

the candidates' papers with the other eye. When he came to me at the end of the examination he had a severe iritis with several adhesions; when asked where was the source of sepsis he said he had a discharge from a molar tooth which had existed for more than two years and had followed an abscess at the root. This tooth was removed within 12 hours, the iritis subsided quickly under atropine and hot bathings, and on the ninth day the eye was white and the vision normal. A week ago he told me he had had no further trouble and it was now the better eye.

Case 5 illustrates the necessity of examining the mouth one's self or getting the opinion of someone who knows what pyorrhœa is, in view of the frequent absence of recognition of the disease.

CASE 5.—The patient, aged 38, was sent to me by Dr. J. F. Bullar of Southampton, who first saw him in January, 1911, with mutton-fat keratitis punctata and vision of 5/5 in each eye. As some teeth were in a bad state Dr. Bullar made him go at once to the dentist and have all out about which there seemed any doubt. In February, 1912, when I saw him he could count fingers at 2 ft. with the right eye, and at 1 ft. with the left eye. The vitreous was too hazy for the condition of the fundus to be seen. The tension was raised and the right field of vision contracted at one part up to the fixation point. I sent him to Mr. J. G. Turner, who condemned all the teeth on account of bad pyorrhœa. Soon after they had been removed the vision began to improve, and to-day the right is 5/24 and Sn. 1.5, and the left 5/6 and Sn. 0.5. The tension is normal and the vitreous steadily clearing.

Case 6 illustrates the rapid recovery from a long-standing and severe inflammation by removing the teeth and the recurrence of the inflammation when the gum was damaged by the spicule of bone.

CASE 6.—A gentleman, aged 52, who had been sent to Harrogate after six months' treatment for a badly inflamed eye, came to see me at Mr. E. S. Steward's suggestion. I agreed with Mr. Steward that the inflammation was due to sepsis from pyorrhœa and that the treatment of the mouth should be continued. After leaving Harrogate the patient was given an unfavourable opinion as to his chance of regaining any useful sight. He came to me the same day with a badly inflamed eye that could count fingers at 3 feet only. The sclerotic, cornea, iris, and ciliary body were inflamed and the vitreous hazy. His remaining teeth, which were all very foul and septic, were removed two at a time, a plan insisted upon by his dentist.

The inflammation began to subside as the teeth were removed, and two days after the last ones came out the eye was free from injection, and he went home assured by me that the inflammation would not return; but at the end of a fortnight the eye was again very red, and at the same time a spicule of bone came through the gum. The loose piece of bone was pulled out, and next day the eye was white again, and ten weeks after the first teeth were removed the vision was normal.

In the next case there was a chronic predisposing cause, and the fatigue of manœuvres was a sufficient exciting cause. It was an early case, and the recovery was most striking.

CASE 7.—A Yeomanry officer, who thought he was in good health, though he had not felt well after breakfast for about a month, whilst out in camp with his troop noticed two days before a brown stain on the newspaper; he had read the paper earlier that morning without noticing anything unusual. On covering one eye there was a blur, the size of a shilling, involving the fixation point. He consulted Mr. Frank G. Thomas, of Swansea, who found a small patch of recent central choroiditis, but could not discover a cause, and sent him to me for my opinion. I found the vision of the bad eye to be 5/6 slowly and Sn. 0.37, the good eye seeing 5/3.5 and Sn. 0.25. In the affected yellow spot there was a small patch of choroidal disturbance. Five days later he no longer saw the brown mist, the vision had improved to 5/5 slowly, and he was feeling better. In the interval his mouth had been cleaned by removing three septic roots and treating the pyorrhœa.

The following case also illustrates the rapid recovery after treating the cause.

