required for the needs of the thyroid gland; failing this additional supply goitre is prone to develop.

Previous work has demonstrated the importance of gastrointestinal infection (in which may be included infection of the mouth, nose, tonsils, etc.) as a goitre-producing influence. The truth of this is now generally admitted. It is therefore reasonable to conclude that goitre develops in animals confined in dirty cages because their food becomes contaminated by their own excreta. Some animals are more susceptible than others to gastro-intestinal infection carried in this way: hence the individual idiosyncrasy both to goitre and to the size of the goitre that is so apparent in such an experiment. It would seem, then, that the second fact above referred to might be restated thus: Under unhygienic conditions of life, as represented by dirty animal cages or by bacteriological impurity of the alimentary tract, an additional supply of iodine is required for the needs of the thyroid gland—or more properly this additional supply is required in order that the gland may secure a sufficiency for its needs. If it does not secure this sufficiency it hypertrophies. It is unnecessary here to go over old ground¹ dealing with the experimental production of goitre in man and animals, and with the production of its congenital manifestations in animals by faecal material or faecal bacteria, or with the curative action of intestinal antiseptics in recent goitres. What I wish to point out here is that there is nothing in the newer work which does not dovetail into the older, although it may involve some reconstruction of ideas and a wider outlook regarding the combination of causes which gives rise to goitre. Before outlining these causes the most recently discovered fact may be referred to—namely, that excess of fat favours the development of goitre. Briefly expressed, this excess involves an additional strain on the resources of the thyroid gland, and may in consequence be a determining factor in goitre production. Its effect is more marked in some cases than in others, and is more apt to manifest itself in the presence of unhygienic conditions, such as are provided by dirty animal cages or pens, and by an unhygienic alimentary tract. Here, too, the fat excess increases the needs of the thyroid for iodine, and the goitre-producing influence of fat excess can be counteracted by increasing the available iodine in the food.

It is obvious, therefore, that the factors which give rise to goitre centre round the supply of iodine and the needs of the thyroid gland for iodine. Now this supply of iodine is dependent on a multiplicity of factors both extrinsic and intrinsic to the body. It is dependent on the iodine content of the food and water; and this in its turn is dependent on altitude, distance from the sea-coast, and the iodine content of the soil from which both vegetable foods and water derive their iodine supply. Again, the supply of iodine to the thyroid gland, and of the gland's iodine-containing secretion to the body cells, is dependent upon the adequate absorption and assimilation of iodine; this in its turn is influenced by factors such as disordered function of the gastro-intestinal tract and bacterial intervention in the digestive tube, both of which may interfere with the adequate absorption and assimilation of iodine. On the other hand, the needs of the thyroid for iodine are dependent on the needs of the body cells for the active iodine-containing principle of the gland, and these in their turn are dependent upon a multiplicity of factors such as food balance (excess of fats, for example, increasing these needs), metabolic variations, age, sex, poverty, sexual activity, pregnancy, menstruation, infections, and season. There is further to be considered the capacity of the thyroid gland to utilize iodine and efficiently to manufacture its hormone; here are involved such factors as heredity and excessive stimulation of the gland, which may be induced by nervous, toxic, or infectious influences, and impairment of its function, which may be induced by hereditary influences or by toxic or microbic agents, the two last being frequently of gastro-intestinal origin.

These, then, are the chief known factors—physiological, nutritional, metabolic, toxic, infectious, and nervous—which in varying combinations may cause goitre. So it comes about that while theoretically goitre may arise in consequence of actual deficiency of iodine in the food, it is much more likely to arise, and usually does arise, when a number of factors combine to render the available iodine in the food relatively insufficient for the needs of the body under the condition of life in which it finds itself. While, therefore, simple goitre is due theoretically to insufficiency of iodine, it is due in practice to a combination of factors which together bring about this insufficiency.

