An Address ON THE EVOLUTION OF TOXAEMIC IRITIS.

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By W. M. BEAUMONT,

FORMERLY SURGEON TO THE BATH EYE INFIRMARY.

A SHORT time ago I read a paper on iritis at the Royal Society of Medicine, and it is with some hesitation that I return to the subject.¹

The origin of inflammation, not only of the eye, but also of all parts of the body, is engaging the attention of workers in the field of biology, with the result that the science and art of medicine and surgery are profoundly influenced by modern conceptions.

As practitioners of a speciality which is as much medical as it is surgical, we are daily confronted by problems which enable us to test the value of the new pathology, the acceptance of which fundamentally affects our treatment. We find that an alveolar pocket or a duodenal ulcer may be a central bureau for the distribution of lethal diseases to all parts of the human economy.

The haematogenous theory has led to the etiological dethronement of iritis from the position of an independent disease to the secondary one of a complication. More than ever we did in the past do we now not alone direct our treatment to the local manifestation, but we search the furthest limits and probe the most hidden depths for the *causa causans*.

In the remarks which I have to make I shall confine my attention almost entirely to manifestations of toxæmia as seen in the iris and ciliary body. Not that I minimize the importance of infection of other parts of the eye, but because what I have to say with regard to the iris applies *mutatis mutandis* to the choroid and other structures.

The stress of the poison usually does not spread from the iris to the choroid. This localization conforms with the usual rule in disease of these parts: the immunity of the choroid being due to the fact that although the venae vorticosae collect the blood from the ciliary body and iris there is no exudation of morbid products in the transit through the choroid.

In reviewing iritis, no matter how it may be caused, we cannot fail to be struck by the frequency with which it is associated with arthritis, or at least with arthritic pains. It is noteworthy, too, that the arthritic and iritic symptoms are often so accurately coadjusted that exacerbations in the eye and joint frequently recur synchronously.^{2,3} On the other hand, practically never is acute rheumatism coincident with iritis. It is this anomaly which explains, perhaps justifies, the position of doubt taken up by some ophthalmic surgeons for many years, on the question whether iritis is ever rheumatic; a position which is enormously strengthened by the modern theory that acute rheumatism is a disease clinically distinct from what until recently has been called chronic rheumatism.

In acute rheumatism it would seem that the toxins do not find in the iris a suitable nidus.

When doubts about the etiology of iritis were first broached it was shown that many cases of so-called rheumatic iritis occurred in the subjects of gonorrhoeal infection. There was, however, a residuum of cases in which there was no suspicion of gonorrhoea. The next step was the suggestion that none of these residual cases were rheumatic, but that they were initiated by some blood-borne poison from a distant focus, in a manner possibly analogous to the iritis of syphilis and gonorrhoea.

Idiopathic iritis, a diagnosis which I think surgeons were always chary of making, also became suspect. It is a term which implies that the case is not syphilitic, gonorrhoeal, tuberculous, or rheumatic. But it is a name which suggests that the iritis has arisen spontaneously, that is, in the absence of any pre-existing disease. Does this ever occur? Is not iritis always a symptom rather than a disease *sui generis*? If so, then iritis from a toxæmic condition becomes extremely probable. An exhaustive

account of the pathogenesis of toxæmia in relation to irido-cyclitis was given by de Schweinitz at the seventeenth International Congress of Medicine last year.⁴

The suggestion that iritis might be due to such a toxæmic infection is, I think, coeval with the doubts regarding rheumatism as a frequent cause of iritis. It was somewhere about the end of the nineteenth century that these views were first expressed, but the repudiation of rheumatism required some audacity, for that disease enabled the physician to give a name to all obscure aches and pains situated in any part of the body. To doubt that such a convenient diathesis caused iritis was then tantamount to a recantation of one's professional creed. But now pathologists and physicians have followed the example of the doubter, and chronic rheumatism has no friends. So rapidly is the bacteriologist supplying the clinician with food for thought that the most omnivorous finds it difficult if not impossible to assimilate the gargantuan feast.

