While the tumour is essentially simple in nature, the microscopic appearances suggest the possibility of rapid growth or rapid recurrence following incomplete removal.

Conclusion

It is generally agreed that haemangioma, particularly the cavernous type, is among the commonest of the orbital tumours, and that its complete removal is usually practicable. Points of note in this case were the early numbness and neuralgic pain around the eye, unaccompanied by any sensory changes, and the evidence of pressure on the optic nerve, reflected in loss of the nasal peripheral visual field, though the optic disc was normal on ophthalmoscopic examination.

Injury to the nerves supplying the iris, ciliary body, and lateral rectus, was an unfortunate sequel to the operation, though probably this was unavoidable in the mobilisation of a tumour of this size in so confined a space.

OCULAR COMPLICATIONS IN RELAPSING FEVER*

BY

MAJOR J. BRUCE HAMILTON

A.A.M.C., 2ND A.I.F., ABROAD

Introduction

WHEN Mackenzie in 1830 wrote his "Practical Treatise on Diseases of the Eye," he mentioned that ocular complications occur and should be watched for in relapsing fever. In those pre-Pasteurian days no idea of the aetiology was possible, nor was it anticipated that numerous types of relapsing fever, each due to a different spirochaete, were to be revealed.

Mackenzie's description was of the cosmopolitan louse-borne type of the disease. The delayed ocular complication of amaurosis followed later by acute iritis, as described by him, is typical of the complications I shall enumerate. Also he noted carefully that the iritis recovered long before the amaurosis cleared. He saw in the epidemic in Glasgow 36 cases of ocular complication within 3 months, which is a much greater number than I have examined in the 12 months May 1941 to May 1942.

Since Mackenzie's day several cases of choroiditis accompanying relapsing fever have been reported in the literature; but it is very

* Received October 20, 1942.

doubtful if they were cases either of louse-borne or of tick-borne spirochaetal relapsing fever as we now know it. After the discovery of the various spirochaetes many writers, chiefly from Teutonic clinics, commented on the ocular complications of this disease; but so far as I am able to ascertain (and it is difficult in a theatre of war to be sure on this point), no further detailed reports have been made since the end of the last war.

The European spirochaete was the first to be differentiated and it was described by Obermeier of Berlin in 1873. Since then six further types of spirochaetes have been discovered, each causing the same relapsing type of fever with definite modifications of symptomatology. Later in this paper I shall briefly sketch the parasitology of relapsing fever, and it will be then seen that a still clearer differentiation of the various spirochaetes is very necessary. This clearer differentiation is urgently needed, for it is becoming very apparent that the complications of one are not the complications of the other. It may also explain why, in 92 cases seen at the 7th Australian General Hospital between May 5, 1941 and May 5, 1942 of which 28 cases came from the Western Desert and Libya, and 63 came from Syria, only four cases of iridocyclitis were seen, all from the Western Desert and Libya. This fact will be stressed further.

Ocular Complications

Elliot (1920) and Feigenbaum (1940) mention the following ocular sequelae of relapsing fever:—

1. Oedema of the lids.
2. Iridocyclitis with vitreous opacities.
3. Optic neuritis.

But in my limited experience of the disease I have found only fibrinous iridocyclitis with gross vitreous exudate of alarming proportions which may manifest itself in two forms:—

- (a) Acute iridocyclitis with gross vitreous exudate (Cases 1-3).
- (b) Chronic cyclitis with gross vitreous exudate accompanied by persistent headache (Case 4).

Both these types have an excellent prognosis and appear to run approximately the same prolonged course. It could be expected that choroiditis would be revealed in all affected eyes when the vitreous has cleared; but I have not found that so, nor have any nodules been found on the iris. The posterior synechiae in the former type have been very gross; but these have ruptured with

ease after mydricain injections. Quite often the exudate into the vitreous is so marked that the fundus picture is screened from view by a dense veil; but even this clears with time, leaving a normal fundus with normal vision.

Those who are sufficiently interested in this subject to have read Mackenzie's account of the Glasgow epidemic will remember that despite the subsidence of all inflammatory signs in the eyes he found that the amaurosis (undoubtedly due to the gross vitreous exudate) persisted for a considerably longer period.

The case records which are to follow will give an intimate picture of what the clinician should be prepared to find when the human eye is attacked by a spirochaete of the relapsing fever group.

