, cocaine anaesthesia being employed as before. The object of establishing mydriasis before the commencement of the experiment was, first, to overcome the uncomfortable subjective phenomena resulting from ciliary spasm, and so permit a greater current application to the left internal rectus in the hope that a more permanent contracture of this muscle might be produced. Secondly, to prove that the exophthalmos was due to spasm of the plain muscle of the orbit, and that this spasm was induced by the stimulation of the sympathetic system as the direct result of the faradisation. Thirdly, to show that the secondary phenomena were the result of sensory impulses originating in the ciliary muscle, and that, as the dorsal nuclei only of certain cranial nerves were involved, the dorsal longitudinal bundle must carry a separate strand of nerve fibres to bring about these reflex autonomic clinical symptoms. Fourthly, to indicate the complete unilateral *regional* independence of the accommodative mechanism as distinct from the convergence faculty of the eyes. This latter point is important in man in view of the close clinical, anatomical, and physiological relationships of the rectus internus, ciliary body, third cranial nerve and the other cranial nerve nuclei in relation to the third and their dorsal connector cell masses and the dorsal longitudinal bundle. While all points have not been fully proved by the experiments, much information has been gained.

The results of the second experiment showed clearly that the exophthalmos was due to the stimulation of the sympathetic system, as only those muscles supplied by this particular system were induced to spasm. The response obtained from the left internal rectus muscle was greater than in the first experiment, the reading of the Maddox Wing test registering "0." As before, conjugate movement of the eyes released the spasm of this muscle and the globes took up the position held prior to stimulation. As the ciliary body was already paralysed, the increase in the tone of the muscle must have been due to stimulation of its sympathetic supply. If the third nerve had been stimulated as a whole the left globe, under the action of the muscles supplied by this nerve, would have turned up and in.

No secondary phenomena were experienced, proving that, as in the first experiment, these effects were the result of sensory impulses arising in the tetanised ciliary muscle.

Summary of Signs and Symptoms

For this purpose the results have been divided into primary and secondary phenomena. This is the applied neuro-physiological division into which they naturally fall, the exception being the primary tetanisation of the ciliary muscle in Experiment 1, due to parasympathetic stimulation.

(A) PRIMARY PHENOMENA.

- (1) Lewis's "Triple response" elicited at the site of stimulation.
- (2) Conjunctival blood vessels elsewhere constricted. (Non-transitory.)
- (3) Exophthalmos induced by faradisation. (Non-transitory.)
- (4) Nystagmus similarly induced.
- (5) Tetanisation of all the plain muscle of the orbit. (Non-transitory.)
- (6) Tetanisation of the ciliary body. (Non-transitory.)
- (7) Accommodative myopia produced by ciliary spasm.
- (8) Equivocal contracture of the pupillary musculatures. (Transitory.)
- (9) Equivocal contracture of the left internal rectus only, all other extraocular muscles quite unaffected. (Transitory.)
- (10) No visual "black-out," presumption being that the retinal and choroidal vessels were unaffected by faradisation.
- (11) Marked stimulation of the sympathetic system.
- (12) Parasympathetic stimulation only in respect of the ciliary body, except as an expression of secondary phenomena resulting from the tetanisation of the ciliary muscle. (Vide *infra*.)
- (13) Hemispherical cephalalgia induced by the spasm of the ciliary muscle.
- (14) Sensory phenomena elicited at the site of stimulation despite 5 per cent. cocaineisation of the conjunctival sac.
- (15) Homatropine completely inhibited the effects of faradisation upon the pupillary and ciliary musculature.
- (16) Cocaine was incapable of achieving this result.
- (17) Spasm of and relaxation of the ciliary muscles seen independently in the left and right eyes respectively.
- (18) Complete regional independence of the ciliary muscles and the internal recti shown.
- (19) Full contracture of the right ciliary body, by the intervention of cerebral cortex (volition), in association with the left ciliary spasm, resulted in accommodative strabismus.

(B) SECONDARY PHENOMENA.

These effects were the direct result of certain sensory impulses originating in the tetanised ciliary muscle affecting the parasympathetic nervous system. It will be seen from the nature of these signs that the reflux stimuli affected the dorsal connector cell masses in close relation to the somatic cranial nuclei of the third, seventh, ninth and tenth cranial nerves.

