I imagine that somebody perhaps while reading this description would ask himself the question: "But what about infection?"
I fully understand this. In fact, I should never think of doing this operation when using a thread as sewing material—even if it were possible. All the fine voids in the thread would be hiding places for microbes and sources of infection. But a hair is a compact cylinder. And there is one thing more; the needle holes in the conjunctiva bulbi will very soon close firmly, "hermetically" around the hair.

However, more important than these considerations is the experience, and I am pleased to tell, that in 107 operations there has not been the slightest trace of infection or even of inflammation.

The results of the operation have usually been very good. In a few cases there was for a time a moderate increase of tension. In such circumstances treatment with pilocarpine will sometimes be sufficient. When the patient was living far off, I have preferred to perform a second operation opposite to the first and then with good result.

I can say, that time and experience have increased my confidence in the operation.

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**ABSTRACTS**

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**I.—TRACHOMA**


MacCallan states that trachoma is a primary epithelial lesion probably due to a virus and that the histological changes seen in subepithelial aggregations, and sometimes diffuse exudations, of lymphocytes differ in no way from other forms of conjunctivitis such as follicular conjunctivitis. The characteristic features of trachoma are the extension of this subepithelial lymphocytic infiltration to the upper fornix and down to the upper corneal limbus, and the formation of pannus. Pannus is the result of direct spread and is not caused by contact with the infected upper palpebral conjunctiva.

The gelatinous swellings seen in stage 2 are not due to development and change in the original follicles but to occluded ducts of Meibomian and sebaceous glands with retention of their secretion.
Epithelial changes are at first proliferation then villous formation, ulceration and subsequently scar tissue epithelium. In some cases there are localised areas of papillary hypertrophy. Hyperaemia of the lid margin may lead to the development of supernumerary lashes and trichiasis.

Pannus begins as an indefinite grey infiltration at the limbus followed by tiny grey elevations 0.2 to 0.4 mm. in diameter. Capillary loops run centripetally from the vascular loops at the limbus. In severe cases Bowman's membrane is destroyed by the pannus and marked corneal opacities are formed.

The author defines the 4 stages into which he divides the clinical manifestations of trachoma. The aetiology and prophylaxis are discussed.

There are many helpful reflections and observations about the treatment of this disease. The author feels that the manipulative treatment of trachoma is best left to the expert, otherwise it is better to allow the patient to treat himself with drops and lotion than for him to submit to the attentions of a partly trained or inexperienced person.

For the gelatinous excrescences seen in stage 2 expression is recommended and for the 3rd stage the application of irritants such as copper.

Epilation of the lashes is condemned by the author, who also disapproves of the use of lid clamps and the removal of skin from the lids. He favours Streatfeild's operation in which a horizontal wedge-shaped strip is removed from the anterior surface of the tarsal plate and van Millingen's operation where a mucous membrane graft is inserted into the free border of the lid posterior to the lashes.

H. B. STALLARD.

II.—MISCELLANEOUS


(1) Castan says that neuroparalytic keratitis commonly occurs after the removal of the Gasserian ganglion, or after alcohol injection of the fifth nerve for trigeminal neuralgia. A few days after either event there is a complete anaesthesia of the eye accompanied by corneal ulceration.

The aetiology is varied and interesting, and many theories have been formulated regarding the exact pathology of the condition. The most frequently recognised aetiology is a lesion of
the fifth nerve, and the cause may be traumatic or due to a tumour or meningitis.

40 per cent. to 50 per cent. of all cases of removal of the Gasserian ganglion suffer from this condition. Tabes, polyneuritis and syphilis are aetiological factors.

Gradertz described a case following poisoning by acetylene. Cases in which a sympathetic lesion is recognised are extraordinarily rare in the literature.

A participation of the sympathetic would revise the "trophic trauma" theory, which has been brought forward in support of a vasomotor lesion.

The author has found many cases of neuroparalytic keratitis where there have been lesions of both the sympathetic and the fifth nerve. A case has been recorded following removal of the larynx, and was attributed to the fact that ligature of the internal carotid had cut off the blood supply to the fifth nucleus, but surely the cervical sympathetic lesion was the cause? It has been shown that lesions of the fifth nucleus and the part of the nerve before it enters the Gasserian ganglion do not cause neuroparalytic keratitis.

The vasomotor theory is based on the presence of the sympathetic fibres which run with the fifth nerve.

Syringomyelia causes changes in the sympathetic nervous system, and in rare cases it has been known to cause neuroparalytic keratitis.

The author has met with Horner's syndrome in 25 per cent. of cases of syringomyelia, and more rarely heterochromia, hemiatrophy of the face, and one case of neuroparalytic keratitis.