Treatment

Unlike syphilitic uveitis the prognosis of relapsing fever uveitis as seen in the Middle East seems to be extremely good, and should not cause the oculist any definite concern. Heat and mydriatics are all that are required. No one has been impressed by the efficacy of the salvarsan group in affecting the course of the disease and the fact that one patient with iridocyclitis (Case No. 1), after his fourth injection of N.A.B., had a fever relapse on the following day with a temperature of 103°F. bears out this fact. What I am still asking physicians is, "can a patient have malaria and relapsing fever at the same time, and if so will the pyrexia of the former diminish the number of relapses of the latter?" I have had no satisfactory answer to date. We know malaria therapy influences the course of an infection of *spirochaeta pallida*; but will it similarly influence these distressing recurrences of Mediterranean relapsing fever?

Major Jones, of the Trans-Jordan Frontier Force, has recently given me his treatment for the relapsing fever found in the Amirate of Trans-Jordan. It is as follows:—

1. 3 c.c. Bisoxyl (B.D.H.) (bismuth oxychloride) intramuscularly every 5 to 7 days. Three injections.
2. Tablets of Stovarsol (4 grains): 4 to 6 tablets daily in courses of three days starting on the day of injection.
3. Mistura potassium iodide, and 500 mgm. daily of ascorbic acid, and 60000 international units of vitamin A daily are given during fever.
4. 100-200 mgm. of ascorbic acid, and 30000 international units of vitamin A are given when each patient is afebrile.

I have had no opportunity of testing the efficacy of his treatment on the Syrian or Libyan fevers; but I thank him for handing the

information on to me. He has found the treatment most successful, and seldom sees more than one relapse as a result.

Concurrent Complications

Here I refer to several concurrent complications which have arisen during the treatment of the nine cases recorded in this paper. They are: (a) Facial paralysis. (b) Herpes febrilis: (c) Encephalitis.

(a) Facial paralysis. I do not want to create the opinion that the 7th cranial nerve is the only one which may be involved; but it is the only affected one which I have encountered. I hear from other authorities that the 6th nerve also has been found involved. I also want to stress this fact, (which again has not come under my direct notice) that in some cases the facial paralysis becomes permanent. But I have no knowledge that keratitis e lagophthalmo has ever arisen as a consequence.

(b) Herpes Febrilis. The virus of this complaint has produced so many troublesome keratides in the Middle East following almost any pyrexial attack, that it is only to be expected that odd cases of herpetic or dendritic keratitis will occur after repeated attacks of relapsing fever. Although I have seen many such cases following malaria, sandfly fever and coryza, I have only seen one case following relapsing fever (Case 3). It must be remembered that this patient had had 3 intramuscular injections of milk, so that the keratitis may have resulted from this therapy. Such cases must be watched for, however, and if they occur it should be remembered that the virus of herpes febrilis only awaits the high pyrexia precipitated by another disease to multiply and attack the victim about the lips, maxilla, lids or cornea. I hope to record these observations in a later paper (*see p. 80*).

(c) Encephalitis. Incessant headache due to a chronic encephalitis is a very common accompanying feature of spirochaetal iridocyclitis; but it too responds to rest and patience, and should not be confused with more serious cerebral lesions of a pyaemic or a neoplastic type. (Cases 6 to 9).

Parasitology

Professor S. Adler of Jerusalem has been good enough to help me with this section by loaning me unpublished articles from his Department at the Hebrew University. I also must acknowledge reference to Hewlitt & McIntosh (1932) Stitt, Clough & Clough (1938) and Manson (1940).

Manson (1940) has tabulated the seven known spirochaetes of relapsing fever as follows:—

Spirochaete	Site	Natural Transmitters
1. Recurrentis or Obermeieri or Noyvi ...	{ N. and S. America ... N. and W. Africa ... North China ... Europe ... India ...	Lice
2. Persica ...	Iran ... N.W. India ...	Ticks
3. Sogdianum ...	Palestine ...	Ticks
4. Hispanica ...	South Spain ... Morocco ...	Ticks
5. Duttoni ...	Central Africa ...	Ticks
6. Turicatae ...	California ...	Ticks
7. Venezuelensis	Central and S. America	Ticks

A.I.F. Technical Instructions No. 16 designates the Palestine and Syria spirochaete as Sogdianum, and the Libyan and Western Desert spirochaete as Hispanica. Adler refers to the Syrian and Palestine organism as Spirochaeta Persica. It is thus obvious that there is some lack of uniformity in the nomenclature.