- (1) Gustatory sensibility altered as regards strength of stimulus and nature of the taste sense.
- (2) Increased salivation.
- (3) Sweat glands of the forehead stimulated.
- (4) Probable increase in the gastric secretions resulting in the nausea. (?HCl.)
- (5) Hypermotility of the stomach musculature. Partly nervous in origin and partly due to the effect of the reflex increase in the gastric secretion affecting muscular tone.

Discussion

For the purposes of more facile presentation the various clinical signs elicited in the experiments will be discussed seriatim in the order of their importance. With regard to the autonomic responses, the discussion is divided into (1) those responses discovered in experiment Two, and (2) those obtained in experiment One; experiment Two will be discussed first, as the autonomic responses elicited in this test were common to both experiments. A preliminary analysis shows that in experiment Two the autonomic responses were the result of stimulation of the sympathetic system, while in experiment One this same result occurred, but had superimposed on it the stimulation of the parasympathetic supply to the ciliary body, with its attendant train of secondary phenomena all referable to the cranial division of the parasympathetic system.

(1) Neurophysiological Interpretation of the Autonomic Responses

(1) EXPERIMENT TWO.—In this test the autonomic impulses were transmitted through the efferent pathways of the sympathetic system, the excitor cell station being in the superior cervical ganglion. From this centre the post-ganglionic fibres proceed via the carotid and cavernous plexuses to their terminations in the various muscles and blood vessels. It is noteworthy that while both plexuses were involved in the production of the clinical signs of this experiment, the choroidal and retinal blood vessels, whose musculature is supplied by these same routes, were unaffected, and there was no amaurosis. The plain muscles involved in this experiment were the rudimentary muscle of Müller, the superior and inferior tarsal muscles and the small slip of plain muscle which is incorporated in the levator palpebrae superioris. In addition the musculature of the blood vessels of the conjunctiva was involved. The fact of the "sparing" of the retinal and choroidal blood vessels is difficult of explanation, and it must be assumed that intervening subsidiary excitor cell stations, placed in the path of the impulses passing along this sympathetic network, were sufficient to insulate this particular section of the pathway against harmful or excessive impulses which might affect the efficiency of the visual apparatus (vide infra-retinal circulation).

(2) EXPERIMENT ONE.—In this test the nature of the clinical symptoms elicited gives a key to the whole response. A brief summary of these effects has been given (vide supra), which should be analysed in relation to these phenomena.

The parasympathetic responses are referable to the cranial

division of the parasympathetic system and, briefly stated, the dorsal nuclear cell mass ("connector cells") of the third, seventh, ninth, and tenth cranial nuclei were involved, the somatic equivalents being unaffected. These "connector cells"—the homologues of the posterior and the lateral horn cells of the spinal cord—communicate with the excitor cell stations of the parasympathetic system outside the brain proper. Depending in what manner the excitor impulse is distributed after leaving these centres, the symptoms will vary according to the strength of stimulus and the resistance experienced by this impulse as it flows along the numerous diverse ramifications of the parasympathetic nerve fibres. It will be evident then, that with clinical effects of such limited distribution, these connector cell masses on both sides of the brain must be in very circumscribed neuro-physiological relationship. This suggests that, while the dorsal longitudinal bundle is a very composite bundle with many connections within the brain, the parasympathetic system has developed within this bundle its own set of communications, discrete and separate from the rest of the bundle proper, a specialised "*tractus para-sympatheticus cranii*," as it were, to serve the intricate reflex mechanisms connected with the cranial division of the parasympathetic system. The evidence in favour of this is negative, viz.: (a) No effects traceable to the somatic cranial nuclei of the related dorsal nuclei were found; (b) the limited nature of the responses; (c) there is no other nervous pathway known which could produce these symptoms without introducing a more complicated clinical picture than that shown.