It is this combination of factors—a combination which

may vary within wide limits in different individuals, at different seasons, and in different localities—that constitutes the true cause of goitre in any individual or locality. Indeed, while in one locality a given grouping of factors may serve to induce goitre in a considerable proportion of individuals of the same age, sex, and race, comparatively slight variations in this grouping may suffice to cause notable variations in the incidence of the malady, or lead only to its sporadic occurrence in another locality. Long familiarity with the experimental conditions under which goitre arises has taught me how subject to variation is the combination of factors that may be concerned in the production of goitre, and how different the results may be when variation has occurred in such factors as race, locality, season, climate, and hygienic conditions of life. It is often a matter of extreme difficulty to ensure in subsequent experiments precisely the same conditions as prevailed in previous ones. The fact that the experiments are carried out at different seasons of the year or in a different place, or that in one experiment the animal cages are more scrupulously cleansed than in another, at once introduces variations in causal conditions the influence of which may be paramount. The most we can do, therefore, in regard to the discovery of the cause of goitre, or, indeed, of any deficiency disease, is to determine the combination of conditions under which it arises in the particular individual or locality with which we are dealing. It is by a knowledge of these conditions and of their relative importance, by the rectification of all of them, and by refusing to confine attention to any one of them, that we may hope to prevent and cure goitre.

I have been prompted to write this brief outline of our present knowledge of the causes of goitre because this knowledge is now sufficiently definite to enable us to prevent and to cure the condition. It is pre-eminently one which need not exist in this country if such knowledge of its causation and of its prevention as we already possess be but applied. A single example in this connexion will suffice. In 1913 I demonstrated that the goitre prevalent in a large European school in India was associated with great bacteriological impurity of the water supply. So prevalent was it that 65 per cent. of girls and 45 per cent. of boys above the age of 16 years were goitrous, and a residence of seven or eight years in this school sufficed to cause goitre in 80 per cent. of the pupils. Measures directed to the purification of the water (first by iodine and later by chlorine) reduced the incidence of the disease by 50 per cent. in a period of six and a half months, and the subsequent introduction of a bacteriologically pure water supply has resulted in its complete disappearance. The prevention of goitre is a matter of attention to food and water supply, to individual and to general hygienic conditions of life, and to the varying needs of the body for iodine.

In this outline of the causes of goitre no attempt has been made to go into detail, my object being to provide a concise sketch of the present state of knowledge regarding its etiology.

REFERENCE.

- ¹ McCarrison: *The Thyroid Gland in Health and Disease*, London, 1917.

DETACHMENT OF THE RETINA, PROBABLY DUE TO EXPOSURE TO LIGHT DURING AN ECLIPSE.

BY

N. BISHOP HARMAN, M.B., B.Ch. Cantab., F.R.C.S.,
SENIOR OPHTHALMIC SURGEON, WEST LONDON HOSPITAL;

AND

PETER MACDONALD, M.D. ABERD.,
OPHTHALMIC SURGEON, YORK COUNTY HOSPITAL.

THERE is perhaps no eclipse of the sun without its quota of damaged eyes. Cases of injury of the usual type were seen after the eclipse of April of last year, despite the many warnings that appeared in the newspapers of the dangers of observation with the unprotected eye. Dr. Oliver Lodge described a case in the *JOURNAL* of May 14th, 1921 (p. 701), and cases of a similar nature were related in subsequent issues (May 21st, p. 756, and June 4th, p. 837) by Dr. G. Moxted and Mr. H. W. Archer-Hall. An unusual case in which detachment of retina of one eye occurred certainly as a sequel and probably as the direct effect of the eclipse exposure has been seen by us, and it appears of sufficient interest and rarity to merit record.

The patient is a professional man, aged 54, who was in the north during the eclipse, and was seen by one of us (P. M.) on April 13th. The eclipse of the sun (annular) occurred on April 8th. He gave a history which is set out below from notes by N. B. H., on whom the chief share in the care of the case devolved.

