with the fields before operation, show a considerable improvement in the patient's eyesight, especially in the right eye. There is a small perforation in the nasal septum, and there are a few adhesions between the septum and the lateral nasal wall. The anterior walls of the sphenoidal sinuses are easily inspected. There is no evidence of sepsis in the nose.

The patient was first seen in April, 1927, examined thoroughly and treated by surgical operation in June, and reported on after examination on October 4th.

NOTES BY DR. R. C. J. MEYER.

Objective examination showed no abnormality as regards the outward appearances, motility, and pupils; the Wernicke pupil reaction was demonstrated later. The fundus was healthy, but there was slight hypermetropic astigmatism; the subsequent wearing of glasses stopped all headaches. Right vision = 6/6, J.1. Left vision = hand reflex centrally, but fingers at 3 metres nasally. This called for examination of fields, the broad results of which are indicated in the diagrams. Fig. 3, A (right eye),

Fig. 3.

shows the right upper outer quadrant lost; the patient was quite unconscious of right quadrant loss of field until examination demonstrated it to him. In Fig. 3, B (left eye), the temporal hemianopia is seen to cross the vertical middle by about 15 degrees. The patient was quite conscious of loss of vision of left eye, which drove him to seek medical aid.

The examination suggested the presence of a pituitary tumour involving the left side much more than the right, but later on under portion of chiasma. I handed the case over to the physician, radiologist, and surgeon, but the patient could only come for observation (May 28th).

Two operations were performed—one on June 13th and the other on June 27th. On July 13th, at the request of the surgeon, fields were again taken (there being no change in any other condition of the eyes). The right upper quadrant scotoma had now diminished to a smaller sector. (See Fig. 4, A.) The left scotoma had also diminished. (See Fig. 4, B.)

Fig. 4.

On September 1st a friend stated that the patient was "quite better," which meant, at any rate, a decided improvement. (N.B.—Patient lives in the country and over a hundred miles away.)

Fig. 5.

On October 4th the patient unexpectedly arrived for examination. This showed the right scotoma to be still further diminished. The left scotoma was also diminished somewhat. Right vision = 6/5, J.1. Patient does not feel the scotoma, and this does not exist for luminous objects at all now. Certainly a most striking improvement. (See Fig. 5, A.) The left disc shows great pallor of nasal side—that is, atrophy is showing actually at the disc. (See Fig. 5, B.)

THE AFFINITIES BETWEEN RHEUMATISM AND TUBERCULOSIS.

BY

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TUBERCULOSIS connotes the morbid changes caused by living tubercle bacilli in the living tissues of man and animals. As the tissues react they present lesions of endless variety in degree, kind, and time. The outstanding and characteristic lesion is the tubercle with its epithelioid and giant cells, but there are also lesions as acute as those of septicaemia or pyaemia, and, on the other hand, as chronic as those of chronic rheumatism. Indeed, it seems likely that many so-called "rheumatic" affections, which do not yield to antirheumatic treatment, are really manifestations of chronic tuberculosis. Even joint affections accompanied by pain and swelling may be tuberculous in nature.

The brilliant investigations of Liebermeister, with the help of his new acetic acid-antiformin method, seem to establish the frequency of chronic inflammation free from any tubercles as a distinct and even independent lesion of tuberculosis. These chronic inflammatory lesions are, indeed, often the end-results both of acute lesions and of true histological tubercle, but Liebermeister holds that they may be independent of either of these lesions and are common in many organs and parts. They have been overlooked because death does not occur in this chronic stage, and there is no chance of seeing them. Such lesions may be the *fons et origo* of the multifarious disturbances of function occurring in the early stages of chronic tuberculosis. The lesions are the most chronic that occur in tuberculosis, and cause mainly mild and transient symptoms. Thus it may be impossible after a long lapse of time to trace them to their true cause or fix the time of their occurrence. Living tubercle bacilli may be found in these chronic lesions, and evidence supports the view that these quiescent lesions may be reactivated. These lesions are most common in the secondary stage of chronic tuberculosis, when tubercle bacilli, in small numbers, more or less isolated, and certainly not in clumps or masses, are circulating in the blood, before tertiary disease occurs in the apex of the lung. Amongst other manifestations, tuberculosis may cause lesions suggesting rheumatism of joints with painful swellings, in some cases with erythema nodosum, in others with erythematata, pains also in muscles and in one or more joints, pains in the chest without signs of pleurisy, pains in the neck and in the eyes or head, disturbance of the heart's rate and rhythm—even pains in nerves (sciatica). Liebermeister has demonstrated the presence of these chronic inflammatory lesions notably in lymphatic glands, serous membranes, even in the meninges, in the lungs, liver, kidney, spleen, veins, skin, eyes, and larynx. May not these lesions be responsible for the various symptoms that may and do occur and pass away in the early stages of chronic tuberculosis, and especially of chronic apical tuberculosis?