In a discussion initiated by the late John Griffith, at a meeting of the Ophthalmological Society, in 1900, he pointed out how frequently iritis was called rheumatic when in reality it was gonorrhoeal.⁵ Gonorrhoeal iritis had of course been recognized since the days of Brodie (1818), but the cases so designated were usually well-marked examples in which gonorrhoea was more or less acutely present. But the prolonged vitality of the gonococcus, and its far-reaching constitutional effects, were not realized. The surmises of Griffith regarding the dormancy of the gonococcus were based upon clinical observation and induction, and they have since been amply supported bacteriologically by Schnitzler, who has shown that pyogenic cocci may remain latent in the human body for thirty-five years, and then become virulent "on some chance disturbance of the normal processes of metabolism."⁶ By this discussion at the Ophthalmological Society attention was focussed on the intimate etiology of rheumatic iritis, and as a sequence the status of the term "rheumatic," as applied to iritis, was questioned, for it was found to be too vague, too ill-defined, and too inclusive. Such was the swing of the pendulum, however, that some of us went so far as to doubt whether rheumatism ever caused iritis. For this view support was found in the researches of Bouchard, the founder of the modern teaching regarding rheumatism. In 1881 he claimed for acute rheumatism an etiology essentially distinct from that of the heterogeneous mass of infective arthritides. But although his views received scant attention at the time, it was an epoch when bacteriology was being actively cultivated and the theory of the toxic origin of disease was being evolved. The result of Bouchard's work could not long be overlooked. In 1900 William Hunter⁶ drew attention to the importance of pyorrhoea and other types of oral sepsis as sources of systemic infection. This expression, "oral sepsis" was, I believe, first used in the sense in which we now use it in that year. In close sequence we find Arbuthnot Lane canvassing the possibility of toxæmia as a factor of unbridled energy in the causation of abdominal disease, and later he was demonstrating the faith that was in him by operating for intestinal stasis at Guy's Hospital. Coincidentally, Lang was advocating at the Middlesex Hospital and at Moorfields the desirability of treating pyorrhoea when it existed, in cases of irido-cyclitis, choroiditis, scleritis, and other diseases of the eye. In 1902 R. J. Llewellyn maintained that rheumatoid arthritis was the outcome of a gastro-intestinal toxæmia, and others have reiterated this view. To-day throughout the whole domain of medicine and surgery, at home and abroad, toxic states of the blood plasma are credited with an all-pervading influence. Whether the pendulum has swung too far time alone will show.

Opinion took on a new phase when chronic rheumatism was allotted, under the term "fibrositis," an anatomical basis, and thus sharply distinguished from acute articular rheumatism. If chronic rheumatism, with which iritis had hitherto been closely connected, was a totally distinct disease from acute rheumatism, with which iritis was not associated, the position of rheumatic iritis became questionable. Those who had anticipated the new pathology with their doubts about rheumatic iritis were supported in their scepticism. The relationship of fibrositis to iritis, and whether the association exists in all manifestation of the forms, call for immediate investigation. What is fibrositis?

Luff is of opinion that it is toxic in origin, and that is, I take it, the important point for us. The experiments of Rosenow of Chicago¹⁴ respecting interstitial myositis in rabbits will probably throw light upon the subject, but they are not yet complete, and we must wait for further knowledge. Until the exact relationship of rheumatism, fibrositis, myalgia, and all forms of muscular and articular aches and pains are more precisely codified we cannot, I think, dogmatize as to the connexion of iritis with them. Theoretically, as there is fibrous tissue in the iris, there should be fibrositic iritis. Is not every iritis a fibrositis? Rheumatism was a convenient term because it predicated nothing, and its flag waved over a heterogeneous battalion of diseases. That flag now lies trampled in the dust.

