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GLAUCOMA, WITH SPECIAL REFERENCE TO MEDICAL ASPECTS AND EARLY DIAGNOSIS*

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The reference in the title of this discussion is very wide, and this paper is concerned only with clinical experience in connexion with that form of primary glaucoma known as chronic or simple glaucoma. By primary glaucoma is meant increased intraocular pressure of unknown causation. The forms of glaucoma referred to as subacute, acute, congestive, or inflammatory are to be regarded as exacerbations in simple glaucoma, from which they differ only in violence but not in essential nature. If the exacerbation is slight it leads merely to slight discomfort and the presence of haloes, symptoms which have been called prodromata. If the exacerbation is rapid and severe, venous obstruction may be brought about and the more violent forms of glaucoma arise. It is an advantage to the patient if he should have slight occasional rises of pressure—especially in the earlier stages—as in this way timely attention may be directed towards the disease.

The cause of glaucoma is still unknown. As predisposing causes many conditions are recognized, such as hypermetropia, shallowness of the anterior chamber and narrowness of the angle, and small size of the cornea. Some of these conditions—for example, shallowness of the anterior chamber—are often the result rather than a cause of glaucoma, and others are manifestations of hereditary imperfection in the development of the eye. None of these conditions cause glaucoma, though they may render its occurrence more probable.

The chief features present in glaucoma cases are, first, that the patients are elderly, and secondly, that they are of an anxious, nervous temperament. When glaucoma occurs in the young there is usually a family history, indicating the predominance of hereditary factors. It is a disease which is common in all classes of society, and is therefore largely independent of nutritive or hygienic conditions. It is also important to note that both eyes are always affected, though the intervening time interval may be considerable. We must therefore regard glaucoma as a disease of the patient and not a disease of the eye, and the recognition of the glaucoma patient before he exhibits a glaucomatous eye may, in the future become an important, though, no doubt, a difficult and uncertain, part of the early diagnosis of the condition. At the present time the diagnosis can hardly be made in this stage unless accidentally during routine examination.

Symptomatology and Diagnosis

The most usual symptom of which the patient complains is the presence of recurrent dimness of vision in one eye. This may last for a few hours or for a day, but disappears

spontaneously. Other symptoms, such as discomfort in the eye, watering, loss of accommodation, and diminution of light sense may be present, but in comparison with diminution of vision and haloes are of much less value in diagnosis. These symptoms indicate exacerbations of pressure, and may be present for many years before a subacute or acute attack of glaucoma occurs, or before cupping of the disk or changes in the field of vision develop. In relation to acute glaucoma they have been called prodromata, but they are clearly signs of the established presence of glaucoma rather than of its commencement. Glaucoma commences without symptoms. That this is true may be inferred from the results of careful and prolonged study of the tension of the normal eye in cases in which definite glaucoma is present in one eye.

The early diagnosis of glaucoma depends upon the correct interpretation of the patient's symptoms and the early recognition of the presence of hypertension. It is essential that the most careful attention should be given to the patient's account of his symptoms, and any inclination to belittle them or explain them away should be resisted.

Tension Changes

In the most insidious type of glaucoma the rise of pressure is so gentle and so gradual that no symptoms whatever may be produced until the later stage. The pressure is now higher and the field of vision is failing, but even the slightest exacerbation may be absent, and no discomfort or haloes occur to attract attention.

In these cases the disease, often in a relatively advanced stage, may be first noticed during the routine examination for glasses, or the patient may accidentally find that he does not see well with one eye. Unless discovered in some such way the presence of the disease escapes detection. It is in cases in which the patient has symptoms but the objective signs are absent or inconclusive that the problem arises in practice.

In such instances the tonometer should be freely used. My experience has led me to believe that this valuable instrument is not used as often as it should be. The standard of normal tension by the original Schiötz tonometer was estimated in 1917 by Cridland¹² as just above 20 mm., but my personal experience suggests that it is usually slightly lower. A much more difficult point is the maximum standard for non-pathological cases. This has been stated to be about 25 mm., but here again I am very suspicious of eyes which show a tension of over 22. Even if the tension should be found quite normal glaucoma is not excluded. This is explained by Löhlein¹³ on the ground that the intraocular pressure rises during the night and may have fallen at the time that the examination is made, and he states that this diurnal variation should not

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exceed 5 mm. in healthy eyes. I have no experience of testing suspicious cases in the early hours of the morning, a procedure which is usually inconvenient and often impracticable, nor have I any personal knowledge of cases in which this method proved helpful.