For the most part these lesions elude us, and we can only bring them to light by means of tuberculin. Twenty years ago I suggested the possible existence of these lesions because doses of tuberculin excited pains in various organs and parts in a manner that seemed to indicate a focal reaction. For many years I have observed pains in special muscles and joints, time after time in the same part after each dose—in the hip, or knee, or little finger, or thumb, or heel; in the back or shoulder; in bones or muscles, such as severe cramps in the calves of the legs; pains also in the epigastrium, doubling up the patient after each dose, and even boring or lancinating or cutting pains in the shoulder radiating down the arm, or deep pain in the chest. There are strange individual idiosyncrasies, because in the same patient, after injections of tuberculin, the same parts are apt to suffer, though the site of injection varies. These pains, in fact, may be related to chronic inflammatory lesions and may be really focal reactions. One has great opportunities of studying these pains, their varieties and situations, after injections of tuberculin. Naturally there is no other way of investigating them.

Moreover, as a rule these chronic inflammatory lesions can only be investigated with the help of subcutaneous injections of tuberculin. But it does happen, fortunately, that these chronic inflammatory lesions of the early stage of tuberculosis may rarely disclose themselves beyond all doubt or cavil when they occur in structures visible to the naked eye, notably in the skin and structures of the eye and even in the larynx. The so-called pre-tuberculous laryngitis may be of this nature.

I know that my old teacher Radcliffe Crocker would not admit that there was any connexion between erythema nodosum and tuberculosis, while S. Mackenzie stated that erythema nodosum is frequently, if not generally, an expression of rheumatism. I may be skating on thin ice if I say that in my limited experience I have not once seen erythema nodosum apart from tuberculosis. All cases have reacted severely to tuberculin, and no case has had any relation to rheumatism. However, identity of lesion does not signify identity of cause. Histologically the lesions of erythema nodosum cannot be distinguished from rheumatic lesions of the skin, but Liebermeister has shown that such lesions may be caused by tubercle bacilli. At present I am bound to believe that erythema nodosum is a tuberculous lesion. Without tuberculin I could not have held any but the general opinion. I first pointed out the relation of erythema nodosum to tuberculosis in 1902, and since then all my evidence strengthens this view.

But, because its lesions are visible and cause unpleasant symptoms, the eye provides the best field for examining the remarkable affinities between chronic forms of tuberculosis and the so-called rheumatic affections which are attributed to chills or other indeterminate causes. For the last thirty years ophthalmologists have been at issue upon the "rheumatic" affections of the eye. The usual lesions in question are conjunctivitis, iritis, episcleritis, and scleritis.

In his great textbook (1926) Sir John Parsons writes that "rheumatism and gout are the chief causes of episcleritis and scleritis"; "most commonly there has been marked rheumatism." Later he says, "When tubercle is suspected injections of tuberculin may be employed." I say that tuberculin is the only means of settling the diagnosis. Unfortunately I have seen only three cases of episcleritis, because this uncommon disease does not come to a tuberculin dispensary, as tuberculosis is not suspected. In these cases tuberculin has not only established the diagnosis, but, as the cardinal remedy, has rapidly brought about extraordinary improvement even in the course of a few weeks. All the pain, tenderness, and discomfort have abated, and photophobia has vanished. One of these cases had been diagnosed several years ago as "rheumatism of the head," and the two worst cases had been under treatment for years without any real improvement. Since tuberculin treatment has been adopted the eyes have assumed a normal appearance and the patients have no symptoms. But prolonged treatment will be necessary before it can be stated with any confidence that there will not be recurrence of the trouble.

