THE USE OF ADRENALINE IN MEDICAL AND SURGICAL PRACTICE.

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THE service of adrenaline in medical and surgical practice is apt to be overlooked. And yet the information to be gained from its instillation in the eye is often of great value in estimating the integrity of the sympathetic system.

The supposed danger of producing glaucoma was founded on a series of five cases in which that disease was thought to be aggravated by the instillation of adrenaline.¹ Subsequent investigations have not confirmed the accusation. On the other hand, it has been advocated that adrenaline is not only harmless, but also that it is useful in glaucoma.

Grandclément in 1904² insisted that by the use of it glaucoma may be cured without operation if the disease has not advanced too far. Sydney Stephenson in 1908³ recorded that adrenaline had shown itself of service in the treatment of glaucoma.

O. Loewi in 1907 proposed that the mydriatic response to epinephrine should be used as a diagnostic sign in Basedow's disease and in pancreatic insufficiency.

Coope⁴ also supports the value of the response in the case of acute pancreatitis. According to Watanabe and Kato the sensitiveness appears to be somewhat increased in chronic nephritis.

Meltzer and Auer in 1903 pointed out that an increase of excitability to epinephrine occurred in degenerating structures of the ear vessels.

Lewandowsky in 1898 showed that the mydriatic effect occurs after degeneration of the sympathetic.

Joseph demonstrated that epinephrine was to some extent counteracted by physostigmine; and Githens and Meltzer state that pituitary extract also counteracts the mydriasis in healthy animals.

The intraocular tension, according to Rupert, is first lowered and then increased.

Sollmann⁵ records that this mydriatic effect occurs readily in frogs, but only in mammals under specially favourable conditions.

The mydriatic response in healthy frogs is especially strong, and may, it is said by Meltzer and Auer, be produced by systemic application, in extremely diluted solution, of adrenaline (1 in 100,000).

Post⁶ has noticed dilatation of the pupil after instillation of adrenaline in cases of glaucoma and keratitis.

H. Straub in 1919 found that mydriasis occurred if the excitability of the sympathetic is increased as in Graves's disease and in acute pancreatitis. Later it was found that mydriasis occurs in all types of hyperaemia. The sensitiveness is also increased in chronic nephritis.

A mydriatic response may be obtained a few days after excision of the superior cervical ganglion. In many of these cases the mydriasis was accompanied by other evidences of ocular stimulation, such as separation of the lids, and protrusion of the eyeball.

W. H. Schultz records that in mammalia mydriasis only follows after large doses given intravenously; and adds that mydriasis may be obtained if the oculomotor tone is low, with weak illumination.

In man the effect is only invoked in exceptional circumstances, and then it usually signifies a lesion which involves the sympathetic. Straub states that it may occur if absorption is hastened by conjunctivitis.

The signification of a dilated pupil in irritation of the cervical sympathetic from wounds or growths is well known, but that dilatation can sometimes be produced by adrenaline in such diseases as acute pancreatitis and exophthalmic goitre is apt to be overlooked. Given a normal sympathetic system the power of adrenaline over the iris in man is null.

This drug, so frequently used by ophthalmic surgeons, should not be banned from the arsenal of the general physician. I have never seen any ill effects following its

use as a test, nor can I find any reports of cases in which trouble ensued.

Foster Moore,⁷ quoting from de Schweinitz, writes: "Instillation of adrenaline, ordinarily inactive in causing dilatation of the pupil, becomes exceedingly active when the sympathetic is cut, or the superior ganglion is removed."

In conclusion it is to be noted that the response does not always occur in the numerous diseases mentioned; and therefore, while a positive reaction is valuable, a negative one does not exclude the suspected disease.

Method of Application.—One or two drops of a solution of chloride of adrenaline (1 in 1,000) are dropped into one eye, the other being reserved for comparison. The light should be subdued, as a glare is likely to impede dilatation. The conjunctiva will immediately blanch. If there is no dilatation of the pupil in ten minutes the instillation should be repeated. A dropper that has once been used for atropine should never be employed.

REFERENCES.

- ¹ *Trans. Ophthalm. Soc.*, 1903, p. 374. ² Grandclément: *La Clin. Ophthalmol.*, 1904. ³ *Ophthalmoscope*, 1908, p. 785. ⁴ Coope, R.: *Diagnosis of Pancreatic Diseases*, 1927. ⁵ Sollmann: *Manual of Pharmacology*, 1926, p. 440. ⁶ Post: *Ophthalmoscope*, vol. vi, p. 786. ⁷ Moore, Foster: *Medical Ophthalmology*, p. 95.

THE 1927 EPIDEMIC OF DENGUE IN EGYPT.

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For several years Egypt was practically free from dengue; the last epidemic, prior to that here recorded, occurred in 1906-7 at Port Said and the Suez Canal zone. Last year, however, a sudden epidemic affected the whole country from the shores of the Mediterranean to the extreme south of Upper Egypt, preceded by equally sudden climatic changes. Thunder-storms, winds, and rains swept the whole country, and were followed by the appearance of dengue and other afflictions, such as the invasion of the Nile Valley by locusts, which constituted a very serious danger to agriculture. The epidemic started at the end of September; daily admissions of 30 or 40 cases of dengue were warded in the Government Fever Hospital, Cairo, while thousands of patients were attacked in the town with remarkable rapidity. Groups of policemen on duty in certain streets were attacked as a whole.

Thanks to the experiments organized by the Medical Departmental Research Board of the U.S.A. army and others, we now know that a patient with dengue fever can infect *Aedes aegypti* (*Stegomyia fasciata*) during, at any rate, the first three days of his illness. The dengue virus appears to remain in the infected mosquito for about eleven days before the mosquito can transmit it to non-immunes, and the infected mosquitoes continue to propagate the disease probably throughout the remainder of their lives.¹

The *Philippine Journal of Science* published in 1926 most important information concerning the insect vector of dengue. It was proved that the vector is *Aedes aegypti* (*Stegomyia fasciata*) and not *Culex quinquefasciatus* (*C. fatigans*). It was also proved that a dengue patient might infect mosquitos during a period from a few hours before the first symptoms appear to the end of the second day of the disease, and possibly, though with less certainty, for another twenty-four hours or so, after which mosquito infection fails.²

The virus of dengue is at one stage, at least, an ultra-microscopical filterable one, and is present in the patients' peripheral circulation up to the third day of illness. It is also transmitted by blood or filtrate to non-immunes. The piroplasma-like organism of Graham is probably an artefact. The incubation period is usually from four to seven days.

CHART 1.—Abortive type. Duration of illness before admission one day.