CASE 8.—The patient was blind in one eye from secondary glaucoma after dysentery in India many years before. The vision with the correcting glasses was 5/4 and Sn. 0.25 in the other eye, which was quite healthy, on the two occasions 4½ years apart, when I ordered a change of glasses. At the third visit he complained of seeing a blur near the fixation point; the vision was reduced to letters of 5/5 and Sn. 0.5 slowly by a small patch of recent choroiditis, just below the macula. He was edentulous, but his plates were foul; nothing else was found amiss, no diarrhœa, no remains of dysentery, but his normal-looking and smelling urine contained staphylococci, streptococci, and bacillus coli. He was put on a course of autogenous vaccine; within two weeks his sight had improved to 5/6 partly, the blur went a few days later, and within seven weeks the vision was again 5/4 and Sn. 0.25, and where the patch of choroiditis had been there was some disturbance of the uveal pigment.

The next case illustrates the length of time an untreated cause may continue to produce recurring attacks.

CASE 9.—The patient, who was married, had from the age of 12 years had many attacks of tonsillitis every August; these recurred at varying intervals for 35 years. Her tonsils were treated, and since then the attacks have ceased. Her first attack of iritis occurred when she was aged 18, and was followed by many others up to the time when the tonsils were treated. In the interval she had almost lost her sight.

The last case shows the influence of a second septic focus which came into existence after the first had been removed.

CASE 10.—A lady sent by Dr. M. Tench, of Dunmow, had episcleritis, which had begun five months before, after getting some metal polish into the eye. The pyorrhœa, which she already had, was treated, and in two months the eye was well. In 1907 the episcleritis recurred, and after it had lasted a year all the teeth were removed. The next day the eye was whiter, the general health improved, and she said "life was worth living." Recovery was rapid. In 1909 she had appendicitis, and the episcleritis returned, but her doctor reports to-day that both diseases quieted down together, neither relapsed, and she is now feeling very well.

In all these cases the septic focus was removed or treated; and beyond that nothing, save local treatment, was done for the eye.

I am convinced that senile central choroiditis is no more caused by old age than is the senile edentulous jaw; both are due to pyorrhœa, and if the mouth were looked after from youth onwards, two of the attributes of old age, *sans teeth, sans eyes*, would not occur.

In conclusion, I regret I have had to travel beyond the strict limits of the matter set down for discussion, but I hope I have made it clear why the ophthalmic surgeon cannot limit his inquiries if he is to rid the patient of the inflammation that is affecting the eye. How dependent he is upon the physician, the general surgeon and his *confrères*, the other specialists, is obvious. As an ophthalmic surgeon I can look forward full of hope to a future when these serious eye affections will cease to occur, because the physician has taught mothers how to feed children properly and the children how to eat properly, so as to avoid intestinal stasis. The dental surgeon has impressed upon the population at large the importance of proper mastication and the hygiene of the mouth.

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THE RELATIONSHIP OF THE THYROID GLAND TO ALIMENTARY TOXÆMIA.¹

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THERE is little room in this discussion for dogma. At the best, as Professor Keith has said, it is but the child of our ignorance. The scientific basis on which it is founded is altogether too meagre to sustain the weight of superstructure which those of us who are taking part in it are building upon it. Yet it must be hoped that the correlation of the views of the different speakers will both lead to a better understanding of this far-reaching condition and promote investigation into the cause of alimentary toxæmia, a territory at present almost entirely unexplored. There is little which can be said that is more than merely speculative, and perhaps that part of the subject with which I am dealing—the relationship of the thyroid gland to alimentary toxæmia—is as intangible as any.

Evidence is accumulating to show that, among the many functions of the thyroid gland, one of the most important is a protective action against circulating toxins. Complete removal of the thyroid leads not only to myxœdema—a condition which may be cured partly or completely by the administration of its internal secretion—but also, as is well known, brings with it a considerable risk from a rapidly fatal toxæmia.