A plan which I have adopted in several cases with apparent success is the administration of eserine. In the normal eye a drop of eserine solution produces only a slight, if any, alteration in tension, but in many glaucomatous eyes, in which the tension does not appear to be unduly high, a distinct fall occurs within an hour. When this occurs the tentative diagnosis of glaucoma is strongly supported. On the other hand, I have not found that temporary mydriasis causes an increase of the tension in such cases, and I have no hesitation in dilating the pupils, if necessary, with a drug such as euphthalmin. (It is interesting to note that in secondary glaucoma a moderately high tension appears to be tolerated much better than in primary glaucoma. Is it possible that the explanation is that in secondary glaucoma the patient is not a glaucoma patient although he has a technically glaucomatous eye?)

Field Changes

Another sign in glaucoma, which is sometimes of value in diagnosis, though as a rule of more importance in prognosis, is the typical change in the field of vision. It is hardly necessary to point out that field changes, while they may occur at a relatively early stage in the patient's history, cannot be initial symptoms, and can only be present when the glaucomatous process has existed for a considerable period. Only very rarely can even the earliest field changes be found before ophthalmoscopically visible alterations are present in the optic disk. From cases sent for consultation one obtains the impression that in eyes in which suspicious symptoms are present the elicitation of the field change referred to as "enlargement of the blind spot" is regarded as of value in determining the diagnosis. The diagnostic value of field changes in glaucoma is relative, and depends on the presence of other symptoms, such as tension. Many years ago the statement "no scotoma, no glaucoma" was widely believed, and in its more recently modified form "no enlargement of the blind spot, no glaucoma" still claims adherents.

Since field changes in glaucoma can only occur as a result of the glaucomatous process, there is a period as long as eight or ten years or more, following the beginning of the disease, during which no field changes are present. This period may be called the *pre-perimetric stage* of glaucoma. The more expert we become in detecting the earliest signs of field alteration the shorter will be the pre-perimetric stage. Similarly, the point at which glaucoma can be diagnosed in the pre-perimetric stage will depend upon our skill and judgement in ascertaining and assessing the value of other symptoms and signs, which the patient may present if he asks for advice in this stage. The more easily we become able to recognize glaucoma in the pre-perimetric stage the more we shall find that the chief value of the field changes is in prognosis, and in supporting a diagnosis already made rather than in initiating it.

Apart from confirming an opinion already formed, perimetry has a definite diagnostic value in a fairly large group of cases in which the usual clinical picture of glaucoma is in one or more important features incomplete, and yet the oculist has reasons for suspecting its presence. It is evident that in the practice of different oculists the line between definite glaucoma and suspected or possible glaucoma will not be fixed, but will vary according to the methods of examination and the attitude of mind of the individual. The less prominent the other features of the clinical picture the greater is the value of perimetry in diagnosis, and the greater the necessity for its careful

application; nevertheless, in a case in which no other signs or symptoms have been found a positive diagnosis on the strength of field changes alone should be made only with the greatest caution.

Classification of Cases

From the point of view of perimetry these cases of possible or suspected glaucoma may be considered in two chief groups, according as to whether gross field changes are present or not.

Group I: No Obvious or Gross Field Changes Present.—These cases fall into five subdivisions. (a) *Neither symptoms nor signs are present.* The suspected eye appears in every way normal and healthy, but is suspected because definite glaucoma is present in the other eye. (b) *Symptoms alone are present.* A history of haloes, periods of ocular discomfort, or other suggestive features is given, but the optic disk and tension appear normal. (c) *Ophthalmoscopic signs alone are present.* Cupping or pallor of the optic disk, alone or together, found during routine examination, is the only sign which arouses suspicion. (d) *Increased intraocular pressure alone is present.* During routine examination the tension is found to be raised, but no other symptom or sign is elicited. (e) *Various combinations* of features already mentioned under (b), (c), and (d) may be present.

