Table of Cases of Concomitant Strabismus.

There were 185 cases in all, of which 100 were girls and 85 boys; in 63, or 34.05 per cent., of these cases the squinting eye was amblyopic.

Alternating convergent strabismus	...	...	51
Right convergent strabismus	...	...	49
Left convergent strabismus	...	...	75
Alternating divergent strabismus	...	...	4
Right divergent strabismus	...	...	3
Left divergent strabismus	...	...	3

Total 185

Table of Cases of Convergent Strabismus for each Age (Number and Sex).

Age:	4	5	6	7	8	9	10	11	12	13	14	Total.
Girls ...	5	15	10	11	13	7	9	5	13	4	2	94
Boys ...	—	6	14	8	21	7	6	5	8	3	3	81
Total ...	5	21	24	19	34	14	15	10	21	7	5	175

Table of Cases of Amblyopic Squints (Number and Sex).

Age:	4	5	6	7	8	9	10	11	12	13	14	Total.
Girls ...	—	2	4	4	3	3	5	1	4	2	1	29
Boys ...	—	1	3	4	9	4	3	1	5	3	1	34
Total ...	0	3	7	8	12	7	8	2	9	5	2	63

I have also more closely analysed the myopic cases, among which I include cases of mixed astigmatism. Under this classification there were 340 cases, of which 183 were girls and 157 boys.

Table of the Myopes for each Age (Number and Sex).

Age:	4	5	6	7	8	9	10	11	12	13	14	Total.
Girls ...	—	2	4	13	24	18	14	34	45	26	3	183
Boys ...	—	—	3	8	17	8	15	29	41	30	6	157
Total ...	0	2	7	21	41	26	29	63	86	56	9	340

The number of cases recorded for age 14 is small, and is accounted for by the fact that 14 is the age at which the child may, and unfortunately generally does, leave school. The more or less progressive increase in the number of myopes for each age will be noted.

I had tabulated the schools from which these myopes were recruited, but the total number attending any one school was too small for any accurate conclusion to be drawn therefrom.

The two following tables deal respectively with the vision recorded before and after treatment in 397 cases, or all those of the 1,118 ordered glasses that returned for re-examination:

Vision before Treatment.

	LEFT EYE.							Total.
	$\frac{6}{60}$	$\frac{6}{36}$	$\frac{6}{24}$	$\frac{6}{18}$	$\frac{6}{12}$	$\frac{6}{9}$	$\frac{6}{6}$	
RIGHT EYE.								
$\frac{6}{6}$...	1	4	3	3	1	1	2	15
$\frac{6}{9}$...	2	—	1	5	9	22	4	43
$\frac{6}{12}$...	2	5	4	16	41	13	—	81
$\frac{6}{18}$...	3	7	8	31	20	2	1	72
$\frac{6}{24}$...	5	9	32	21	13	2	3	85
$\frac{6}{36}$...	4	19	18	8	4	3	1	57
$\frac{6}{60}$...	25	8	5	2	4	—	—	44
Total ...	42	52	71	86	92	43	11	397

Vision after Treatment.

	LEFT EYE.							Total.
	$\frac{6}{60}$	$\frac{6}{36}$	$\frac{6}{24}$	$\frac{6}{18}$	$\frac{6}{12}$	$\frac{6}{9}$	$\frac{6}{6}$	
RIGHT EYE.								
$\frac{6}{6}$...	2	3	1	2	10	16	93	127
$\frac{6}{9}$...	—	1	—	4	16	98	25	144
$\frac{6}{12}$...	1	—	1	10	53	15	9	89
$\frac{6}{18}$...	—	—	1	8	7	2	1	19
$\frac{6}{24}$...	—	—	2	1	1	2	2	8
$\frac{6}{36}$...	—	1	1	—	—	4	2	8
$\frac{6}{60}$...	—	—	—	—	1	1	—	2
Total ...	3	5	6	25	88	138	132	397

I am indebted to Messrs. Lang, Treacher Collins, and Hepburn for kindly permitting me to publish this report.

