origin, with considerable haemorrhage between the membranes. The blood had forced its way between the frontal lobes, damaging considerably both inner frontal convolutions.

CASE II.

In another case, a woman aged 46, the diagnosis of uraemia had been made, because the doctor in attendance had treated her previously for high blood pressure and occasional albuminuria. She was eight months pregnant, and had been taken suddenly ill when returning home about 6.30 p.m., five hours before I saw her with Dr. Woodcock, of Palmer's Green. She was then quite comatose, with slight generalized rigidity, pin-point pupils, and extremely raucous stertorous breathing. The blood pressure was then 160, the knee-jerks brisk, and bilateral extensor plantar reflexes. Lumbar puncture revealed extensive haemorrhage into the cerebro-spinal fluid, which was intimately mixed and quite opaque with bright red blood. Death occurred five hours later, but the diagnosis made of pontine haemorrhage seems probably correct.

Meningeal haemorrhage resulting from fractured base, and lacerations of the brain due to concussion, will in like manner cause bloodstained cerebro-spinal fluid, the depth of stain varying in proportion to the amount of exuded blood. The staining may be no more than a distinct yellow tinge after the lapse of three or four days, as after the third day a pigment derivative from the blood, of the nature of bile pigment, appears in the cerebro-spinal fluid, due to absorption and chemical changes in a localized collection of blood in the membranes or brain substance. This yellow pigment, appearing after the third or fourth day of an intrameningeal haemorrhage, was shown many years ago by Froin to be practically identical with bile pigment, and gives the Gmelin reaction for bile pigment, but does not give the ordinary test for blood. Thus, a small localized cerebral haemorrhage in the neighbourhood of the internal capsule or other deep area within the brain substance, which has neither reached the surface nor ruptured into the lateral or third ventricle, will signalize its presence by the appearance of this yellow stain in the cerebro-spinal fluid within three days, rather than by the appearance of free blood mixing with the cerebro-spinal fluid, as is the case with the larger cerebro-spinal haemorrhages. In fractured base and laceration of the cortex the injury may be sufficient to cause death within a week, and yet the fluid may be stained only a light yellow, with never any free blood mixed with it.

Meningeal venous haemorrhage may occur from the rupture of a vein, usually into the posterior fossa, in periods of intense vascular congestion, as in men during the act of coitus, especially when performed in an abnormal position, such as when standing, and if the man is in poor health or suffering from headache at the time. Violent headache and faintness, with, very probably, vomiting, occur at once, and intense headache may persist for several days, though complete recovery is the rule after three or four weeks. Free blood in the cerebro-spinal fluid will be found by lumbar puncture during the first few days, the blood being probably recoverable within half an hour or so of a direct haemorrhage into the subarachnoid space. Venous haemorrhages may also occur upon the cortex during the severe congestion of an epileptic fit, or during the strain of violent coughing, as in whooping-cough, the blood either appearing free and intimately mixed with the cerebro-spinal fluid, or indicating its presence after the third day by the yellow tinge of bile pigment.

Spontaneous haematorrhachis, or intraspinal haemorrhage, may occur as the result of an intense muscular effort, and if the bleeding occurs low down in the spinal canal, amongst the roots of the cauda equina, the blood may clot *in situ*, and cause pain in the lower extremities, and motor and sensory paresis for years. Such a case I published in 1912 (*Proc. Roy. Soc. Med., Neurolog. Sect.*, February 15th, 1912, p. 115).

When the blood is extravasated higher up in the cerebro-spinal axis, then the mixture of blood and cerebro-spinal fluid obtained by lumbar puncture does not clot. Spontaneous haematorrhachis, with the formation of clot around one or more roots of the cauda equina, I believe, accounts for not a few cases of persistent pains in the lower extremities following a heavy muscular effort, such cases being usually diagnosed as ruptured muscle, fibrositis, or neuritis, and if the pain is in the front of the thigh the condition may be labelled "meralgia paraesthetica." Lumbar puncture would at once decide the diagnosis in such a case, possibly even one or two years after the onset of symptoms, as in the case quoted above, in which I obtained by lumbar puncture a canary-yellow fluid eighteen months after the primary attack. Another example may be given in which the lesion was almost certainly a spontaneous haematorrhachis.