Neuroparalytic keratitis starts with a superficial punctate keratitis. The cornea loses its brightness, and many small grey points appear, on which the epithelium appears to be heaped up and irregular, some of this disturbance is also in Bowman's membrane.

The condition spreads and exfoliation and loss of surface epithelium occurs in the centre of the cornea. Ulceration progresses until nearly the whole of the central area of the cornea is involved and a circular ulcer is produced. Occasionally the upper or lower segment is affected instead. Hypopyon may occur if the ulcer becomes infected and iritis accompanies it.

The disease is characterised by lack of any reactive symptoms, i.e., pain, blepharospasm and lacrimation. If infection does occur the consequences may be serious.

The author's case was a man of 36 years, with syringomyelia, the left eye was normal, the right eye was enophthalmic and had ptosis. There was very slight conjunctival injection, nearly the whole surface area of the cornea was desquamated. There were
folds in Descemet's membrane and some changes in Bowman's membrane. The corneal reflex was much diminished, and the iris was hyperaemic. The pupil was small and active. There were no subjective symptoms and the patient had only come to hospital because his sight was bad.

The patient showed vaso-dilatation on his cheeks, ears and hands, the fingers of the right hand showed bruises, owing to loss of sensation. The syringomyelia was of the cervical type, C.S.F. and Wassermann test were negative. The author then had to determine whether there was any relation between the neuro-paralytic keratitis and the syringomyelia.

In syringomyelia there is a proliferation of neuroglia accompanied by a vacuolation of the medulla. The symptoms are loss of sensibility to heat, cold and pain, with preservation of sensation of touch, at the same time there are trophic and vasomotor lesions.

The cervical sympathetic, the trigeminal and the vestibular nuclei and the nuclei of the ocular nerves may be affected. There may be nystagmus due to lesions in the vestibular nucleus. The sixth nucleus is most frequently affected.

Changes in the pupil reactions are common; the other eye symptoms have been mentioned previously. In the author's case the pupil was contracted, the palpebral aperture diminished and the eye enophthalmic, there was marked ptosis, the facial vessels injected, the skin temperature raised and excessive sweats occurred.

There was no fall of ocular tension, as sometimes occurs. Which was responsible in this case, the trigeminal lesion or the sympathetic?

Against the sympathetic being responsible is the fact that sections of the sympathetic and removal of the cervical gland do not cause neuroparalytic keratitis, but many lesions of the trigeminal and Gasserian ganglion are not followed by this condition. For example there are cases of complete anaesthesia of the cornea (due to trigeminal paresis) which remain in statu quo for years and never develop neuroparalytic keratitis. A case has been described of a fractured base with complete fifth nerve paresis and lagophthalmos with facial paresis and no neuroparalytic keratitis. A case of trigeminal paresis with trichiasis never developed neuroparalytic keratitis.

Occasionally the cornea is hypersensitive in neuroparalytic keratitis, in fact neuroparalytic keratitis has occurred with an anaesthetic or a sensitive cornea, with a dry cornea or with good lacrimal secretion, with ptosis or with lid retraction. Neuro-paralytic keratitis does not occur with fifth nuclear lesions or lesions of the nerve trunk.
So other authors have sought for an explanation in vasomotor and trophic changes or anything interfering with the nutrition of the cornea.

Vasomotor and trophic changes are observed in most cases of neuroparalytic keratitis, and sympathetic nerve fibres run with the fifth nerve. A paralysis of the sympathetic has been observed after removal of the Gasserian ganglion. The author thinks that the sympathetic fibres that run with the fifth nerve are responsible for most cases of neuroparalytic keratitis attributed to the fifth nerve.

Behr says that the keratitis is produced by an abnormal trophic reaction, and is due to an affection of the ganglion cells.

The author concludes in his case that the syringomyelia caused the sympathetic changes and these gave rise to considerable vasomotor and trophic disturbances in the eyes, hands and ears.

Because neuroparalytic keratitis has appeared in a cornea protected by ptosis and with much sympathetic paresis, in the author’s “humble opinion” the aetiology in his case is the trophic vasomotor changes, produced by the sympathetic lesion during the course of a syringomyelia of the cervical type.

E. E. Cass.


(2) Young describes 2 cases of ataxia affecting 2 young males in the same family and in whom were present clinical features of Friedreich's ataxia and Marie's disease (familial spinocerebellar ataxia). These cases were essentially of the Friedreich type but showed some transitional signs of Marie's disease.

Nystagmus, a constant feature of Friedreich's ataxia but absent in Marie's disease, was present to some extent in both patients and optic atrophy, which is an early and quite constant sign in Marie's disease but absent in Friedreich's ataxia, was also evident.