An important manifestation of this toxæmia is the symptom-complex known as tetany. On very good grounds tetany is thought by many to be produced by toxins generated in the alimentary canal. It occurs in gastric dilatation where stagnation is great, and disappears if the stomach can be washed out; moreover, in such cases a substance has been isolated from the gastric contents by Bouveret and Dévic which, when injected into animals, has reproduced the clinical picture of tetany. I have described a series of cases where the same syndrome has accompanied dilatation of the colon, when stagnation in the bowel was accompanied by grey pulsatious and offensive motions, tetany followed, and was only relieved by colonic lavation. As is well known tetany in this country is most common in rickety children in whom some degree of atony and dilatation of the stomach and intestines is common; and I think most observers will agree that the tetany is almost invariably preceded by an unhealthy condition of the stools. Tetany is sometimes treated with success by the administration of thyroid extract. Tetany, then, is a condition which, on the one hand, is produced by removal of the thyroid gland, and on the other is related to alimentary toxæmia. It seems, therefore, a reasonable inference that the thyroid gland, among its antitoxic functions, includes that of combating poisons absorbed from the intestinal tract.

¹ A paper read at a special meeting of Fellows of the Royal Society of Medicine on May 5th, 1913, being a contribution to the discussion on Alimentary Toxæmia.

In cases of excessive absorption of alimentary toxins we should expect some compensatory hyperplasia of the thyroid gland, and it is interesting that in the course of this discussion Mr. Arbuthnot Lane, Mr. George Rowell, and Mr. H. W. Carson all state that they have seen ileo-sigmoidostomy followed by shrinking of a goitre.

Another clinical link in the chain of evidence is afforded by rheumatoid—sometimes known as infective—arthritis. This disease is not uncommonly accompanied by enlargement of the thyroid gland, by Graves's disease, and by tetany, or a condition of the hands and feet closely resembling it. It is also regarded by many as an effect of oral sepsis or intestinal intoxication. An important support of this view is the work of Mr. Kenneth Goadby on the subject. Points which favour this contention are the almost invariable presence of oral sepsis, the beneficent effects, sometimes amounting to complete cure, which follow attention to the mouth; the improvement after lavage of the colon in some cases, and in a case recorded by Mr. Lane after ileo-sigmoidostomy.

In rheumatoid arthritis, then, we have a condition probably in many cases due to alimentary intoxication. In this condition overgrowth of the thyroid is apt to occur, and also tetany—a symptom of ineffective functioning of the thyroid gland—is found. Moreover, rheumatoid arthritis is occasionally distinctly benefited by the administration of thyroid extract.

Mr. Rupert Farrant has studied the effects of several types of clinical toxæmia upon the thyroid gland. The following changes occur: (1) the colloid becomes granular; (2) vacuolated and partially absorbed; (3) the cells become more numerous, elongated, and arranged in masses; (4) the colloid is entirely absorbed, and the walls of the vesicles become crenated and folded; and (5) the infolding and cell-increase go on to transform the vesicles into solid masses of cells. In other words, there is a reaction of the thyroid gland, with signs of hyperplasia and increased functioning, amounting in some cases to changes found in Graves's disease. This holds true for such diverse toxæmias as those of infantile diarrhoea, diphtheria, measles and broncho-pneumonia, and whooping-cough and broncho-pneumonia. A similar hyperplasia was caused in guinea-pigs by the injection of diphtheria toxin, but was mitigated if thyroid was given at the same time.

Equally conclusive inferences are to be drawn from the epoch-making researches embodied in the recent Milroy lectures of my friend, Major R. McCarrison, I.M.S., who has studied goitre as it occurs endemically in the valleys of Chitral and Gilgit in Northern India.² He has shown beyond dispute that thyroid enlargement can be produced experimentally in man, within a few weeks, by the administration of suspended matter separated by filtration from goitre-producing waters; and can be cured by taking thymol, one of the most powerful of gastro-intestinal antiseptics. He has shown that if the source of the toxin be avoided or removed the gland will return to normal size, unless secondary changes have occurred.

It is to be expected that, like other glands, the thyroid may undergo degenerative and retrogressive changes as the result of over-action. Normally at the menopause with the atrophy of the ovary part of its work ceases, and it is conceivable that if it has previously been subjected to undue activity its normal involution may overshoot the mark and lead to hypothyroidism and myxoedema. The last three cases of myxoedema which I have had under observation have all shown, together with severe oral sepsis and very offensive motions, anæmia of the pernicious form, which Dr. William Hunter has for so many years ascribed to lesions in the alimentary tract.