The sympathetic responses common to both experiments have already been discussed, with the exception of the reaction of the left internal rectus, which is also claimed as a sympathetic response. In both experiments this muscle underwent transitory increase in tone, which was abolished by conjugate movement of the eyeballs in a direction away from the movement of contracture. This may be claimed as a somatic response by some, but if the clinical data are analysed it will be seen that there was (1) no upward component in the movement of the globe, which one would have expected if the third cranial nucleus was stimulated, *i.e.*, contracture of all those extrinsic ocular muscles except those supplied by the fourth and sixth cranial nerves. (2) That if the third, fourth, and sixth cranial nuclei were involved together, the increase in tone of all these muscles would have maintained the Maddox Wing findings at their original figure. (3) There was no diplopia for reading in the first experiment and none for distance in the second experiment. Ruling out the somatic nuclei, the increase in tone must have been due to (a) autonomic stimulation, or (b) local stimulation of the actual muscle fibres. It is known

that faradisation does not affect contracture of muscle by stimulation of the actual muscle fibres, so that the contracture was produced through the mediation of the autonomic system, in all probability the sympathetic system, since the other plain muscle contractures were affected through this same system, by means of the efferent impulses arising in the superior cervical ganglion. For reasons previously stated the left internal rectus muscle only was stimulated to contract, hence each individual extrinsic ocular muscle must have a cell unit in the cervical sympathetic ganglion, thus introducing a local autonomic reflex control of extrinsic ocular muscle tone.

(2) Tetanisation of the Orbital Plain Muscle. (Non-transitory Contracture)

Halliburton and McDowell state that plain muscle cannot be tetanised, but it has been shown that all the plain muscle of the orbit underwent tetanic contraction which lasted for a minimum of six hours, resolving only under the influence of sleep. These contractions were of a painless nature, with the exception of the ciliary muscle, which was most painful.

The question of biochemical effectors at myoneural junctions is dealt with later, but it is important to note here the relation of the biochemical effectors and the nerve supply to the tetanised muscles, as it not only throws light on the mode of tetanisation, but incidentally gives a clue to the nerve supply of the ciliary body, about which there is still some doubt. With the plain muscle of the orbit the biochemical effector liberated at the muscle terminations of the sympathetic system was adrenalin, while acetylcholine was liberated at the parasympathetic nerve endings in the ciliary body. In each case tetanisation resulted. The pupil, however, gave an equivocal response, due to the fact that the sympathetic and the parasympathetic systems were represented in this portion of the eye, with its two opposing types of plain muscle and their physiologically opposed biochemical effectors. For this reason the view put forward that the ciliary body has no sympathetic supply seems well founded, the contraction and tone of this muscle being controlled solely by an acetylcholine threshold maintained by parasympathetic activity. As the volitional control of the ciliary muscle is via the third cranial somatic nucleus there is no conflict of effector action, since the biochemical effector in this case is also acetylcholine.

Finally, one sees that the plain muscle of the orbit obeys the "all or none law" with respect to the whole muscle under faradisation, *provided that the plain muscles involved are innervated by only one section (sympathetic or parasympathetic) of the autonomic system.*

(3) *Exophthalmos*.—All the characteristic signs of this were present in the left side only. Slight dilatation of the pupil, wide palpebral fissure, lid lag on downward movement of the globe, exposure of the sclera, above and below the cornea, were present, but there was not true proptosis of the globe (as far as I was able to judge). This exophthalmos was present in both experiments, and in no case could it be resolved except by sleep. The contractures of the muscles inducing this condition were proved to be painless, since, apart from the electrical sensations at the site of stimulation, there were no other painful impressions in the second experiment.

(4) *Nystagmus*.—During the progress of the experiments a regular lateral nystagmus took place, which was composed of a sharp medial movement of the stimulated left internal rectus and a slower recoil. The rate of nystagmus was much slower than the electrical oscillations operating in the electrical circuit under the influence of the "make and break" mechanism. The nystagmus was unilateral, and at no time was vertigo experienced in association with this phenomenon. This result was probably due to the mediation of the autonomic system. Faradic nystagmus must therefore be added to the long list of causes of nystagmus.

(5) *Lacrimatory "suppression"*.—There was no production of tears during or after the experiment, which one would have expected as a simple protective mechanism, apart from the known fact that the lacrimal gland is supplied by the sympathetic system. One can understand the anaesthetisation of the conjunctiva producing this under conditions of simple local stimulation of the conjunctiva or cornea, but when the electrical impulses were distinctly felt, the "triple response" of Lewis was obtained, and there was general stimulation of the sympathetic system in the greater part of the orbital area; the lacrimatory "suppression" is difficult to understand.