Group II: Gross and Easily Detected Field Changes Present.—Here the problem is not the detection of early field changes, but the proper interpretation of well-developed field changes, and this group does not come within the consideration of the early diagnosis of glaucoma.

Investigation of Group I Cases

In the first group the question is that of detecting the earliest field changes in glaucoma. These changes will be those which are found when the weakest practically applicable stimulus is used. Changes which are found by stronger stimuli are not likely to be the earliest changes. The stimulus may be reduced by diminishing the size of the test object, by increasing its distance from the eye, and by reducing the illumination by darkening the room. All three methods may be used; if the last is used the object need not be so small.

Under the usual clinical conditions the weakest stimulus which I have found to be reliable is a 1 mm. white object used at 2 metres on the Bjerrum screen. If no defect is found with this test the examiner has been unable to elicit perimetric evidence of glaucoma on that day, but, as already pointed out, glaucoma is not absolutely excluded. If, however, other symptoms and signs are absent the eye may justifiably be regarded as healthy. The early field changes of glaucoma are of two types: (1) depression, and (2) the beginning of arcuate scotoma.

Depression of Central Field ("Baring of the Blind Spot")

The isopter for 1/2000, which normally extends to about 26 degrees all round, and includes the blind spot, is more or less contracted. It may fall within the 12-degree circle so that the blind spot is entirely outside it, or it may skirt round the inner side of the blind spot, leaving its outer or outer and upper part exposed, or, as it were, bared. It is rare to find this exposure at the lower pole of the blind spot. When the 1/2000 isopter is much contracted the baring of the blind spot may be demonstrable with an object twice the size—2/2000.

This baring of the blind spot at its outer and upper part was shown as long ago as 1906 by Sinclair,^{9 10} who drew attention to it as an early sign of glaucoma and referred to it as "Bjerrum's symptom." It is the earliest field change I have found in cases of suspected glaucoma. It would be unjustifiable to assert that it is never present

apart from glaucoma—I have, at any rate, never found it, nor has it ever been described in connexion with any other condition. A somewhat similar barring of the upper part of the blind spot occurs in the early stage of temporal hemianopia due to pituitary tumour, but its character is quite different, the defect lying more in the upper outer quadrant of the 1/2000 field.

It is interesting to note that although barring of the blind spot is undoubtedly the precursor of the arcuate scotoma, the arcuate scotoma does not appear to grow out of the barring, but seems to arise independently as a small curved scotoma on or about the 15-degree circle a little distance from the blind spot. The barring of the blind spot may be present in the upper part of the field, with an early arcuate scotoma in the lower part. When the

FIG. 1.—“Barring of the blind spot.” After Sinclair.^{9 10}

A, The left central field of a patient who had definite glaucoma in his right eye—1/2000. V. = 6/6. Peripheral field normal. No other symptoms of glaucoma present.

B, The left central field of a patient whose right eye was blind from glaucoma—2/2000. V. = 6/6. Peripheral field normal. No other symptoms of glaucoma present. Both of these fields are from apparently normal eyes in cases in which the other eye was glaucomatous. There was no abnormal tension in either case. It is not recorded whether the eyes ultimately became glaucomatous.

scotoma develops the barring seems to become merged in the depression of that part of the field, so that it is not present together with the more fully developed Bjerrum scotoma. Eserine may cause the barring wholly or partly to disappear.

CASE RECORD

Miss G. B., aged 32, was seen in June, 1925, complaining of haloes in the left eye. There were nearly seven dioptres of hypermetropia in each eye, with 6/9 vision with correcting lenses. When seen, both anterior chambers were shallow and both optic disks were normal. The tension was 15 Schiötz in the right eye and 13 in the left. The field of vision of the left eye showed depression for 1/2000, and a good field for 2/2000, but weak outside the blind spot. The field of the right eye was normal.

The patient was given meiotic drops for daily use and advised to report again soon. She did not return until nine years later, in 1934, when an attack of subacute glaucoma with high tension occurred in the left eye. In the intervening nine years there had been frequent attacks of haloes which had always been relieved by the drops, so that she had not considered it necessary to seek further advice. On the last occasion, however, the drops had not given relief. The tension of the right eye was 18. Under eserine the tension of the left eye dropped to 21-27 in six days.