Notes made on April 13th (P. M.) show that: Vision right eye was perception of light in the upper part (less than half of field). Vision left eye was 6/6 with a small degree of hypermetropia, about 0.5 D. The right eye showed a very extensive detachment of the retina, involving the whole of the upper half of the eye, and the detachment ballooned downwards so as to obscure the disc and macula, which could not be seen. The vessels running downwards from the disc could be seen from a point which was estimated as being about one disc's breadth below the disc.

The point of greatest interest was the relationship between the patient's looking at the eclipse and the onset of the ocular trouble. He himself quite definitely assigned his looking at the sun as the origin of his visual defect. On first thoughts this view was not regarded with favour (P. M.), but on reconsideration of his history of the onset of the disease in this case; of the fact that the etiology of detachment of the retina is not established; that a violent stimulation of the retina is known to be able to cause damage to the nerve elements of the retina; that it is not unreasonable to regard such violent stimulation as competent to set up an exudation behind the retina; that in this case there was a history of such definite violent stimulation; that exposure to light sets up exudation elsewhere—for example, blistering in sunburn—we decided that we were justified in publishing the case as one of retinal detachment due to excessive exposure to sunlight during an eclipse. Since the patient's home was in London and he desired to return thither he was referred to N. B. H. for further observation and treatment.

When seen on April 15th he stated that he viewed the eclipse with the right eye through a piece of cinematograph film, and also through a tunnel formed by his hand. Half an hour after exposure he saw sparks in the eye; the sensation was worse on the next day. Three days later he noticed that part of the sight of the right eye was obscured; the defect appeared at first downwards and then to the right side. It rapidly got worse. He stated that his eyes had always been good, but that he had an impression that the right eye was always a little less keen of sight than the left. He had seen spots in both eyes some six months ago, but the appearance was only temporary.

Left eye: Vision 6/9, hypermetropia 0.5 D = 6/6 all; Jaeger 1 with additional 2 D. Right eye: Superfices normal, pupil slightly smaller than that of left eye; reacts to light. Fundus reflex dull. Examination showed a huge detachment of the retina as described above, but even of greater extent, for now only a narrow band of the normal lower part of the fundus could be seen. A very few fine vitreous opacities were visible. The patient was in an extremely jumpy and nervous state; he wife said he was always so.

Taking the statement of the association of the eclipse exposure with the incidence of the detachment of the retina at its face value, it seems that there was a probability of some damage to the macular region which alone would be sufficient seriously to injure vision. It therefore did not appear advisable to attempt to relieve the detachment by operation; another consideration supporting this decision was the experience (N. B. H.) that operations do not secure better end-results than the simpler treatment by rest; and, finally, the neurotic temperament of the patient and his strong aversion to operation were conclusive against it. The treatment adopted was: confinement to bed—he was not allowed up even for the natural offices; low diet, mainly slop food; elastic bandage to the one eye; blisters to the right temple on alternate nights; and a potassium iodide mixture.

April 28th. The patient says he sees more of things with the eye. The view of the normal lower part of the fundus is wider, but there is no view of disc or macula. Treatment continued.

May 12th. The great mass of the detachment has gone back, there are only patches of rugous retina, up and to the left, up and to the right, and in the macular region. The vitreous is slightly foggy. It is possible to see all the details of the disc and to trace every vessel therefrom in continuity.

May 26th. The patient says that the eye seems better. Vision 3/6. The vitreous is full of fine dust-like opacities. The retina is not quite so flat as on the last examination; there is a bulge up and outwards. Patient has come to the limit of his toleration of confinement to bed. Short walks allowed out of doors. Fuller diet given. Eye still to be bandaged. All other treatment stopped.