(6) *Retinal circulation*.—Although there was marked sympathetic stimulation of the excitor cells of the superior cervical ganglion, this in no way affected the calibre of the retinal and choroidal vessels, since at no period of the experiment was there any amaurosis, vision 6/6 with a myopic lens and J.1 at short distance without any correction. It is apparent that there are subsidiary excitor centres for these intraocular vessels either within this ganglion or elsewhere in the path of the sympathetic impulses. If this last is the case, it is probable that the impulses from the superior cervical ganglion were unable to liberate a "synaptic transmitter" at these excitor cell stations, and that sufficient resistance was introduced in the path of the impulses to insulate the blood vessels against these excessive and foreign stimuli produced by faradisation. It illustrated, however, the

wonderful insularity of the synaptial junctions, which in their way act as protectors of vital tissue against the numerous impulses which pass along the sympathetic chain during the course of one's existence.

(7) *Antidromic response*.—Despite the removal of the exteroceptive sensory stimuli by cocainisation of the conjunctiva, the electrical stimuli and/or trauma, produced by pressure of the electrode on the left globe, was sufficient to evoke the "Triple response" of Lewis.

(8) *Biochemical effectors at the synaptial junctions*.—It is believed that the liberation of acetylcholine at the synaptial junctions is responsible for the transmission of a nerve impulse across a synapse. If this is so, the acetylcholine threshold for these junctions must differ with the section of the nervous system undergoing stimulation as well as for the particular tissue supplied by the nerves involved, since we have seen that, although the autonomic nerves conveying impulses from both the carotid and cavernous plexuses were involved, certain structures supplied by these self-same nerves were not stimulated. The classic example was the lack of response exhibited by the retinal and choroidal blood vessels of the left globe.

(9) *Biochemical effectors at the myoneural junctions*.—Where plain muscle of the orbit is concerned (inclusive of the ciliary muscle), both acetylcholine and adrenalin appear to exert an equal power, in that they are both capable of inducing tetanus in these muscles, *provided that the two biochemical effectors are not represented in the same muscle*. Where they are both represented in the same muscle adrenalin appears to possess priority in what might be termed "the Power Table of Humoral Precedence" under the conditions of this electrical experiment. This priority, however, was but temporary, since the contractures initiated by adrenalin are readily overcome by acetylcholine liberated reflexly through the parasympathetic system, *e.g.*, as seen in the equivocal pupil response or in the reduction of the spasm induced in the left internal rectus by the volitional act of conjugate movement of the globes, through the intermediary of the third cranial somatic nucleus of the same side. The only exception to these rules was the musculature of the conjunctival vessels, which exhibited dilatation at the site of stimulation and the constriction elsewhere of a more permanent nature.

(10) *Pain impulses*.—The cephalalgia which accompanied the tetanisation of the ciliary body has been described (*vide supra*). It remains to analyse the reason why pain occurred in this case and not in the case of the tetanised plain muscle of the orbit.

The threshold for pain in the ciliary body appears to be less than that for the other ocular muscles. This threshold factor may

have been lowered by the contracture of the ciliary muscle reducing the size of the blood vessels, and so producing stagnation of the pain producing substance "P" of Lewis, which, accumulating rapidly, inversely affects the pain threshold. On the other hand, the situation is complicated by the fact that the acetylcholine liberated in the muscle under stimulus should have been more than necessary to counterbalance this diminution in the vessels. The alternative postulate is that the sympathetic activity which caused contracture of the conjunctival blood vessels produced a similar contracture of the vessels of the ciliary body, quite independent of the biochemical effector acetylcholine which mediated in the contracture of the ciliary muscle. This latter explanation is probably the correct one, although it is difficult to imagine the choroidal vessels being "spared" in these circumstances.

(11) *Conditioned reflexes.*—The relation of the ciliary body to the dorsal cranial nuclei of the seventh, ninth, and tenth cranial nerve nuclei having been demonstrated in these experiments, it shows how easily migraine may develop as a conditioned reflex in subjects with inherently unstable vasomotor systems, the overflow of impulses from any organ in relation to the cranial division of the parasympathetic system spreading from their localised tracts in the above system to the vasomotor supply of the cerebral vessels, resulting in dilatation and hemicrania.