In one case exophthalmos of 1 mm. was noted in the right eye and the author comments on the fact that this physical sign has hitherto not been recorded in the literature of this disease.

The neurological features of ataxia, standing on a wide base, the gait, pes cavus, dorsal extension of the big toes, and altered tendon reflexes are recorded.

The literature, aetiology and points in differential diagnosis of Friedreich's ataxia and Marie's disease are discussed.

H. B. Stallard.

Lloyd in this paper reviews the main clinical features of arachnodactyly and discusses the ocular disorders associated with this defect. Congenital miosis and bilateral ectopia lentis are chief among these. In the former the pupil will not dilate with atropine and the latter complication was noted in 5 or 6 cases seen by the author and according to the literature occurred in half the number of cases of this disease. Two Italian families are described by the author, 5 out of 8 persons were affected in one family and 4 out of 6 in the other. In one family the defect was traced to the mother and the maternal grandfather.

The lens is generally displaced up and out and in some cases has dot-like opacities. The fibres of the suspensory ligament are clear and strong, and fine red-brown dots were seen on these and in the vitreous.

In 2 cases quoted from the literature the lens was removed after dislocation into the anterior chamber. It was found to have the weight and diameter of the lens in a new-born infant, increased thickness, and defective development at a period in later intra-uterine life. On the posterior capsule there were remains of embryonic tissue and a fissure at the pole in one case. The fibres of the lens were arranged irregularly. In another lens there was heaping up of the nuclei in the posterior part of the lens and pathological changes suggestive of an over-development of the anterior portion of the lens.

In the discussion that followed this paper Vail made the observation that arachnodactyly was sometimes associated with megalocornea, a deep anterior chamber, persistent pupillary membrane, atrophy of the superficial layers of the iris and was a progressive disease.

H. B. Stallard.


Swab discusses the aetiology of retinal haemorrhages and champions the cause of tuberculosis as an aetiological factor in the production of perivasculitis of the retinal vessels and subsequent haemorrhages. He reports 5 cases, the ages of the patients varied from 22 years to 46 years, 3 were females and 2 males. In the patient aged 46 years the vascular changes resembled those seen in hyperpiesis. This case and 2 others
responded to tuberculin therapy and apparently one case required this treatment to prevent recurrences. Two other cases cleared up after the removal of focal sepsis and administration of autogenous streptococcal vaccine. All the tuberculous cases showed retinitis proliferans whereas the vitreous became clear in one of the streptococcal cases and manifested a "dense, grey fibrous sheath" in the other. In the 4 younger patients the retinal veins were involved and in the older (aged 46 years) the arteries were affected more than the veins.

The author claims that the 'clinical experience' of response to tuberculin therapy justifies the diagnosis of a tuberculous cause for the retinal and vitreous haemorrhages.

H. B. STALLARD.


(5) King has described a case of chloroma in the orbit of a boy aged 9 years who had left exophthalmos, enlarged cervical lymph nodes, an enlarged liver, a palpable spleen and swellings in the scalp and alveolar margins. He was anaemic and had a sallow complexion.

The swelling in the left orbit disappeared in 25 days after X-ray therapy. Papilloedema and haemorrhages around the macular region and in the inner layers of the retina were present in both eyes. Ulcerative pharyngitis, tonsillitis and broncho-pneumonia terminated in death.

The author comments on the rarity of chloroma lesions affecting the eyes and orbit. Post-mortem examination revealed much fibrosis and cellular degeneration in the orbital tissues on the affected side. The other lesions in the scalp and elsewhere were composed of myelogenous cells in a very sparse connective tissue stroma. The author quotes Cunningham's views about the importance of supra-vital staining methods in examining affected tissues and the blood in order to avoid mistaking young abnormal myelocytes for small lymphocytes.

A discussion on the pathology of chloroma is included in this paper. The author quotes Lehndorff's view that chloroma belongs to a group of borderline cases between genuine neoplasms and leukaemias and that Sternberg maintains that chloroma is a leucosarcoma or myelosarcoma. He mentions recent experimental work by Fischer-Wasels and Bungeler who have produced leukaemia and lymphosarcoma experimentally by long continued administration of indol. They believe that the myeloses and lymphadenoses represent real neoplasms of the blood forming tissues.

H. B. STALLARD.

Oguchi’s disease is a rare familial condition, seldom met with except in the Japanese; the chief symptom is night-blindness which does not alter with years; and the retinal condition is a diffuse whitish grey coloration of the fundus, allowing the vessels to stand out conspicuously, apparently darker than usual by contrast. Mizuo noted that after prolonged dark adaptation, the retina regained its normal appearance and the light sense improved temporarily. Giannini describes two cases of “punctate albino fundus” and separates them from retinitis punctata albscens since they have no contraction of the visual field and no disturbance of colour vision. As to the pathology of the disease, nothing is certain. It seems probable that both the discoloration and the loss of function of the retina depend on some disturbance of the photochemical change which has been demonstrated to occur in the outer segments of the rods; it may be that exposure to light develops an opaque substance in the rods, which limits their activity and accounts for the change of colour of the fundus; when in darkness this substance disappears, the fundus resumes its colour and the retina its function.