June 11th. A shallow detachment of about 4 D up and outwards, and some wrinkling inwards. In the macular region is a small white opaque elevation (the size of the disc) with a dark shadow under the summit; this appearance may be either due to a dimple or pigment—parallax suggests the former. Radiating outwards from the little hillock are a number of striding edges or folds which spread upwards into the shallow detachment. Disc seen

clearly. Vitreous haze much less. Vision 2/60; the wall of the room is seen clearly in all its parts; there is no sense of deficiency; fovea is regular. Bright light dazzles him.

June 28th. The small elevation in the macular region is of less size, and there are fine glistening lines radiating out from it; the striding edges that pass outwards to the remains of the detachment are sharper. Vitreous clear. Vision 1/60. Patient allowed to return to work three days a week.

August 31st. Conditions unchanged, patient working four days a week. "Eye does not bother him, but he does not see so well with it."

November. Report received to same effect.

The sequence of events may be fairly summarized as follows: 1. Severe damage to the macula consequent on exposure to sunlight. 2. A succeeding infiltration of the macular region. 3. Extension of the exudate in such quantity as to cause a gross detachment of the retina. 4. Absorption of the greater quantity of the fluid during the weeks of rest. 5. Exposure of the initial area of damage in the macula. 6. Onset of scarring in the macular region.

It is probably fortunate that operation was decided against. Had operation been performed no benefit could have accrued to the patient, since subsequent events proved that the macula was practically destroyed. It is even possible that, had operation been performed, it might have been held to be responsible for the macular injury; in any case, there would have been no certainty that the eclipse was alone to blame for this.

There are good grounds for believing that the majority of detachments succeed some injury or tear of the retina. In this case there was no such injury, unless it can be held that the damage to the macula consequent on the exposure constitutes a point of permeable tissue. It is much more likely that the accumulation of fluid behind the retina was an inflammatory collection—a suggestion to some extent supported by the gradual onset of the cloudiness of the vitreous some time after the detachment reached its full measure.

A METHOD OF PREVENTING PUERPERAL INFECTION.*

By F. H. WHYTE, M.B.,

LATE EXTERN ASSISTANT, COOMBE HOSPITAL, DUBLIN.

In making a vaginal examination or putting the hand into the uterus abnormal ascent is substituted for the natural descent, with great risk of carrying up infection. The frequency with which these apparently trivial and necessary proceedings either kill, sterilize, or are the cause of chronic invalidism is one of

the worst features of obstetrics. Auto-genous infections are rare, and cases arising from germs lying in the lower genital tract being pushed up by previously sterile gloved fingers are not autogenous. That the vulva and vagina (particularly its lower part) are septic towards the end of pregnancy is an undisputed fact. As a rule, the lower down in the tract the more numerous, mixed, and virulent

FIG. 1.

FIG. 2.

FIG. 1.—Digital funnel. 1, A diamond-shaped piece of tough white rubber 9 inches long and 5 inches wide, to cover pubis, perineum, and sides of thighs. 2, Tube part of funnel. Diameter of tube $1\frac{1}{2}$ inches. Length of upper border 3 inches, lower border 3 inches, so that free end is cut like a Fergusson's speculum. 3, Canvas covering to strengthen angles of funnel. 4, Eye for threading tape. The manual funnel is the same except that the tube part has an oval section; its vertical diameter is $3\frac{1}{2}$ inches and its transverse is 2 inches, to admit the hand. The upper border of tube is 4 inches long and its lower is 5 inches. The width of its diamond-shaped portion is 7 inches.

FIG. 2.—Repositor for digital funnel. 1, Eye through which the tube is threaded. 2, Mark, $2\frac{1}{2}$ inches from eye. 3, Handle. The instrument is made from round steel about the size of No. 7 catheter. The larger repositing for manual funnel is similar, but the eye is bigger to receive the larger tube, and the mark is $3\frac{1}{2}$ inches from the eye.

do the germs become. Bacteria are common also in the urine, and the germs in the bladder may frequently cause no symptoms there, but if they reach the injured uterus their behaviour may be very different. The proximity of the

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