(12) *Tetanisation of the left internal rectus muscle.*—The extrinsic ocular muscles are capable of voluntary contraction, but their physical properties on histological analysis show them to be quite different from skeletal muscle. Moreover, their reactions under certain experimental conditions also differ in a similar way. The muscles appear to have properties akin to the plain as well as the skeletal muscle. In these experiments tetanisation of the left internal rectus muscle was produced, which did not resolve when the faradic stimulation ceased, but was "broken" by the conjugate movement of the eyeballs in a direction opposite to that of contracture. In view of the above facts and the proved marked sympathetic reaction following faradisation, together with the absence of reaction of any of the other muscles supplied by the third somatic cranial nucleus, one must conclude that the tetanisation of the left internal rectus muscle was due to stimulation of the sympathetic system.

Recommendations

Should this paper arouse sufficient interest in other investigators to perform more detailed research in respect of faradically induced currents and the ocular musculature, certain precautions should

be observed. These relate to the persons on whom the experiments are to be performed and the possible further reaction which may affect the eye with more sustained applications.

With personnel: (1) One should avoid performing the experiments continuously on the same subjects. (2) Experiment on one eye at a time and alternate the days on which the tests are made. (3) Take certain clinical precautions with regard to the ocular health of the patient. These conditions are listed under contra-indications below.

With induced faradic currents: (1) Under increasing strength of current it may be possible to cause spasm of the retinal and choroidal arteries with a resultant retinal and choroidal ischaemia resulting in blindness, as the spasm of the delicate ocular vessels is very intense and constant. This is well seen in the conjunctival vessels, which were still in a state of spasm six hours after the experiment. Should this occur the therapeutic application of a sub-conjunctival injection of acetylcholine (5 mm. of B.D.H. solution) may remove this spasm and improve the blood supply, as this effector in therapeutic concentrations is a violent antispasmodic of the vessel musculature.

Contra-indications.—(1) Age. A healthy young adult is the ideal subject for the experiment. Arteriosclerosis from any cause would be a definite bar to this form of investigation, as the changes induced in the vessel wall might result in further pathology.

(2) Hereditary predisposition to nervous diseases such as migraine,

(3) Myopia. Detachment of the retina may result.

(4) Glaucoma. May precipitate an acute attack.

(5) Old trauma to the globe (even slight injuries to the cornea). An irritable eye may result from further trauma attendant on electrical stimulation.

Research

In the light of modern scientific knowledge further research into the action of faradism on the ocular muscles in certain types of muscular diseases should yield fruitful results, as the ocular muscles, with their easily recordable movements, their intricate neuronal affiliations and the differing types of effector substances acting within this limited anatomic field, make this portion of the body the research area "par excellence." Such diseases as myasthenia gravis, in which 78 per cent. of all cases have the oculomotor muscles involved, and Thomson's disease—myotonia congenita—would supply important facts, as these diseases are believed to be due to congenital aberrations of the muscle biochemistry, whereby there is an imbalance between the rate of

liberation of acetylcholine in the affected muscles and its destruction by acetylcholine esterase locally present in the muscle.

Apart from the above spheres for research, the inter and intranuclear synaptial insularity of the brain nuclei make possible the more accurate localisation of the neural pathways in the brain, since there are no irradiating electrical responses within the brain during the conduction of the delicate induced currents.

General Summary

(1) A short description of the Smart-Bristow electrical induction apparatus is given, together with the very simple modification required to undertake the experiments previously outlined.

(2) The phenomena resulting from faradic stimulation of the left internal rectus muscle is described in detail:—

(a) Under cocaine anaesthesia.

(b) Under cocaine and homatropine anaesthesia and mydriasis.

(3) A brief recapitulation of the experimental signs and symptoms is given under the titles of:—

(a) Primary phenomena.

(b) Secondary phenomena.

(4) The physiology of the reactions experienced in the two tests is discussed.

(5) Faradically induced electrical currents have been shown to be capable of producing three important ocular signs, namely:—

(a) Exophthalmos.

(b) Nystagmus.

(c) Spasm of accommodation.

} Via the mediation of the autonomic system.

(6) Some recommendations and contraindications have been laid down for other research workers desiring to undertake these experiments as a preliminary to research work of a similar nature.

(7) Under conditions of consciousness the subjective effects of faradism upon the ocular tissues prevent its use as a therapeutic agent in the treatment of the heterophorias, paralysis of accommodation and the extrinsic ocular paralyses. It is considered that with the advent of modern general anaesthesia, due safeguard as regards type of patient and strength of current used, with or without homatropine as required, it should prove useful for treating the various muscular anomalies resulting from war trauma or other cause. It should also prove useful for diagnosis and prognosis of ocular muscle lesions.

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