Iritis is a common lesion in syphilis, and may arise from septic infection—sometimes secondary to carious and foul teeth—but tuberculosis may also cause iritis. Infection directly from the air is conceivable, but evidence points rather to infection by the blood current. Since removal of the eye has been advocated, it is very important to distinguish between a direct infection from the outside, when a lesion might be acute and progressive, and haematogenous infection, when the lesion would be chronic and circumscribed.

Again, early diagnosis requires the use of tuberculin, and since the lesion is generally chronic, tuberculin also offers the best hope of arresting the disease and preventing its recurrence. Sir John Parsons says that tuberculin "often fails to ameliorate the condition." I cannot presume to criticize such an authority without evidence, as I have not had an opportunity of treating a case of iritis, but reasoning from experiences in other cases I am inclined to think that failure such as this great authority has observed may be due not to the tuberculin, but to the administrator. Tuberculin treatment must be carried out by an expert with his own hands, and it must not be used merely to check

the disease temporarily, but to such a degree and in such doses that recurrence of the disease is prevented.

My chief experience has been with the common tuberculous lesion of the eye—"chronic conjunctivitis"—either as superficial ulcers or small granulomata and translucent cockscomb-like projecting masses. These conditions are often tuberculous; they occur in a tuberculous family; they all react to tuberculin, and, in my limited experience, always clear up under tuberculin in large doses—not less than 0.8 c.cm. or 1 c.cm. or more. But again success depends upon the skill of the operator, just as much as in the surgical treatment of glaucoma or cataract. Accordingly I am bound to hold the view that these eye lesions, like other lesions, belong to the secondary stage of tuberculosis and run a favourable course under tuberculin treatment. It is not enough to say "tuberculin may be used," because there is no other remedy that can consistently do so much real good.

These atypical lesions of tuberculous origin have a special interest. They have been called "rheumatic" because they do not conform to the histological or clinical picture of tuberculosis. There are no giant cells and no signs of caseation. They are mild, transient, and tend to recur. Both in the skin and eye they may pass away and never return, but without specific treatment, and even with it, recurrence at long or short intervals of time is common. They may be accompanied by swellings in joints and pains, and therefore may be designated "rheumatic." They are like the secondaries of syphilis which occur in the secondary stage and tend to heal. They bear this outstanding feature—that they occur only in mild forms of tuberculosis when slowly progressive or even occult lesions exist in the bronchial lymphatic structures. Thus they may be the obvious pacemakers for the common chronic disease—chronic phthisis—which subsequently fixes upon the apex of the lung. Tuberculosis may not be the exclusive cause of these chronic inflammatory states, but certainly the symptom-complex hitherto associated with "rheumatism" may be the effect of tuberculosis.

Thus certain diseases of the eye and skin, hitherto called "rheumatic," are really chronic forms of tuberculosis which, like scrofula, lack entirely the character of a typical histological tubercle. Ophthalmologists have brilliantly illuminated this interesting chapter upon the affinities of rheumatism and tuberculosis; little wonder, seeing that these lesions in the eye in the secondary state of tuberculosis are essentially vascular and can be seen directly by the eye. Thus, after many years of controversy, ophthalmologists have furnished clinical, anatomical, and experimental proof that, at any rate in the eye, atypical lesions of a chronic inflammatory nature, without either giant cells or caseous changes, may be truly tuberculous.