Harold Grimsdale.


To produce the block Accardi chooses a substance whose toxic action on the tissues is practically nil; colloid sulphide of mercury. This substance if introduced in colloid form rapidly changes to solid granules and remains fixed in the tissues, undergoing only very slow change to oxide. With these injections, the author found that the intra-ocular tension constantly rose, and the refractive index and albuminoid content of the aqueous, rose also. To prove that it was the block that gave rise to these signs, he injected other animals with equivalent quantities of gelatine, and others with a solution of sublimate of mercury; these animals showed no rise. These experiments confirm the important relationship between conditions of the vascular system and the intra-ocular tension.

Harold Grimsdale.

(8) The relative frequency with which spring catarrh is associated with conical cornea suggests some definite causal relation between the two diseases. Padovani’s patient was a man of 22 years who had suffered from spring catarrh since the age of 16 years. He was seen in November, 1933, saying that the acuity of his right eye had failed. R.V. 1/150. The left eye then showed no corneal change but in January a definite conical ectasia was observed in the left eye and vision had fallen to 1/6. An examination gave signs of endocrinal insufficiency, particularly of the pituitary, thyroid and suprarenals.

HAROLD GRIMSDALE.


(9) It has been shown that this system of tissues has very important functions in protecting the body against infection. Its various parts are more active in the face of infective and parasitic disease. It has also been shown that the cells of the reticulo-endothelial system take up Congo Red when this is injected into the circulation, in quantity varying directly with their activity; thus in the presence of disease, when the cells are specially active, the Congo Red disappears from the blood more rapidly and completely than in normal health. It has been held by many, and especially by Angelucci and his school, that trachoma is in some sense the local expression of a general lymphatic hyperplasia; and that it is in some way connected with tuberculosis.

Tristaino has experimented in a number of cases of trachoma in various stages, injecting Congo Red and examining the blood after the lapse of an hour, to find how much has been taken up by the tissues. He finds an almost constant high level of ingestion, showing an activity of the reticulo-endothelial system above normal, and therefore suggesting some general morbid state; the reaction is very similar to that found in tuberculous patients and he thinks that this serves to make more probable a definite relation between the two conditions.

HAROLD GRIMSDALE.
(10) Scullica (Messina).—Avitaminosis and xerophthalmia. (Avitamino

(10) It is not very rare to observe small series of cases of xerophthalmia but whereas these are usually seen in spring, Scullica noted a small epidemic in the autumn of 1931. It seems clear that these cases are often due to a diet deficient in vitamin A; this is provided normally in the food which contains some substance or substances which are converted by the liver into vitamin A. When the liver is put out of action, by disease or experimentally, the syndrome due to avitaminosis is often seen.

The conjunctival lesions seem to be due partly to trophic changes in the membrane and partly to infection by bacteria. The night blindness depends on the delay of regeneration of retinal purple. It has been supposed that the visual purple takes origin from vitamin A; this is a pure hypothesis, though the vitamin has been shown to be a normal finding in the retina. The author saw in the majority of his cases changes in the pigmentation of the retina. There were in many small whitish areas where the pigment seemed lacking; this change lasted after the night blindness had been relieved.

HAROLD GRIMSDALE.


(11) van Poole in describing the histology of leprous lesions draws attention to certain pathological features similar to those seen in tuberculosis such as the intercellular and intracellular presence of the acid-fast bacilli, the extension of the disease by the lymph and blood streams and the appearance of certain chronic inflammatory cells common to these chronic granulomata.

The author gives a brief account of the large polygonal cells which contain vacuoles, one or more poorly staining nuclei and brownish coloured clumps of bacilli. Sometimes these cells are arranged in an alveolar pattern with collagenous centres, sometimes in broad columns around the papillae, sweat glands, hair follicles and nerves.

The paper describes leprous lesions of the lids, eyebrows, nerves, orbicularis muscle, sclera, cornea, anterior chamber, iris, ciliary body and the anterior part of the choroid and retina.

The author comments on the fact that leprosy like tuberculosis affects mainly the anterior part of the eye. He discusses the endogenous route of ocular infection.