Although we have isolated instances in which iritis has been produced experimentally by inoculation of the *Diplococcus rheumaticus* from the cardiac lesions of malignant endocarditis, I am not aware of any indisputable clinical proof that iritis occurs as a result of acute rheumatism. If, then, we still postulate a rheumatic iritis it is associated with the chronic form of rheumatism, as opposed to the acute articular; the former is now accepted as distinct from the latter, inasmuch as the typical anatomical lesions are admittedly capable of production by a variety of organisms, including various strains of streptococci of attenuated virulence. The deduction is obvious that iritis occurring in association with the clinical features of chronic rheumatism should, like the other phenomena, not be classed as rheumatic, but referred to the particular organism responsible.

Present views regarding iritis make it necessary for us as practical surgeons to get behind the veil and to track back the infection to its birthplace. The almost invariable success of our search shows that there are very few cases of iritis not intimately connected with an infection or sub-infection. In the dyscrasia of syphilis and in that of gonorrhoea a source of infection is clear and unmistakable. In traumatic iritis, due to a perforating wound, germs obtain direct access to the iris, and even in operating under the strictest asepsis the surgeon's path of incision may become the road of sepsis.

Possibly, too, in sympathetic ophthalmitis toxins are elaborated in the exciting eye, which have a specific affinity for the iris and other structures of the sympathizing eye.

There is an ill-defined type of iritis arising from a dislocated lens in which there is apparently no path of entry for organisms from without. It is noteworthy that the iritis in such cases shows few signs of inflammation, although there are discoloration of the iris and posterior synechiae. Such a low form of infection is probably caused by organisms of attenuated virulence.

In diabetes the marked tendency to the dermic formation of boils and carbuncles of obviously infective nature may possibly explain the source of iritis, but, on the other hand, they may be the coeffects, with the iritis, of a common infection.

Iritis, although credited to malaria, is a rare complication in paludal disease; while that which sometimes follows late in cases of herpes of the fifth nerve would seem to be undoubtedly of infective origin. I have seen iritis in association with adiposis dolorosa, but as the patient suffered at the same time from dental sepsis it was probable that the latter was a factor common to both, inasmuch as adiposis dolorosa is due to an inflammation of the fibrous tissue of the panniculus adiposus.

Except in sympathetic ophthalmia the iris does not appear to be readily accessible to microbic invasion following injuries of distant parts of the body.

There is a side issue with regard to gonorrhoea which I think is of some importance—namely, that not only iritis but also conjunctivitis may be produced endogenously by that disease.^{7,8} In these cases of conjunctivitis there is usually evidence of systemic infection, such as metastasis either in joints, tendons, endocardium or in other structures. In the purulent discharge from the eye, however, if we accept the authority of Elschnig, there is absence of the bacteriological certainty of its identity, for although gonorrhoeal it is not gonococcal. The fact that these cases lack the unanswerable proof of gonorrhoeal origin—that is, the presence of the gonococcus—explains their being overlooked. In ophthalmia neonatorum the gonococcus is absent in a certain percentage of cases, and in such it may

occasionally happen that the disease has been conveyed endogenously. For although the fetal vascular circulation is not actually continuous with the maternal, yet so intimate is their coaptation that practically they may be considered indivisible, and there is nothing to prevent the gonorrhoeal poison from being carried to the eyes of the embryo transplacentally. Ophthalmia being produced endogenously by gonorrhoea it is probable that other specific infections may similarly initiate conjunctivitis. Garrod has pointed out that dysentery is a cause of muscular rheumatism, and also he says of conjunctivitis, an association which supplies food for thought.⁹ Passet and Longard have shown that in inoculated mice staphylococci pass into the conjunctival secretion.¹⁵ In mucopurulent ophthalmia, in which both eyes may be affected, we are apt to assume that the patients have been infected by microbic dust or air-borne germs. Possibly some of these cases are not exogenous.