In 1935, three weeks after operation (cyclodialysis) on the left eye, the conditions were as follows: right eye—vision = 6/9; disk healthy; field shows contraction for 1/2000 and a normal outline for 2/2000; tension 19. Left eye—vision = 6/12; disk healthy; field shows contraction for 1/2000 and slight weakness above and outside the blind spot for 2/2000; tension 8.5.

It is to be noted that although glaucoma was present in the left eye for at least nine years neither cupping of the disk nor “enlargement of the blind spot” developed. The diagnosis was made without difficulty in the pre-perimetric stage, and the only field change was a mere weakness above and outside the blind spot.

FIG. 2.—Central fields of the case, three weeks after operation on left eye. Contraction of isopters for 1/2000 and slight indentation above and outside blind spot for 2/2000 in left field. V. = 6/12. T. = 8.5. Right field normal for 2/2000, depressed for 1/2000. V. = 6/12. T. = 19. Under eserine.

FIG. 3.—Left central field of patient who had lost the right eye from glaucoma. T. = 37, after eserine 23. Test objects 2/2000 and 1/2000. Barring of the blind spot. V. = 6/6. Disk normal. Two years later the tension rose to 40/50 and vision fell to 6/12, but even then “no constriction of the fields and no prolongation of the blind spot” were found. (B., 1923.)

FIG. 4.—Case of suspected glaucoma. (January 14th, 1933.)

A, Left eye. V. = 6/9. T. = 23. Disk cupped. Field shows barring of blind spot for 2/2000 as well as for 1/2000. No arcuate scotoma or enlargement of the blind spot. Eserine reduced the tension to 18 in forty minutes.

B, Same field after fourteen days' treatment with eserine. Field for 2/2000 now normal. Barring of blind spot still present for 1/2000. This has remained. T. now = 18. V. = 6/18. In this case glaucoma has been present for two years at least without the development of “enlargement of the blind spot.” (N., 1933.)

FIG. 5.—Barring of the blind spot in upper field, early arcuate scotoma and nasal step in lower field. Test objects 2/2000 and 1/2000. The scotoma is unconnected with the blind spot. T. = 23 (under eserine). V. = 6/9. Optic disk cupped. The other eye showed well-developed glaucoma. Shading indicates a dim area for 2/2000. (Mc., 1935.)

The Arcuate Scotoma (Bjerrum Scotoma)

A later field change is the development of an arcuate scotoma, which usually appears between one pole, more often the upper pole, of the blind spot and the vertical margin of the field 10 to 20 degrees from the centre. It is characteristic of this scotoma that it is narrower at the end which lies towards the blind spot and widens as it passes towards the nasal side of the field. It is, as a rule,

FIG. 6.—The patient had seen many oculists in America and England before the last one diagnosed glaucoma and referred her as she wished a second opinion. Right eye: V. = 6/6+. T. = 15. Disk cupped. Field shows a typical arcuate scotoma above the nasal step, and a faint arcuate scotoma below. These changes were elicited with 1/2000; a larger object only showed the core of the upper scotoma. Had a larger object only been used the defect would have resembled a Seidel scotoma. It is to be noted that the most intense, and therefore presumably the oldest, part of the scotoma is unconnected with the blind spot. Left eye: V. = 6/6+. T. = 15. Field of 1/2000 shows slight depression, especially to outer and upper side of blind spot. Six months later her oculist reported that the changes in the right were less intense, and that the lower scotoma could not be elicited. (A., 1932.)

unconnected with the blind spot, and only establishes such a connexion in its more fully developed stages. It is therefore in no way an enlargement of the blind spot, and does not make its appearance as an out-growth from the blind spot.

Since the pre-existing retinal function is approximately the same all over the area corresponding to an arcuate scotoma near the fixation point it is unlikely that the

FIG. 7.—Right eye, early arcuate defect with nasal step which would have been missed had not a small object, 2/2000, been used. No enlargement of blind spot. V. = 6/5. T. = 25. Left eye definitely glaucomatous. (P., 1933.)

functional impairment would grow from one end to the other of a nerve fibre bundle in this situation. Such a development has never been demonstrated. In my experience the scotoma appears rapidly, and approximately simultaneously and evenly, over the area of a nerve fibre bundle when near the fixation point. Van der Hoeve,³ Peter,⁴ and Sloan⁷ support the view that this scotoma begins independently of the blind spot. I have also noticed that the early arcuate scotoma is more frequently and more easily found in the upper field.