But let me turn to the figures of cases from the two regions east and west of the Nile admitted to the 7th Australian General Hospital.

Cases from the Western Desert—28. Of these four developed iridocyclitis.

Cases from Syria—63. Of these none developed iridocyclitis.

Cases from Greece and Crete—1.

We know conclusively that the Syrian and Palestinian disease is carried by ticks found in the many caves especially about Sidon. We are not yet clear as to the name of the spirochaete; but whatever be its true name it does not appear to produce iridocyclitis with any readiness.

On the other hand, the natural transmitter of the Western Desert spirochaete is very doubtful, and further the type of spirochaete seems very uncertain. We know from the century-old figures of Mackenzie that the louse-borne fever is very prone to ocular complication. He saw 36 cases in three months in Glasgow in 1843. During the years 1843 and 1844, out of 1,877 eye admissions to the

Glasgow Infirmary, no less than 261 cases suffered from ophthalmitis following relapsing fever.

The guinea-pig test has been held specific for the tick type; but Adler says "whereas infectivity for guinea-pigs is a practical proof that the spirochaete concerned is not louse-borne, the reverse is not of universal application." Further, he quotes Nicholls and Anderson who have suggested that in North Africa the same spirochaete may be carried by either louse or tick. This last remark may throw light on the confusion of spirochaete and vector in the Western Desert, and at least allow for my theory (gleaned alone from clinical findings) that the Western Desert fever is louse-borne and the Syrian is tick-borne. If this is so it explains the above quoted disparity in ocular complications from the two groups.

Diagnosis

As the condition of iridocyclitis often follows the last relapse of fever it has been difficult, even with the thick drop blood method or in the cerebrospinal fluid, to discover the spirochaete in the film of some of my patients. In two of the four patients with iridocyclitis the spirochaete was never found.

The following two methods have recently been suggested to me; but have not so far been given an adequate test.

1. If the organism cannot be located in the blood or the cerebrospinal fluid, the spirochaete may sometimes be recovered from the bone marrow following sternal puncture.

2. Even more successful is the following: A small quantity of blood is run into a little citrate solution. The specimen is then centrifuged and separates into three layers. A tiny, fine pointed pipette is used to remove material from the central "white" layer, and this is examined for spirochaetes.

Case Records

1-3 ...	...	...	Acute Iridocyclitis.
4 ...	...	...	Chronic Cyclitis and Encephalitis.
5 ...	...	...	Facial Paralysis.
6-9 ...	...	...	Encephalitis with Headache.

CASE 1. Male, aged 23 years

April 4, 1941. Admitted to 8th British General Hospital: Pyrexia, general aches, scattered rhonchi left chest. Had been in Western Desert. Pulv. Doveri gr. V and Aspirin gr. X nocte, Mist. Expectorant t.d.s.

April 13, 1941. Generalised aches and pains, plus temperature.

April 16, 1941. M & B 693 tabs. 2 t.d.s. Temperature normal. Improved.

April 18, 1941. Transferred to 9th British General Hospital. Admitted as Coryza. Temp 98.4. Recovered. No further treatment required.

April 22, 1941. Transferred and admitted to 2nd Australian General Hospital. Feels well but weak. Nothing abnormal detected.

April 25, 1941. Temp. elevated. Spirochaete recovered from blood.

May 6, 1941. Slight relapse.

May 29, 1941. No further relapse.

June 4, 1941. Admitted to 7th Australian General Hospital. Has had trouble R.E. for few days. Went to 1st Aust. Conv. Depot one week ago, after relapsing fever. R.E. became painful with poor vision next day. Following day could hardly see at all from R.E.

Examination:—Injected ++. Cells on posterior corneal surface. Post synechiae ++. Pigment on anterior lens capsule. Good reflex. No fundus details. R.V.=<6/60, <J.12. L.E.=Normal. L.V.=6/5, J.1.

R.E., Unguentum Atropine and Cocaine 2 per cent. Steaming.

June 5, 1941. Temp. 103°F.

June 6, 1941. Pupil fully dilated. N.A.B. 0.45 gm. Temp. 103°F.

June 8, 1941. Temp. 99°F.

June 10, 1941. Pupil dilated. Much vitreous opacity. Optic disc not clearly seen N.A.B. 0.6 gm. Temp. 99°F.

June 11, 1941. Fundus seen through vitreous exudate seems very pale. No active retinitis seen.

June 14, 1941. N.A.B. 0.6 gm.