If, then, these atypical chronic inflammations are caused in the eye by tubercle bacilli, and can be seen, why may not similar mild tuberculous lesions occur in other vascular structures that are out of sight, and cause those disturbances of function which express themselves in an endless array of vague, various, and vagrant symptoms in so many organs and parts of the body during the secondary stage of tuberculosis? Thus the hitherto multifarious functional disturbances of the early stage of tuberculosis may possibly be traced to an anatomical lesion of delicate vessels caused by tubercle bacilli—few in numbers and perhaps attenuated in virulence. Like the lesions of scrofula, of episcleritis, of chronic conjunctivitis, and even of erythema nodosum, there may be vascular tuberculides, mild and even transient, in many organs and regions of the body.

These widely scattered tuberculides do not obtrude themselves, and may escape detection. The more one uses tuberculin for purposes of diagnosis, the more one may become impressed with the idea that lesions may be responsible for these erratic symptoms of early and mild tuberculosis. When these mild and obscure symptoms occur in the early stage of tuberculosis, the use of tuberculin in diagnosis is safe and certain and has no rival. Such obscure symptoms and aberrant lesions may be easily overlooked or misinterpreted. But any serious student of tuberculosis, trained in the use of these scientific methods, may find tuberculosis in strange places and in unfamiliar forms. Special knowledge may enable him to "mark

down," accurately and precisely, these masked forms of tuberculosis in the eye, in the nose, in the larynx, and even in internal organs and systems, at a time when diagnosis is otherwise extremely difficult and treatment relatively easy. Certainly in such cases the effects of tuberculin used as a remedy by an expert are often magical in suddenness and completeness.

General hospitals, and some special hospitals, might help earnest students and suffering humanity by establishing a special ward and an out-patient department for the study of chronic tuberculosis in both its common and erratic forms.

ENDOCRINE IMBALANCE AND ITS RELATION TO CHRONIC ARTHRITIS.*

BY

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THE views and opinions of the ancient authorities before the days of cellular pathology and bacteriology are of distinct interest in the light of our present knowledge. Hippocrates laid down the axiom that "Gout never occurred in women until the menses had ceased." Trousseau¹ and Charcot² believed that chronic articular rheumatism was associated with the beginning and end of menstruation and with pregnancy. Orde,³ in 1879, suggested that there was evidence to show that there was some relationship between uterine disorders and rheumatoid arthritis—such as severe arthritis with dysmenorrhoea and arthritic paroxysms coincident with menstruation—views with which I am in full accord.

At all these stages in a woman's life considerable strain is being thrown on the endocrine glands. These glands begin to assert themselves in early foetal life, and as they have no function in respect of external relation, their secretions must be entirely occupied with internal regulation, so that from the first any endocrine defect must disturb the balance. In childhood the endocrine system retards maturity to give time for further development, and utilizes glands such as the thyroid, subsequently katabolic in action, in the service of active growth. As puberty advances, the retarding gland—the thymus—atrophies, and the katabolic glands, like the thyroid, take on their own action. During pregnancy all the endocrine glands are working at full capacity, though some are antagonistic to each other. This continues during lactation, whilst at the climacteric the ovarian hormone disappears and more work is thrown on the thyroid, which itself is on the down grade.

There are two groups of endocrine glands:

1. Parasympathetic or extended vagus—anabolic in action—the cell-islets in the pancreas, and the parathyroids.

2. The sympathetic—katabolic in action—adrenals, thyroid, and pituitary.

The rhythm of life largely depends on the fluctuating balance between these two.

All the ductless glands, especially those co-operating with the sympathetic nervous system, participate in the development of sexual characters, the thyroid and possibly the post-pituitary tipping the balance towards femininity, the adrenal cortex and the anterior pituitary towards virility. Thus the thyroid shows increased activity at puberty, the catamenia, marriage, and pregnancy, and undergoes partial involution at the climacteric.

There is a close association between the gonads and the pituitary: neither appears to be able to get on and develop without the help of the other. The internal secretion of the ovary stimulates the secretion of pituitrin, and the development of a corpus luteum in pregnancy checks this process on behalf of the foetus until the time for the uterus to contract and for the milk to flow.