When more fully developed the arcuate scotoma crosses the vertical meridian and terminates on the nasal side of the field on the horizontal meridian, forming the nasal step. Occasionally this nasal portion of the scotoma with the nasal step is the only part demonstrable (Fig. 8A).

Unless a test as small as 1/2000 is used it may not be possible to elicit the whole extent of the scotoma, though the denser portion may be found with larger tests. If, however, only a part of the defect is discovered misconceptions as to its extent and relations may arise. Similar misconceptions may also be due to the method of examination, especially when the test object is moved in one direction only as when a revolving scotometer is used.

FIG. 8.—From van der Hoeve.

A, Central field showing nasal end only of arcuate scotoma. No enlargement of blind spot.
B, Arcuate scotoma without enlargement of blind spot.

FIG. 9.—Case recorded by Roenne¹ as showing deep cupping, failing vision, and enlargement of the blind spot. Tension 8/7.5 Schiötz. Roenne regarded the state of the blind spot as evidence definitely against glaucoma.

FIG. 10.—Glaucoma suspected on account of deeply cupped disks and impaired vision. Tension 18 in each eye, not appreciably reduced by eserine. R.V. = 6/12. L.V. = 6/9. Blind spot enlarged and bared, but very large test objects (20/2000, 40/2000) required to show this. Peripheral field for 125/330 greatly contracted. The enlargement of the blind spot is definitely against glaucoma. The field changes indicate functional amblyopia. Two years later, *in statu quo*. (W., 1932.)

The statement that enlargement of the blind spot is a pathognomonic early sign of glaucoma is therefore unacceptable. Glaucoma may be present without alteration in the blind spot, and the blind spot may be enlarged or modified apart from glaucoma. This doctrine appears repeatedly in the literature and has obtained wide acceptance, but if its origin is investigated it will be found that the statements on which it is based occur in papers written about twenty years ago (Fleischer,⁶ Seidel²), that the test objects used were unnecessarily large, and that the findings were not adequately checked by the use of small test

objects and a large projection of the field defects. Thus the defects depicted by Seidel, for example, cannot be regarded as representing an initial stage. They were elicited by only moderately small objects and they were often present simultaneously at both poles of the blind spot, with which they were always continuous. Kirwan,¹¹ referring to glaucoma in epidemic dropsy, states that

FIG. 11.—In this case glaucoma was first discovered when the patient's glasses were being revised. There had been no symptoms. The right field shows a nasal step for 5/330, with an arcuate scotoma approaching but not reaching the blind spot. Small objects did not show a scotoma continuous with the blind spot, but 2/2000 shows baring of the blind spot and a nasal step. V. = 6/6. T. = 23. Disk deeply cupped. Left eye normal. (T., April 30th, 1925.)

FIG. 12.—The same case as Fig. 11. Five years later the blind spot of the left eye, which had been carefully watched, was found to be bared (2/2000). V. = 6/6. T. = 35. Disk normal. Anterior chamber a little shallow. Peripheral field normal. (July 30th, 1931.)

FIG. 13.—Two years later. 1/2000 shows baring of the blind spot, and a nasal step, not elicited by 2/2000. The latter object shows an indentation above dipping down towards the blind spot. V. = 6/6. T. = 23. Disk normal. Peripheral field normal. There is no enlargement of the blind spot or tuft-like scotoma. (July 1st, 1933.)

Seidel's sign can be demonstrated in the *later stages* according to the duration of the disease. In my experience the commencement of the arcuate scotoma is only discoverable by a very weak stimulus, and appears near, but not connected to, one pole of the blind spot, and not simultaneously at both poles. The scotoma of Seidel, unlike the Bjerrum scotoma, becomes narrower as it leaves the blind spot, and has a pointed termination, a differential feature to which attention has also been directed by Thomasson.⁸ The Seidel scotoma is much more suggestive of a tracing of blood vessels than a nerve fibre bundle defect.