In 206 cases of leprosy he has observed hyperaemia, venous congestion and blurring of the optic disc margins, particularly
on the nasal side, in 49 cases (23.7 per cent.). He quotes Santonastaso who has seen such changes in 26 out of 50 cases. The author believes that these changes in the optic disc which he refers to as optic neuritis are possibly caused by an allergic reaction to the protein of the Hansen bacillus. Many of the patients in whom he made this observation also manifested signs of a general allergy. He quotes Bieling and Oelrichs who have stated that members of the acid-fast group of myco-bacteria are alone capable of inducing a generalised allergic state in which all the body tissues are involved.

Thirty-one out of 49 of the author's cases showing optic disc changes were uncomplicated but the remaining 18 had nephritis, diabetes or lues.

At the end of this paper there is a short account of 260 patients from mixed races who exhibited tuberculous lesions. In 3.4 per cent. of these, were the fundus lesions solely due to tuberculosis.

H. B. Stallard.


(12) It is generally agreed that in all cases interstitial keratitis affects not only the cornea but that the uvea takes part in the inflammatory process also. De Sanctis has had the opportunity of observing the onset of the disease in a number of patients in whom the one eye was already attacked and the second became inflamed while under observation. He examined the unaffected eye from day to day, while the patient was attending the clinic, by means of the slit-lamp, and finds that in all cases the disease begins as an inflammatory process of the anterior part of the uveal tract.

Harold Grimsdale.


(13) Zavalia and Oliva say that primary malignant tumours of the lacrymal sac are very rare; cases of lymphoma, sarcoma, plasmocytoma and epithelioma have been described, but in the literature in all only 50 cases have been recorded.

They describe two cases that they themselves know of.

The first was a man aged 36 years, who for a year had suffered from epiphora in the left eye; he had no other symptoms; and did not pay any attention to this condition until a tumour
began to develop in the region of the lacrimal sac. On examination this tumour was found to be ovoid in shape, was quite painless and felt as though it was a collection of tense fluid; it was not adherent to the skin, which appeared to be quite normal. On pressure, however, there was no regurgitation through the lacrimal passage or through the nose.

A mucocele was diagnosed but at operation it proved to be a solid tumour of the sac which was removed without any difficulty, and sent for section. It was described as a malignant tumour of epithelial origin.

On account of the nature of the tumour an application of X-rays was given; but the patient then disappeared as he felt perfectly well, and was not seen again until 3 months later. There was then another tumour about the same size as that which had previously been removed and this was treated by radium. The patient left the country and was treated by Dr. Sena at Buenos Aires, and after having radiotherapy was considered to be cured, but 8 months later extensive cicatrization involving both lids, the left side of the nose and a large part of the cheek, with total symblepharon and atrophy of the eye occurred. Recently the authors heard of the death of this patient, but, unfortunately, do not know the cause.

The second was a man aged 47 years who had suffered from a painless epiphora without any other signs. For a year he had noticed a small, painless, hard tumour in the region of the lacrimal sac.

On examination this tumour was found to be the size of a small nut; it was sessile and was pushing the lower lid up so that the palpebral aperture was diminished; it was painless and on examination was found to have invaded the orbit, in the region of the lower-nasal angle.

The skin which covered the tumour was a little erythematous, but not adherent, the palpebral conjunctiva was reddened and hypertrophied; the lacrimal passages, however, in this case were freely open. There was one hard enlarged gland in the angle of the jaw.

A small portion of the tumour was excised for microscopy and was reported to be carcinomatous. The tumour was removed surgically, and some days later radium was applied. For 6 months the patient appeared to be cured, but he then developed a small tumour in the left lower lid. This tumour was removed.

It was very adherent to the periosteum, and was extending into the orbit. After some days radium was again applied. Up to the present, i.e., 4 months later, there has been no recurrence.

The authors stress the importance of diagnosing the condition as early as possible.
Until recently the only treatment employed for these tumours was surgical, but there is no doubt that the application of high frequency currents in the modern methods of electro-surgery will make it more probable that the carcinomatous cells will be destroyed and diminish the danger of a recurrence.

But whether they are operated on or not, the authors consider that they should always be treated with X-rays or preferably radium.

E. E. Cass.


(14) Baurmann here puts forward a view of the causation of retinal tears in the region of the ora serrata based on a study of the vitreous of four clinically normal eyes of patients between 16 and 55 years of age with the ultra-microscope; in all these he found definite degenerative changes in the form of cavities or spaces filled with fluid, and granular material between the adjacent fibrils which in places were markedly coarser in texture, these changes increasing in extent with the age of the patient.

It is this tendency to degeneration of the vitreous and its firm attachment to the retina at the ora serrata and orbiculus ciliaris that accounts for tears at the anterior limit of the retina, due to the traction that may be exerted by the vitreous on the retina in this region on movements of the eye.