June 18, 1941. N.A.B. 0.6 gm.

June 19, 1941. Vitreous much clearer. Temp. 103°F.

June 20, 1941. N.A.B. 0.6 gm.

June 21, 1941. Temp. 98°F.

June 25, 1941. R.V. 6/9, L.V. 6/5. Fundus of right eye appears normal.

June 28, 1941. Cease steaming. Continue gut. atropine 1 per cent. thrice daily.

July 3, 1941. R.V. with +0.75 D.sph.=6/6 pt. Add +4.0 D.sph.=J.1. L.V. 6/5; J.1. R.E. Old k.p. Pupil fully dilated. Vitreous opacities clearer. Fundus normal. L.E. No k.p. Optic disc is normal.

July 4, 1941. Discharged to 1st Aust. Conv. Depot.

CASE 2. Male, aged 37 years

October 16, 1941. Left eye has been bloodshot for three days. Pain in head and eyes for the past two months. Came on during relapsing fever. Was in Tobruk. No spirochaetes found in blood film while in 4th Aust. Gen. Hospital on September 7, 1941, but has had 3 attacks of fever since August 1941.

Examination:—R.V. 6/6, J.1 at 16". L.V. 6/36, J.12 at 6". R.E. Normal. L.E. Injected ++ Cornea bright. Pupil active to light. New vessels on iris. Optic disc not seen (undilated).

Atropine 1 per cent. left: Pupil dilates irregularly with posterior synechiae nasally. Optic disc normal; but fundus not clearly seen owing to vitreous opacities.

Treatment. Ung. atropine and cocaine 2 per cent. and steaming left eye.

October 18, 1941. Left subconjunctival mydrin, 5 minims.

October 20, 1941. Pupil fully dilated.

October 24, 1941. 240 lymphocytes but no spirochaetes in cerebrospinal fluid. No spirochaetes in blood. Temp. 101°F.

October 27, 1941 Temp. normal.

November 3, 1941. Kline negative.

November 5, 1941. Refraction left eye under atropine: with +0.75 D.sph. +0.25 D.cyl. ax. 45° = 6/6. Fundus normal.

November 10, 1941. Discharged.

CASE 3. Male, aged 38 years

April 27, 1941. M.R.S. (1) Sacro-iliac strain. (2) Pyrexia of unknown origin.

May 16, 1941. At 2nd A.G.H. Fibrositis.

September 27, 1941. Fever, followed by two relapses in November and December. Patient had been at Tobruk.

February 6, 1941. Admitted to 7th Australian General Hospital. Left eye has been sore for a week. Scum over pupil.

Examination: R.E. Normal. L.E. Injected ++. Pupil fully dilated. Tension normal. Much pigment on anterior lens capsule. Much vitreous haze and floaters. Fundus appears normal. R.V. 6/5, J.1. L.V. 6/60.

February 8, 1941. Blood film for relapsing fever negative. Treatment, atropine 1 per cent. and steaming. Mist. sod. sal. Milk injections.

February 4, 1942. R.V. 6/6 J.1. L.V. 6/60. Not improved. Left eye shows gross vitreous opacities, but is white.

March 4, 1942. R.V. 6/6. L.V. with +4.0 D.cyl. ax 150° = 6/18.

March 17, 1942. Increase in vitreous floaters. One tooth removed.

March 30, 1942. Left eye red. Marginal keratitis. Vitreous clearer. Treated with silver nitrate 2 per cent. paintings for three alternate days.

April 8, 1942. Cease atropine. Eye excellent.

April 13, 1942. Left eye with -2.50 D.sph. $+3.0$ D.cyl. $75^\circ = 6/18$ pt., J.2 at 12". Cornea clear. Pupil contracted. Vitreous clearer. Fundus appears normal.

April 19, 1942. Left eye red, multiple corneal erosion + + + silver nitrate 2 per cent. painting for 3 successive days.

April 25, 1942. Patient discharged to 6th Australian General Hospital.

CASE 4. Male, aged 39 years

October 21, 1941 to January 17, 1942. Initial fever on day of admission to 2nd Australian General Hospital. Had come from Tobruk. During fever had severe headaches and was relieved by lumbar puncture. On October 28, 1941, cerebrospinal fluid pressure was 240 mm.; cells 170 leucocytes per cmm., mainly lymphocytes were found increased to 70 mgm. per c.c. Spirochaetes were found in blood smear during fever on October 22, 1941; but no type mentioned. Had four relapses on November 6, 28, December 5 and 16.