This arcuate scotoma often appears to spring into existence very rapidly and to remain in its initial stage for a very short time before it merges into the complete Bjerrum scotoma. If this were not the case it should be possible to discover it more frequently in cases of suspected glaucoma. In no case of suspected glaucoma of the type included in Group I, which I have examined with

FIG. 14.—Nine months later. Left eye. Anterior chamber very shallow. Pupil minimal (eserine). T. = 20. Disk normal. Blind spot bared for 1/2000; for 2/2000 a weak area is present above blind spot. Field for 1/2000 now comes down almost to horizontal meridian. Small nasal step present. V. = 6/9. T. = 20. (December 18th, 1934.) Six months later, field practically unchanged. V. = 6/9. T. = 23. Eserine in constant use since 1931.

a small visual angle, have I ever found an enlargement or extension of the blind spot of a tuft or flame-like shape.

Discussion

In the first group, therefore, depression of the central field with baring of the blind spot is very suggestive of glaucoma and strongly supports the evidence afforded by the presence of other symptoms. The presence of a relative arcuate scotoma may be regarded as indicative of glaucoma which has existed for some time and has passed beyond the pre-perimetric stage.

The blind spot may be normal or may be enlarged or modified from causes other than glaucoma. Not only is this the case, but a true enlargement of the blind spot—that is to say, an enlargement with little or no alteration in shape but an increase in its diameters, as if it were viewed through a magnifying glass—(Fig. 10) is an uncommon and really accidental accompaniment of glaucoma. Enlargement of the blind spot of this type may be due to circumpapillary choroidal atrophy, which sometimes occurs in advanced glaucoma—especially in elderly people—or it may be functional. In itself it is not a positive sign of any value.

In Group II, referred to earlier in this paper, gross and easily detected field changes are present. This is a small group and the problem of diagnosis is much simpler. Well-developed field changes are never present in glaucoma without increased tension or pallor or cupping of the disk. Though in certain cases the field changes form a valuable feature of the clinical picture, and are of material assistance in the recognition of the disease, the question of early diagnosis does not arise in connexion with them and this group is consequently omitted from the present consideration.

Summary

1. Primary glaucoma is a disease of unknown origin mostly affecting elderly people of nervous temperament. It is essentially a disease of the patient rather than of the eye.
2. The various forms of glaucoma known as subacute, acute, congestive, or inflammatory are exacerbations in the course of primary glaucoma, and not different kinds of glaucoma.

3. The so-called prodromata of acute glaucoma are slight exacerbations, and are evidence that glaucoma has been present for a considerable period.

4. Glaucoma may exist for many years without producing any symptoms whatever and without producing any field changes.

5. The period between the beginning of the glaucomatous process and the development of field changes may be called the pre-perimetric stage.

6. The earliest field change produced is "baring of the blind spot."

7. "Enlargement of the blind spot" or a tuft-like or flame-shaped scotoma is not an initial field change. The arcuate scotoma is a relatively late field change and does not begin as an outgrowth from the blind spot.

8. There are three stages in early glaucoma. (1) The initial stage, without symptoms—the stage of the glaucoma patient. (2) The pre-perimetric stage, without field changes. (3) The stage presenting early field changes. The early diagnosis of glaucoma cannot be made in the first stage, but should usually be made in the second stage.

9. Glaucoma is to be diagnosed by the clinical picture as a whole, not by any one sign. The only pathognomonic sign is increased tension, and this is not always easily elicited.