CASE III.

A man, aged 61, strained his right leg in pushing a heavy iron safe with his knee. Two days later he noticed great tenderness along the front of the thigh, so that he could not bear the bed-clothes over it; the pain then spread to the calf and the sole of the foot, which has ever since for the last five years felt as if he were walking on sand or pebbles. The right foot is always cold and blue; there is no anaesthesia, but a varying degree of hyperaesthesia. The knee-jerks and Achilles jerks are unaffected.

Though lumbar puncture was never done in this case, which did not come under my observation until two and a half years after the onset of symptoms, I have no doubt that spontaneous haematorrhachis was the cause, and that lumbar puncture would have shown free blood in the cerebro-spinal fluid in the first few days, and that subsequently the fluid would have been yellow-stained, probably for months.

SIMPLE GOITRE.

BY

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It may be useful at a time when the prevalence of simple goitre in this country is attracting attention to outline our present knowledge with regard to its causation. I include in the term "simple goitre" those sporadic or endemic cases often encountered in towns and country districts in England. Simple goitre is a deficiency disease due to an insufficient supply of iodine for the needs of the thyroid gland, or more properly for the needs of the body cells in the form of the gland's iodine-containing hormone. Two facts may be stated in proof of this definition: (1) Thyroid hyperplasia is readily produced by the use of diets deficient in iodine; (2) iodine in minute doses will prevent and cure goitre when administered at the proper time and season. But while theoretically goitre can, and sometimes does, arise in consequence of a deficient supply of iodine in the food, it arises more commonly in spite of the fact that there is present in the food a sufficiency of iodine. There is abundant proof of this statement; it will suffice, however, to mention in this connexion the results of one fundamental experiment. If animals be confined in dirty cages they will develop goitre spontaneously, the size of the goitre being dependent on the season of the year and the duration of exposure to the unhygienic conditions. If, however, they be confined in scrupulously clean cages of the same dimensions goitre will not arise. Since the food supply, and therefore the iodine supply, was the same in both cases, it follows that there are factors provided by unhygienic conditions of life, as in dirty animal cages, which render the available iodine in the food insufficient for the needs of the body in these particular circumstances. A sufficiency may become an insufficiency in the presence of such unhygienic conditions. Further, if an additional supply of iodine be provided, or cod-liver oil be administered, goitre will not develop in animals confined in dirty cages. This action of unhygienic conditions of life calls to mind the effect of want of sunlight in favouring the development of rickets when animals are fed on a diet deficient in vitamin A, calcium and phosphorus. The parallel must not, however, be pressed too far, for the mode of operation of the two factors—dirt and want of sunlight—differs; the parallelism is mentioned to emphasize the importance of such general hygienic conditions of life as cleanly surroundings and sunlight in the maintenance of health, an importance which is apt to be minimized in our search for more specific excitants of disease.

This effect of iodine in preventing enlargement of the thyroid gland (goitre) is to some extent exhibited also by free chlorine, although I am not yet certain how far this halogen prevents thyroid hyperplasia. So far as my experiments go they agree in their results with those of Marine, who found that iodine prevents both thyroid hyperplasia and goitre; but while chlorine has in some recent experiments prevented thyroid enlargement, it has not always prevented thyroid hyperplasia without enlargement. Two of the facts proved regarding the production of simple goitre may therefore be stated as follows: (1) That it may arise from an insufficient supply of iodine; and (2) that under unhygienic conditions of life, as represented by dirty animal cages, an additional supply of iodine is—in certain cases especially—

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.

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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.