February 3, 1942. Admitted to 7th Australian General Hospital. No fever for one month. Last five days has had a scum over right eye. No trouble otherwise. Never wore glasses.

Examination: R.E. White ring on cornea. Cells on posterior corneal surface. Gross vitreous opacities but no fundus lesion seen. Pupil dilates regularly. L.E. Normal. R.V. 6/9 J.1. L.V. 6/6 J.1.

February 11, 1942. Increased vitreous floaters.

February 17, 1942. Headaches severe.

March 3, 1942. R.V. with $+0.75$ D.cyl. ax. $90^\circ = 6/5$. L.V. $= 6/5$.

March 11, 1942. Right vitreous opacities still marked. Both fundi normal.

CASE 5. Male, aged 25 years

September 15, 1941. Admitted to 1st Australian General Hospital with relapsing fever contracted at Tobruk. Persistent headaches followed. Facial paralysis started October 7, 1941. Has had five relapses; last one January 1, 1942. Spirochaetes found in blood.

March 7, 1942. Admitted to 7th Australian General Hospital. Headaches unchanged, but facial paralysis is cured.

Examination: R.V. 6/5, J.1. M.H. 0.50. L.V. 6/5, J.1. M.H. 0.50. Maddox rod: 0-0. Maddox wing: 4-6 exo. -0. R.E. Normal orbicularis. Good corneal sensation. Eye normal. L.E. Weak orbicularis. Poor corneal sensation. Eye normal. Refraction under 2 H. and C.: R.E. with +0.50 D.sph./+0.25 D.cyl. 80°=6/5. L.E. with +0.50 D.sph.=6/5.

Comment:—No ocular cause for headaches which are undoubtedly due to relapsing fever encephalitis. No treatment for facial paralysis indicated.

CASE 6. Male, aged 23 years

February 16, 1942. No injuries prior to enlistment. Stretcher bearer. Contracted relapsing fever in Tobruk. Has had five attacks since September 1, 1941. Last attack one month ago. Pains at back of both eyes for past month; but no relation to reading. Spirochaetes found in blood.

Examination: R.V. 6/5 and J.1. L.V. 6/5 and J.1.

No muscle imbalance. Convergence normal. Under homatropine and cocaine: R.V. with +0.50 D.sph.=6/5. L.V. with +0.50 D.sph.=6/5.

Right fundus shows several small patches of old retinitis and one large recent patch below. Left fundus normal.

Comment:—This patient has an active retinitis possibly due to relapsing fever; but not typical. Kline test and rest advised.

March 17, 1941. Kline test was negative. X-ray of eye negative. Retinitis is unchanged and is certainly not due to relapsing fever. Headache due to encephalitis.

CASE 7. Male, aged 32 years

January 22, 1942. An out-patient. Present general health good. Contracted relapsing fever in Syria. Had about eight relapses. Since then eyes ache and gets headaches after reading or close work. Has never worn glasses. Spirochaetes in blood.

R.V.=6/5 and J.1. L.V.=6/5 and J.1.

Dilated, right fundus shows in temporal periphery a small patch of retinitis with corresponding vitreous exudate. May be a sign of relapsing fever; but needs no further treatment.

January 26, 1942. After refraction under homatropine and cocaine, and post mydriatic test, prescription given for constant wear as follows:—R. +0.25 D.sph./+0.50 D.cyl. 90°=6/5 and J.1. L. +0.25 D.sph./+0.50 D.cyl. 90°=6/5 and J.1.

It is very doubtful if these headaches are due to a refractive error—most possibly they are due to encephalitis following the spirochaetal infection.

CASE 8. Male, aged 24 years

March 30, 1942. Bad headaches since contracting relapsing fever in Syria, extending from June to December 1941. Print blurs on reading. Had 12 relapses. Spirochaetes found in blood.

Examination: R.V. 6/6 and J.1. L.V. 6/6 and J.1.

Maddox rod: 4 exophoria -0. Maddox wing: 6 exophoria -0. Rather poor voluntary convergence. Fundi normal.

Refraction:—Right, with +0.50 D.sph./+0.25 D.cyl. ax. 175° = 6/5. Left, with +0.50 D.sph./+0.25 D.cyl. 50° = 6/5.

Constant glasses ordered to relieve headaches which, however, are primarily due to the relapsing fever encephalitis.