Where the vitreous is either completely liquified or is a uniform gel there would be no pull on the retina on movement of the eye, but in cases of advanced liquefaction of the vitreous, where only small portions of normal structure remain attached to the retina in front, the greater, liquefied, part of the vitreous remains stationary on movements of the eyeball, but the normal parts move with it and pull on the retina at the point of attachment, thereby causing a tear, with a force that is calculated according to the area of vitreous attachment and the velocity of the ocular movements.

Thos. Snowball.


(15) Fontana has repeated his researches on the position of glycogen in the retina, making use of the methods of Best and Langhans instead of that of Fischer with which his previous
experiments were made. The present confirm his former findings.

In the rabbit and in the frog, glycogen is found in the pigmented epithelium, in the rods and cones and in the external limiting membrane. No change in the amount of glycogen is found under different conditions of light except in the pigment epithelium; here it is increased largely after exposure to strong light.

The author produced experimental hypo- and hyperglycaemia (the one by injections of insulin, the other by injection of pilocarpine) and found glycogen diffused through all the layers of the retina in each case.

Harold Grimsdale.


(16) Examination of the macular colour sense by means of the chromatic equation, has sometimes shown a reduction of perception of green in cases where there is known to be affection of the optic nerve, though the form vision is normal, and the ophthalmoscope shows no fundus change. But little attention has been paid to variations of the peripheral colour sense in disease. Lo Cascio has devised a chromatoscope by which can be estimated the amount of coloured light which must be added to white light of constant intensity to produce the sensation of colour. Colomba has used this apparatus in the investigation of the colour sense of a number of patients having definite disease of the optic nerve, and publishes tables to show the degree of defect. His conclusions are as follow: (1) colour sense is reduced, both in the macular and the peripheral zones to some extent, in all cases of disease of the optic nerve. (2) In tabetic and descending atrophy, green is lost more than any other colour. (3) There is no close relation between central acuity and colour sense. (4) The visual field usually has a condition corresponding to the state of the colour sense, but sometimes colour sense is reduced though the field for white is normal.

Harold Grimsdale.


(17) Regnier in 1924 announced that he had been able to increase the anaesthetic power of cocaine by administering it in alkaline solution. In 1933 Baldoni made similar experiments
with novocaine and percaine and announced similar results; all these experiments seem to have been made on animals. Anastasi has repeated the experiments using very dilute solutions of cocaine (0.25 per cent. and 0.5 per cent.) adding a varying quantity of alkali, and has tested their anaesthetizing power on the human cornea by noting the reduction of the lid-reflex on irrigation of the cornea. He comes to the conclusion that with the solutions employed, the greatest anaesthetic effect was produced by the simple solution and that as the pH increases with the addition of alkali, the effect diminishes. It is known that any salt, to have any action on the organism, must undergo ionic dissociation and it seems probable that the presence of the alkali impedes this progressively with the increase of pH.

HAROLD GRIMSDALE.


(18) The reason for the researches quoted in this paper was a statement that "Lacmin" (Ruata's antitrachomatous vaccine) was dangerous to the eye on account of the carbolic acid which it contained. Fontana has tested the action of phenol in solution of strengths from 0.25 per cent. to 6 per cent. dropped into the eyes several times daily for a fortnight. He finds that solutions up to 2 per cent. are incapable of producing any reaction; that from 2.5 per cent. to 3 per cent. solutions produce a slight hyperaemia which disappears in a few hours. In rabbits stronger solutions produce intense hyperaemia with much secretion, and some corneal desquamation. Since "lacmin" contains only a very small percentage of phenol the author concludes that the alterations sometimes observed after its use, cannot be due to the phenol.

The weaker solutions were tested on healthy and trachomatous eyes.

HAROLD GRIMSDALE.


(19) Goulden in the early part of this lecture quotes liberally from the works of Buffon (1743) in the "Mémoires de l'Academie des Sciences" and from his book "Histoire Naturelle de l'Homme," passages of which are rich with sound observations about all the essential features of strabismus. The author's lecture contains a well-balanced survey of present views concerning strabismus especially its physiological aspects.

He describes the general principles for piloting a case of
strabismus from amblyopia, suppression, abnormal retinal correspondence and false projection to restoration of vision and the achievement of binocular vision. Technical details and descriptions of the large number of instruments employed in orthoptic training are omitted.

In discussing the indications for surgery and the correct time at which these measures should be undertaken he says "I think it should be remembered that concomitant squint is the derangement of the perception of convergence, and has not its primary cause in the muscles of the eyes. Operations upon the muscles of the eye would seem to be tackling the problem from the wrong end, and they are, after all relatively gross measures to try to correct such a very delicate mechanism as the correlated movements of the two eyes. We are not yet in the position to make a considered judgment as to the final results of orthoptic training combined with operation.