If the thyroid undergoes enlargement to combat undue toxæmia, it is reasonable to suppose that relieving it of part of its burden may cause a simple goitre to shrink, but this, of course, cannot be expected if adenomata or cysts are present. McCarrison has caused goitres to dwindle or disappear by using vaccines prepared from different forms of organisms—staphylococcus, a coliform bacillus, and a spore-bearing bacillus. The good results which he has obtained have led me to try a similar method of treatment, and I have followed out the plan which he adopted. The vaccines which I have generally employed have been prepared from the coliform

bacilli of the patient's own bowel. The bacterial growth has been obtained from the fæces by culturing on Musgrave's medium, which consists of beef extract 0.5 gramme, sodium chloride 0.5 gramme, agar 20 grammes, tap water 1000 c.c., and is of an alkalinity of minus 1. This gives a growth of coliform bacilli of fairly constant character. The initial dose has been usually 125,000,000 of the dead organisms, suspended in about 1 c.c. of saline. Subsequently, if the goitre has shown signs of diminishing I have increased the dose weekly by 25,000,000 or 30,000,000. As a rule the injections have been given at weekly intervals.

It is too early to make any definite statement as to the results, but in the few cases, eight in all, treated they have been very encouraging. In one case the goitre disappeared entirely. In all the others it has diminished. As a rule, after the first injection the tumour becomes softer and, after subsequent doses, it gradually gets smaller, although occasionally periods of quiescence supervene. Two of the patients have been able to do up their neck-bands for the first time for several months after the first or second injection. A man who had taken to larger-sized collars is now wearing those of his ordinary size. One girl who had definite stridor, even when lying in bed, has now no embarrassment of breathing except slightly after exceptional exercise. All are charitable enough to say they are better and free from the symptoms which caused them to come for treatment, such as a feeling of suffocation and difficulty in swallowing and breathing.

Another effect has been that, by producing absorption of the parenchymatous enlargement, adenomata previously undetected have become obvious, and operative treatment has in this way been facilitated.

To sum up, the thyroid is a gland a useful function of which is to counteract intoxication. One of the sources of this intoxication is probably the bowel. If the intoxication be proportionately great for the gland, it undergoes hyperplasia and visible enlargement. If the amount of intoxication can be lessened the extra burden is removed and the gland diminishes. This has been done by the use of thymol and of vaccines, especially those prepared from cultures of the coliform organisms obtained from the patient's own intestinal flora.

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INDICATIONS OF NERVE LESION IN CERTAIN PATHOLOGICAL CONDITIONS OF BLOOD-VESSELS.

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SOME time ago it became apparent that the vascular symptoms so frequently associated with the sensori-motor syndrome of the affection often laconically designated "cervical rib" were probably produced by a lesion of the nerves supplying the blood-vessels of the arm. It was then obvious that the same explanation might afford a clue to the elucidation of many other obscure conditions. It was therefore my intention to investigate those other diseases to discover, if possible, whether any such relation existed in them between nerves and blood-vessels as has been shown to be present in cervical rib. Being aware of the difficulty of the problem and the many pitfalls which it presents I have been unwilling to write anything further on the subject until I possessed more extensive data than it is possible to submit at present. The appearance, however, of two recent papers bearing on the subject of blood-vessel changes consequent on nervous lesions would seem to make it advisable to review the work of recent investigators as a preliminary to further research.

Of the two papers mentioned above the first is one by Manouélian¹ showing that damage to nerves supplying the abdominal aorta and pulmonary artery results in localised areas of arterio-sclerosis typified by degeneration of elastic fibres and smooth muscle fibres with hyperplasia of the connective tissue of the media, together with intimal proliferation. The same results have been obtained in the case of the vessels to the ear after section of the cervical sympathetic by numerous workers, among whom may be

² THE LANCET, Jan. 18th and 25th, and Feb. 8th, 1913.