CASE 9. Male, aged 25 years

April 8, 1942. Contracted relapsing fever at Tobruk in July 1941. Spirochaetes isolated. Had four attacks. No eye complications but severe headaches ever since.

Examination: R.V. = 6/9 and J.1. L.V. = 6/6 and J.1.

Maddox rod: 0-0; Maddox wing: 4 exophoria -0. Poor convergence. Gross old anterior retinitis in right and left fundi. No vitreous opacities. Retinitis most probably the result of boxing rather than due to relapsing fever.

April 13, 1942. Correction after refraction and post mydriatic test: Right, with +0.25 D.cyl. ax. 75° = 6/5 most J.1. Left, with +0.25 D.sph. = 6/5 and J.1.

Spectacles not required. Fields full each eye to 5/333 white. Headaches due to chronic encephalitis after relapsing fever, and accentuated by a convergence insufficiency. For orthoptics.

April 15, 1942. Kline negative. Headaches showing marked improvement with convergence stimulation.

Comment on Nine Cases

These cases I think illustrate several points:—

1. That although we had 92 patients suffering from relapsing fever at 7th Australian General Hospital, every one whom I saw with iridocyclitis contracted his fever in Tobruk (Libya) or the Western Desert.

2. Although there were four cases of iridocyclitis, all having very similar features, in only two of these was the spirochaete isolated. When ocular complications arise, the febrile period has usually (but not always) passed. In the afebrile period it is often impossible to find the spirochaete even in the cerebrospinal fluid or thick film method. I have no doubt that all four cases were of the same clinical entity.

3. The four patients with iridocyclitis had respectively 5, 3, 3 and 5 relapses before the cyclitis appeared, giving an average of four attacks before onset.

4. Two of the four patients with uveitis had relapses following the onset of iridocyclitis. One had two such attacks and the other had one.

Case	Attacks before iridocyclitis	Relapses after
1	5	2
2	3	1
3	3	—
4	5	—

Four cases averaging 4 and 0.75

5. In this small series all cases were unilateral, although Elliot (1920) quotes one bilateral case from literature.

6. Although two cases showed retinitis, in both cases this was inactive and I am certain had no relation to the spirochaetal fever.

7. No case of optic neuritis was seen although 92 cases of relapsing fever passed through the hospital in the year.

8. One patient with facial paralysis came under observation; but he is recovering. This is not always the result, and in at least one of our 92 cases permanent bilateral facial paralysis resulted.

9. Headaches are a very worrying complication both with and without ocular complications of iridocyclitis. The iridocyclitis produces little pain; but the encephalitis is persistently aggravating. When it occurs without apparent ocular disease, the patient is often referred for a refraction opinion as illustrated in several of my cases.

10. The percentage of iridocyclitis following relapsing fever is difficult to determine; but possibly it is in the vicinity of 20 per cent. from Western Desert cases and nil from Syria. As the complication is a delayed one the affected persons are often treated in one hospital for the initial disease, and in a second one for the iridocyclitis.

11. I have no helpful suggestions regarding the prevention of ocular complications, or of lessening the severity of the attack; but in my experience the visual result is nearly always satisfactory. The lack of response of our patients to intravenous injections of N.A.B. is disappointing and we must look to our research workers and pharmaceutical chemists for help in this direction.

Acknowledgement

In conclusion, I thank many members of the staff of the 7th Australian General Hospital both medical and clerical for their help and advice in treating these cases and preparing these notes. Also I acknowledge my gratitude to our Commanding Officer for permission to publish these case reports.

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NOTES ON FORMS OF KERATITIS PRESUMABLY DUE TO THE VIRUS OF HERPES SIMPLEX

BY

MAJOR J. BRUCE HAMILTON

A.A.M.C.—ABROAD

Introduction

THE idea of writing these notes has been germinating for some considerable time but it has been brought to the surface by many facts and figures, both written and verbally communicated to me recently. Besides these intimations I have been able to make certain personal observations myself, in the Middle East, on this particular subject and these should be noted immediately.

In the *Brit. Jl. of Ophthal.* of January, 1941, I recorded a survey of superficial punctate keratitis in Tasmania over an 8½ years period. I here clearly showed from recent clinical and laboratory observations that this disease, at least, is due to the virus of herpes simplex. At the same time I indicated that superficial punctate keratitis in man was accompanied by a number of other corneal lesions, but these two facts do not yet seem to have entirely grasped the attention of my readers, nor have other important facts in my paper been appreciated. One has only to