There will be a need to follow these cases for several years, and the opportunity for this has not yet come."

H. B. STALLARD.


(20) Wolff reviews the literature on this subject and comments on the clinical facts of these cases in the majority of which amblyopia occurs after repeated haemorrhages, particularly from such organs as the gastro-intestinal tract and the uterus. Cases have been recorded where blindness followed repeated venesection, after a single haemorrhage and in 2 cases during the Great War where it occurred following a secondary haemorrhage.

The author quotes the pathological investigations of Ziegler, Görllitz and Seese who found degenerative changes in the ganglion cells, cytoid bodies in the nerve fibre layer, retinal oedema and fatty degeneration in the optic nerve fibres confined to a region behind the lamina cribrosa (Görllitz's case) 22, 11 and 5 days respectively after the onset of blindness.

The author suggests that the changes leading to blindness are the result of defective oxygenation. In support of this he contends that the haemoglobin content of the blood is lowered in cases of gastric ulcer and uterine haemorrhage and that a further reduction of the haemoglobin leads to spasm in the central retinal artery, damage to the endothelium of the retinal capillaries and subsequent retinal oedema and degeneration of the ganglion cells and nerve fibres.

He quotes the experimental work of Uhthoff who clamped the optic nerve previous to excision of the eye and noted contraction
of the central retinal artery some 5—10 seconds afterwards. This fact suggested that the arterial contraction or spasm was not produced by the cutting off of the blood supply directly but by the fall of the oxygen supply below a certain point.

H. B. STALLARD.


(21) Miss Mann has brought forward an embryological theory in explanation of the changes seen in the eyes and skull in oxycephaly. The visceral mesoderm plays an increasingly important part in the development of the skull on ascending the animal scale to mammals and man. Its texture is denser than the paraxial mesoderm. The alisphenoid, pterygoid and temporal bones are developed from visceral mesoderm called into being by the lateral growth of the brain and accompanied by narrowing of the angle between the optic nerves. She states that oxycephaly is a dysgenetic condition showing itself as a localised arrest of development of the extreme anterior portion of the visceral mesoderm (maxillary process) possibly of atavistic significance. Since the eyelids, eyes and other orbital contents (many of which arise in paraxial mesoderm) appear much earlier in phylogeny than the lateral expansion of the brain we should not expect them to be abnormal.

The optic atrophy which is evident in some cases of oxycephaly is probably the result of kinking of the optic nerves on their steep passage up from the abnormally depressed middle fossa to the intra-cranial openings of the optic canals, or the result of papilloedema from raised intra-cranial pressure early in life.

The authoress’s theory supports such clinical features of oxycephaly as the wide distance the eyes are set apart, shallow orbits, exophthalmos, the prognathous jaw and high palate.

H. B. STALLARD.


(22) Sorsby classifies certain cranial deformities subdividing those generally included under the term oxycephaly. He discusses ocular defects and orbital abnormalities in association with these anomalies.

Exophthalmos and ophthalmoplegia are noted in cases of acrocephalo-syndactylty. In dysostosis cranio-facialis where there is a boss in the line of the frontal suture, a parrot beak nose, sunken atrophic upper jaw and prognathous lower jaw, exophthalmos and divergent strabismus have been observed as in oxycephaly.
The orbits are situated laterally in cases of hypertelorism, a cranio-facial deformity, the result of early ossification of the lesser wings of the sphenoids. Divergent strabismus, mental deficiency, syndactyly, undescended testis, high arched palate, enlarged terminal phalanges and congenital cardiac disease are other defects seen in association with this anomaly.

The author describes an unnamed syndrome consisting of a large skull, ugly face, coarse hair, mental deficiency, corneal opacities in the anterior part of the substantia propria and enlargement of the spleen and liver.

One case of unilateral exophthalmos associated with craniocleido-dysostosis has been recorded in the literature, and brachydactyly and microcorneae have been noted in a father and son.

The ocular defects seen in association with arachnodactyly, polydactyly and retinitis pigmentosa, the Laurence-Moon-Biedl syndrome, syndactyly with aniridia, apical dystrophy of the hands and feet and macular coloboma, and fragilitas ossium and blue sclerotics are also mentioned in this paper.

H. B. Stallard.


(23) Hohn reports the case of a female child aged 5 years with iridocyclitis and ribbon-like keratitis complicating Still’s disease. The interphalangeal joints, wrists, knees and ankles were swollen, painless and showed impaired motility but no osseous change. The author quotes only one other case recorded in the literature.

He discusses the ocular complications accompanying other forms of chronic polyarthritis in childhood due to sepsis and ‘rheumatism’.