The diagnosis of toxæmic iritis is obviously surrounded by difficulties, and in pursuing the germ we cannot confine our quest within the borders of our speciality; we have to become general physicians in the fullest interpretation of the term, and wander far afield to seek a focus of infection in oral, naso-pharyngeal, auditory, gastro-intestinal, or genito-urinary regions. We explore the digestive tract from its alveolar gates, through its devious windings, to its nethermost terminals for some breach in the armour.

Much may be learnt from the nature of the joint pains and swellings which are so often present. For, although these symptoms are common to tubercle, syphilis, gonorrhoea, fibrositis, and rheumatoid arthritis, there are considerable differences of material assistance in the diagnosis. The "rheumatism" associated with toxæmic iritis is frequently situated in the big joints of the lower limbs usually of mono- or of oligo-articular site, and in the plantar fasciae, but affection of the latter is rarely so severe as in the worst cases of gonorrhoea, or so prolonged as in tubercle. The iritis which sometimes occurs in the early stage of rheumatoid arthritis may often be diagnosed by the spindle-shaped finger-joints. When writing on this subject in 1908¹⁰ I was unable to hazard an opinion whether the iritis was caused by the rheumatoid arthritis or whether both were independently the offspring of a common parent.

Iritis in toxæmia generally indicates an intense systemic infection, and there are usually present the dull, sallow complexion, constipation, neurasthenia, and lassitude. Given a patient with these symptoms well marked, who is not syphilitic, and the diagnosis may not be difficult. But in other cases we have to contend with the uncertainties which always attend a diagnosis by exclusion.

It is not for those of us whose connexion with toxæmia is chiefly clinical to consider whether the pathological process is an autointoxication, a term which Adamson so much objects to, or whether it is better described as a low infection.¹¹ As practical surgeons we recognize and treat the cause and the results. Lawford has pointed out that at present we have to be satisfied with the general term "toxæmia" as a working hypothesis,¹² but there is every reason to believe that in the near future we shall be able to recognize diverse species. In septic and tuberculous iritis, for instance, we have clinical similarity with pathological dissimilarity. The clinical similarity, however, is not clinical identity, and we can diagnose syphilis from tuberculosis by the ocular symptoms. But at present cases of iritis due to many forms of pyogenic organisms cannot be distinguished one from the other by clinical methods alone. The diagnosis and the treatment of iritis require the harmonious co-ordination of the laboratory and the ward, the mutual collaboration of the bacteriologist and the clinician.

The association of dental disease with iritis is perhaps more generally observed than are other sources of sepsis. The patient may complain of joint aches, myalgic discomforts, and little else besides. On examination of the teeth there is found to be pyorrhoea alveolaris, which we are told has existed for some months or even years. Lang's statistics¹³ show that about 64 per cent. of cases of iritis attributed to sepsis, are caused by pyorrhoea. Less frequently we find otitis, sinusitis, or some gastro-intestinal infection. The difficulty in the diagnosis is the connecting link. The iritis is there, the septic focus is there, and we

conclude that the blood vessels are the highway of communication. But clinical investigation gives us little evidence that the stream is toxæmic. We are, too, continually confronted by the difficulty of deciding whether *post hoc* is in conflict with *propter*, or on the same side. The characteristics of the iritis are not pathognomonic. Deposits on the posterior surface of the cornea may or may not be present. The iritis is an anatomical lesion capable of initiation by manifold organisms, but it is an iritis and nothing more. Lacking the nodules of tubercle, and the condylomata of syphilis, it begins insidiously, progresses leisurely, and clears up tardily. If the iris alone be treated relapses will occur, but if the cause be discovered and removed a satisfactory prognosis may confidently be given.