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- ¹² Cridland: *Brit. Journ. Ophthalmol.*, 1917, i, 352.
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A meeting of the Tuberculosis Association will be held at 26, Portland Place, W., on Friday, November 22nd, at 5.15 p.m., when addresses will be given by Sir StClair Thomson on "Tuberculosis of the Upper Respiratory Tract" and Mr. H. R. Bickerton on "Tuberculosis of the Eye." At 8.30 p.m. Dr. E. L. Middleton and Dr. P. Heffernan will speak on "Industrialism and Tuberculosis." Further meetings of the association have been provisionally arranged as follows: In London, January 17th, 1936, Dr. Noel Bardswell, Dr. J. G. Johnstone, and Miss Marx, "After-care of the Tuberculous in London"; Dr. James Maxwell, "Intestinal Tuberculosis"; February 21st, Dr. H. H. Scott and Dr. C. H. C. Toussaint, "Primary Tuberculosis in Children and its Relation to Meningitis"; Sir Henry Gauvain and Mr. G. R. Girdlestone, "Treatment of Tuberculous Lesions of Bones and Joints"; May 15th, Sir Leonard Rogers and Dr. Andrew Morland, "Climate and its Relationship to Tuberculosis"; Mr. J. E. H. Roberts and Dr. F. G. Chandler, "Recent Advances in Thoracoscopy and Adhesion Section." Cambridge meetings: April 2nd, Dr. Hagius van den Berg (Amsterdam), Dr. R. H. Hazemann (Paris), and Dr. N. Tattersall, "Dispensary Organization"; Dr. Russell Reynolds, "Cinematography of the Chest"; annual dinner at St. John's College. April 3rd, Professor Sayé (Barcelona), "Chronic Miliary Tuberculosis"; Dr. Maurice Davidson and Dr. L. B. Stott, "Capacity for Work in Pulmonary Tuberculosis"; visit to Papworth Settlement. April 4th, cases for discussion; annual meeting.

OIL-SOLUBLE ANAESTHETICS IN RECTAL SURGERY

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It is the purpose of this communication to introduce an improved formula of anaesthetics in oil, having a prolonged action, and being painless on injection, for the treatment of painful conditions particularly in and around the anus.

Since 1927, when Yeomans, Gorsch, and Mathesheimer introduced benacol for the treatment of pruritus ani, much attention has been given to local anaesthetics having a prolonged action. These workers used a solution consisting of equal parts of para-amino-benzoate and phenomethylol in ninety parts of rectified sweet almond oil. Oil was the solvent because, owing to the slow rate of diffusion from it of the dissolved anaesthetic substances, anaesthesia and relaxation of the musculature is prolonged. In this country Gabriel in 1929 employed A.B.A. (3 per cent. ethyl-para-aminobenzoate, 5 per cent. benzyl alcohol, and 10 per cent. ether in almond oil); an alternative solution being 0.5 per cent. procaine, 10 per cent. benzyl alcohol, and 1 per cent. phenol in almond oil. These solutions often gave rise to pain on injection and very often to severe pain several hours afterwards. The anaesthetic effect and the relaxation of the anal musculature were good but rather uncertain.

During the past two and a half years trial has been given to many and varying combinations of oil-soluble anaesthetics at St. Mark's Hospital, in the search for a solution which does not produce immediate or delayed pain after injection. Details of these experiments are given at the end of this article. The following formula has proved in a large number of cases to be the best: procaine base 1.5 per cent., butyl-para-aminobenzoate 6 per cent., benzyl alcohol 5 per cent., in sterilized almond oil. This solution owes its painlessness on injection to the adequate concentration of procaine base and to the elimination of ether. Its prolonged anaesthetic property is due partly to the slow rate of diffusion from the oil and partly to the prolonged action of the butyl-para-aminobenzoate. If this solution be sufficiently warmed and injected slowly it appears clinically that anaesthesia is almost immediate.

The advantages of this solution over those that have been previously tried are: (1) Its effect is almost certain. (2) The injection is painless, if given slowly. (3) In no case has there been any severe after-pain. (4) Anaesthesia or hypo-aesthesia is produced for periods varying from seven to twenty-eight days or longer. (5) Relaxation of the anal musculature is much greater and more prolonged. (6) The preparation is comparatively non-toxic; 20 to 30 c.cm. may be injected without any general ill effect. (7) Even when a large quantity (30 c.cm.) is given on one occasion, no local reaction is observed if the injection is skilfully performed.

General Technique of Injection

Though the technique of injection has been admirably described by Gorsch, Gabriel, and others, there are certain modifications and improvements, as well as an extension of the uses of oil-soluble anaesthetics, that justify a full description of the method of injection. The large branches of the motor and sensory nerve supply of the perianal region, mainly the inferior haemorrhoidal nerves and the perineal branches of the fourth sacral nerves,