TRANSITORY BIGEMINAL PULSE.

BY

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IN relation to the case of abnormal pulse rhythm described by Dr. Johnstone in the BRITISH MEDICAL JOURNAL of October 5th, it may be interesting to record the occurrence of a bigeminal pulse without any apparent cause other than the slight excitement of applying the sphygmograph.

The patient was a young man, aged 19, who had been suffering from albuminuria for more than two years; he also had enlarged glands behind his sterno-mastoids, in the axillae and left groin. The pulse was small, soft, and regular. The tension was only 97. The urine contained a few swollen renal cells, very few casts, and no crystals. With the exception of slackness he had no symptoms, and when excited showed considerable activity and would play four sets of tennis one after the other. He had no other complaint.

I saw him last on September 23rd and found his systolic tension 108 and the diastolic 60. The cardiac dullness and sounds were perfectly normal. There was still a large amount of albumen; he had several glands in the back of the neck, in the groins, and in the front of the abdomen. His pulse was quite regular, but while I was taking it it assumed a bigeminal character, as is shown by the accompanying tracings, which were taken without removing the sphygmograph from the arm.

FIG. 1.—Pulse tracing showing normal pulse and bigeminal pulse which came on while the sphygmograph was still on the arm.

It is difficult to explain the exact causation of this alteration in the pulse beat, but it gives me the impression of being due to the interference of two rhythms. We

FIG. 2.—Diagram showing increase of sound or light by coincidence of waves.

FIG. 3.—Diagram to show abolition of sound or light by interference of waves.

know that sounds or lights generally strengthen each other so as to cause a brighter light or a louder sound

because the waves of which light and sound are composed become superimposed one upon another, as in Fig. 2. But if, by chance, one sound or one light is thrown half a wave length behind the other, the two sounds make a silence, and once when I was a student in 1862 I actually saw the late Professor P. G. Tait of Edinburgh show the experiment of two lights making darkness (Fig. 3). When two rhythms do not quite coincide, beats of alternate strength and weakness are produced, as shown in Fig. 4,

Fig. 4.—Diagram to show the effect of interference of waves of different wave lengths. The two systems having a relation of about 4 to 7 are shown above, and their resultant below. (From Ganot's Physics.)

and I think that in Dr. Johnstone's case and mine the peculiar rhythm of the pulse was due to the partial interference of two rhythms—muscular or nervous, or both—though the exact *modus operandi* may require a prolonged investigation and discussion.

EIGHTIETH ANNUAL MEETING

OF THE

British Medical Association.

Held in Liverpool on July 19th, 20th, 22nd, 23rd, 24th, 25th, and 26th.

PROCEEDINGS OF SECTIONS.

SECTION OF OPHTHALMOLOGY.

EDGAR ATHELSTANE BROWNE, M.Ch., President.

PRESIDENT'S INTRODUCTORY REMARKS.

MR. EDGAR BROWNE limited his introductory remarks to an expression of welcome to those present, and a few references to the work to be performed, and then called for the opening of the first discussion.

DISCUSSION ON CHRONIC IRIDO-CYCLITIS.

OPENING PAPERS.

I.—GEORGE COATS, F.R.C.S.,

Assistant Surgeon, Royal London Ophthalmic Hospital.

It is not my purpose to attempt a review of all the forms of chronic irido-cyclitis, but rather to select a few points here and there in order to elicit opinions on them. First, I should like to examine some recent views on autointoxication in relation to eye disease; I shall then speak of the exceedingly chronic form of cyclitis which is associated with heterochromia and cataract. Afterwards I shall mention briefly one or two points in connexion with sympathetic ophthalmitis.