Llewellyn⁴ first drew attention to the association of the endocrines and chronic rheumatism, especially of the chronic villus type occurring in women at the menopause, expressing views which have been corroborated by Ribierre,⁵ Rosen,⁶ H. K. Thompson,⁷ Cecil and Archer,⁸

and many others. At a discussion on climacteric arthritis⁹ the subject was considered under the wider term of "hypoglandular arthritis." It was pointed out that this condition of joints was by no means confined to women at the climacteric, but that it was seen in young women due to thyro-pituitary failure. Cumberbatch concluded that there is a type of arthritis closely associated with, if not entirely due to, disturbance of the endocrine function of the ovary, and gave the following reasons for these conclusions.

1. Some of the cases occurred in young women in whom the menstrual function was being established. They developed arthritis of the rheumatoid type and amenorrhoea occurred. There was no evident primary focus of infection. The application of diathermy to the interior of the pelvis brought the arthritis to an end and menstruation returned.

2. Other cases occurred in women at the other end of menstrual life. In many the climacteric commenced at an early date and menstruation ceased. In these, again, there was no evident primary focus of infection. The application of diathermy usually arrested the arthritis and menstruation recommenced, in the cases in which the onset of the climacteric was premature.

Robinson considered that arthritis is directly or indirectly due to inactivity of the corpora lutea of the ovary. This inactivity may occur at any time after puberty, and may or may not be associated with absence of menstruation. Heating the ovary by diathermy increases the activity of the ovarian function.

Returning to the view of Trousseau and Charcot that chronic arthritis is a disease associated with the commencement and cessation of menstruation and also with pregnancy, I have seen quite a number of young girls in their teens in whom the start of menstruation had been delayed, or who had had one or two periods and then stopped. They were large, fat, well-developed girls, who complained of pains in joints and muscles.

One girl, aged 12½, had for six months complained of pain in her right knee; she was listless, had never menstruated, and had all the appearances of thyro-pituitary deficiency. X rays showed a small rarefied area just beneath the articular face of the cartilage of the tibia. Her tonsils were found to be septic, and removal was advised; this was carried out, and thyroid in small doses was also given. This case did not begin to improve until six months after the enucleation, when she started to menstruate, pain ceased, and she resumed her previous activity. Her mother suffered from chronic villus arthritis of the menopause.

In another case the patient's mother suffered from Raynaud's disease, which was not of a severe type. This girl was very much like the previous case in appearance; she was over-developed, was affected by amenorrhoea, and the rheumatic condition was more in the nature of a fibrositis of the erector muscles of the spine, though there was some doubt as to the condition of the dorsal vertebrae as seen with x rays. When I first saw this patient she had had her tonsils enucleated about fourteen days previously and was in a markedly hyperthyroidic state, with rapid pulse, muscular tremor, and thyroid gland slightly enlarged.

Crammer¹⁰ remarks that "it is well known that acute infections are sometimes followed by symptoms which may not unreasonably be attributed to disturbances of the thyroid-adrenal function. The stimulation produced by these toxins may develop into a Graves's disease." The thyroid is one of the defensive agents against infection, and I believe in this case the removal of the septic tonsils produced a flooding with thyroxine, the patient having most probably been hypothyroidic before.

Langdon Brown¹¹ points out how frequently tonsillar sepsis may be a factor in producing hypothyroidism in childhood. We are familiar with the fact that tonsillar sepsis is likely to excite hyperthyroidism after puberty, but until the gland has experienced the activating effect of puberty—that is, before the gland has taken on its katabolic duties—it is apparently more easily exhausted than excited to over-activity. Once the thyroid has taken on its katabolic duties we find patients with all the symptoms of rheumatoid or atrophic arthritis—as, for instance, the young woman in the early twenties.

Llewellyn had frequently pointed out that the early symptoms of rheumatoid disease, prior to the development of joint swellings, are very similar to the secondary features of Graves's disease and also Raynaud's disease, and he believes that all these diseases are due to endocrine instability or imbalance. McCarrison had pointed out that in the goitrous regions of the Himalayas rheumatoid arthritis is very common.

* Abstract of a paper read before the Harrogate Medical Society, February 17th, 1928.