H. B. Stallard.


(24) This research by Behr is intended to supply a fuller knowledge of the finer anatomy of the optic nerve and its blood supply, and thereby throw more light on various physiological and clinical problems.

In summing up his findings the author refers to the fact that the ophthalmic artery is not the only source of blood supply to the orbit and optic nerve; between the lacrimal and the middle meningeal arteries there is an anastomosis through the sphenoidal fissure which may be the main artery to these parts.
All vessels supplying the optic nerve penetrate into it from the pial sheath. From the moment that the arteria centralis retinae passes through the pial sheath into the nerve it gives off no branches for its blood supply; it is strictly a retinal artery. The clinical picture of embolism of this artery may, therefore, arise by a block in the vessel within the nerve far behind the retina.

The septal framework of the nerve serves a double function, mechanical (being strengthened specially where protection of the nerve is most required through movements of the eye) and nutritive, in that it is the sole carrier of the vessels supplying the nerve fibre bundles. It is most dense behind the globe and just before and in the optic canal, less dense in the central intra-orbital portion and just behind the canal, while in the posterior intracranial part the septa close to the chiasma disappear.

While in these various parts of the nerve the arrangement and density of the septa is very uniform, there is in the intracranial portion a marked change in the septa running axially in which the papillo-macular bundle runs. They are thinner and less abundant than those in other parts, so that the nerve fibre bundles are there much thicker than elsewhere in the nerve. The finer septa for carrying the capillaries being also nearly absent here, the conditions for the nutrition of this bundle in this portion of the nerve are not so favourable as in other parts. This anatomical peculiarity offers a satisfactory explanation of the origin of retrobulbar axial optic neuritis or the central scotoma in toxic amblyopia, multiple sclerosis and the Foster-Kennedy syndrome.

The blood supply to the optic nerve is derived from three systems of vessels which freely anastomose with one another, (1) Branches from the posterior ciliary arteries around the lamina cribrosa: (2) The anterior arteries of the optic nerve, which penetrate the nerve with the central artery for the anterior and middle thirds of the intra-orbital portion: (3) The posterior arteries, branches of the ophthalmic artery, given off at the apex of the orbit, for the posterior third.

The central vessels in their passage through the dural sheath are not bound fast to the dura by their adventitia, but are surrounded by a system of fine spaces or channels (quite independent of the perivascular lymph system) which permit of a flow of cerebral fluid from the subdural into the epidural spaces. It is, therefore, impossible for the cerebral fluid to produce such a rise of pressure in the subarachnoid space in increased intracranial pressure as to prevent a return of venous blood from the retina and papilla by compression of the central vein, or to allow of its being pressed alongside the central vessels into the papilla.

All vessels passing from the orbit into the nerve (including the central vessels) are enveloped in a connective tissue covering as
they pass from the dura to the pia; this mantle, derived from the dural sheath, is bound more or less closely to the adventitial coat, and shuts off the vessels and their perivascular spaces from the subarachnoid space and liquor cerebralis; it merges into the pial sheath as the latter accompanies the vessels into the nerve. The central vessels lie in a groove on the inner surface of the dural sheath in their short sagittal course in the subdural space, an arrangement which prevents any pressure on the nerve by the pulsations of the artery and the compression of the vessels by the nerve.

During its course from its point of exit from the ophthalmic artery to its distribution in the retina the central artery takes at five places a change in direction which may amount to 90°.

**Thomas Snowball.**


(25) The two Tisconias say that few cases of congenital familial anophthalmos have been recorded.

In anophthalmos there is a complete failure of development of the eye which is usually bilateral, although in some cases there is unilateral anophthalmos with microphthalmos of the other side. The cause is due either to an arrest or upset of development of the optic vesicles. Some authorities say it is due to destruction of the vesicle, whereas others say that the vesicle is never formed.

Various degrees of microphthalmia and anophthalmia have been produced experimentally. The cause of the condition in man is thought to be chronic infection such as tuberculosis or syphilis.

In a family of nine children who were examined by the authors, four had congenital anophthalmos; the parents were healthy and denied venereal disease, but unfortunately they would not submit to a bacteriological examination of the blood.

All cases showed the typically small palpebral aperture, and all had complete absence of both eyes; in two out of the four cases in one orbital cavity was found a very small lump the size of a grain of maize, which the authors consider was the rudimentary remains of the eyes.

In all other respects the children were normal and showed no signs of congenital syphilis.

The authors record one other case in which, again, the parents were both healthy but they were relatives; the patient herself was an intelligent child aged 8 years, again she revealed no other abnormality. On palpation nothing could be found in either orbital cavity.

E. E. Cass.