A marked tendency of recent opinion is to seek the cause of chronic irido-cyclitis in poisoning either from a septic condition of the teeth, or from an abnormal state of the intestinal canal. The first of these I shall not discuss, as I believe Mr. Ormond intends to deal with it. The second, autointoxication from the intestinal tract, has been the subject of a good deal of discussion in recent years. Obviously the explanation must be received with a good deal of circumspection, for it is one which can be assumed in almost any case, and positively disproved in

almost none. An evident fallacy lies in the great prevalence of constipation and intestinal putrefaction, which would render their association with irido-cyclitis in a certain number of cases not surprising, even although, in fact, no connexion existed between the two.

It may be asked, If intestinal putrefaction is the cause of irido-cyclitis why is the one so common and the other relatively so rare? This point, however, does not really tell against the explanation, for there are many instances in pathology to show that the condition of the tissues as regards resistance may be as important as the presence of the poison; even if irido-cyclitis were caused by intestinal putrefaction therefore we should not expect it to occur in every instance of that condition, any more than we should expect every one who swallows tubercle bacilli to become tuberculous. On the other hand, it would obviously tell against the autointoxication hypothesis, or at least against its universal application, if cases occurred in which there was no intestinal putrefaction. It would be interesting to hear the experience of members on this point. One meets sufficiently frequently with cases of chronic irido-cyclitis in which there is no constipation, but then an absence of constipation does not necessarily imply an absence of intestinal putrefaction. I can quote one case of typical chronic cyclitis, however, in which a careful examination of the faeces by Dr. S. H. Browning, both with regard to the completeness of digestion and the presence of an abnormal bacterial flora, was quite negative. The patient also had no pyorrhoea, the Wassermann reaction was negative, and his general health was quite good.

It would be a desirable link in the chain of evidence if the same microbe could be cultivated from the eye and from the faeces, or should this be too much to expect, if clear evidence could be brought forward of improvement in the eye condition under the use of a vaccine prepared from the faecal flora.

In recent years an attempt has been made to approach this question from a more scientific standpoint. One of the results of intestinal putrefaction is the excessive production of conjugate, or ethereal sulphates—combinations of sulphuric acid with organic radicles. To a member of this group—indoxylsulphuric acid—the name "indican" has been given, because indigo blue can be formed from it by means of agents which free and oxidize the indoxyl (Jaffé's test, Obermeyer's test). Minute traces of these substances are found in the normal urine, but it has been suggested that a persistently increased reaction may be a valuable indicator of the presence of intestinal putrefaction. It is not suggested that the sulphates themselves cause autointoxication; indeed, it does not seem to have been proved that any of the known products of intestinal putrefaction give rise, when injected experimentally, to symptoms analogous to those usually attributed to autointoxication.

Using this reaction, and also the therapeutic test of improvement with suitable treatment, Elschnig has sought to prove that enterogenous autointoxication plays a part not only in chronic irido-cyclitis and recurring iritis, but also in migraine, marginal ulcer, scleritis, episcleritis, occasional cases of glaucoma, and possibly some instances of post-operative infection, choroiditis, cataract, and recurring hordeolum. De Schweinitz supports some of these claims, and discusses the influence of intestinal catarrh on the etiology of tobacco amblyopia. On the other hand Uhthoff, writing earlier than these authors, takes a much more conservative view, and considers that the influence of autointoxication has been proved only for cases of paralysis of the ocular muscles, especially the ciliary muscle, as, for instance, in paralysis of accommodation from ptomaine poisoning.

In a recent work also v. Hippel attacks Elschnig's conclusions. He states that it is not universally agreed that indicanuria is evidence of autointoxication. Among 416 miscellaneous cases 16 gave a positive indican reaction, and most of these positive results occurred in patients suffering from a variety of conditions—wounds, ulcers, etc.—with which autointoxication can have no causal relationship; in two cases, one of scleritis and one of irido-cyclitis, in which the reaction was positive, a tuberculous etiology was almost certain. In considering these results it is to be remarked, in the first place, that 16 positive reactions out of 416 is not a very large number. If it could be shown that in chronic irido-cyclitis the