It is, I think, agreed that pyorrhoea alveolaris is the most frequent source of toxæmic iritis, and the reasons would seem to be, first, the great prevalence of pyorrhoea, and, secondly, that the infection in these cases is direct into the circulation, whereas in many other forms of alimentary toxæmia the toxins have to undergo the ordeal of the hepatic furnace. But this prevalence of pyorrhoea may lead us astray if, on finding it present, we at once conclude it to be the cause. It cannot be too insistently brought to mind that a patient with pyorrhoea may also be a sufferer from syphilis or gonorrhoea. There may also be present more than one source of "gastro-intestinal auto-intoxication," evidences of which may be unexpectedly manifested when the urine and faeces are examined. Whatever organ is discovered at fault it should receive treatment at once, the patient being referred to the proper authority—physician, surgeon, laryngologist, rhinologist, aurist, or dentist—without delay. In the case of pyorrhoea the latter will naturally wish to save as many teeth as possible, but when iritis is already established the bias should be in favour of drastic treatment of the periodontal foci of disease. The slower methods by autogenous vaccines, and by local applications to the alveolar pockets, may suffice before the onset of iritis, but the important point is to stop the auto-intoxication by the most expeditious method; that is, to remove all the affected teeth. The cure of the iritis is of paramount importance, and it is better to lose thirty-two teeth than one eye. Whether the oral sepsis is the cause or the result of intestinal stasis should make no difference in the verdict; the teeth must suffer the penalty for guilt as accessories before or after the event. When dental treatment has been carried out, although immediate cure may not follow, we have at least removed a fruitful source of general ill-health, thereby increasing the power of resistance and simplifying the work which atropine will consummate. In the treatment of iritis the primary cause calls with insistent voice for detailed hygiene. Salicylic acid may have a specific action in acute rheumatism, but in iritis, apart from its anodyne effects, it is probably quite useless. The old formula of mercury and chalk is beneficial, but vaccines have proved disappointing. Occasionally a case seems to respond most satisfactorily, but in many no advantage accrues.

I will not take up time by considering the question of diet, except to remark that we shall see less pyorrhoea, and therefore less iritis, when finely ground flour as an article of food is banished from our homes. Some day perchance it will be illegal to bake or to buy, to sell or to swallow, bread made from flour which bakers call "firsts."

The auto-intoxication theory of the origin of fibrositis, rheumatoid arthritis, and other diseases involves such a reversal of old teaching, such a change in diagnosis and treatment, that it is not easy to view the subject in all its bearings. As far as the eye is concerned, the iconoclastic conclusion to which we are led is that iritis is an indication of an infective process in some part of the body, and, conversely, that infection or subinfection is an indication that iritis may be threatening. I have called the subject of this address toxæmic iritis in order to intimate the point to which I wished to draw attention, but the infective theory connotes a toxæmic basis for all types of iritis. No iritis is primary; it is invariably secondary, always infective.

In the future statistics should show a decreased incidence of diseases of the eye if it be proved that those rheumatic twinges which have hitherto been accounted

for by morning mist or evening chill are early warnings of a chronic septic invasion.

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INSECTS AND WAR:

II.—THE BED-BUG (*Cimex lectularius*).

BY

A. E. SHIPLEY, Sc.D., F.R.S.,

MASTER OF CHRIST'S COLLEGE, CAMBRIDGE.

In "x" finita tria sunt animalia dira;
Sunt pulices fortes, cimices, culicumque cohortes;
Sed pulices saltu fugeunt, culicesque volatu
Et cimices pravi nequeunt foetore necari.

AMONG the numerous disagreeable features of the bed-bug is the fact that it has at least two scientific names—*Cimex* (under which name it was known to the classical writers) and *Acanthia*. The latter name is favoured by French and some German authorities, but *Cimex* was the name adopted by Linnaeus and is mostly used by British writers, and will be used throughout this article. One cannot do better than take the advice of that wise old entomologist, Dr. David Sharp, and allow the name "*Acanthia*" to fall into disuse."

The species which is the best known in England is *C. lectularius*, but there is a second species which is much commoner in warm climates (*C. rotundatus*). As regards carrying disease, this latter species is even more dangerous than its more temperate relative. Other species, which rarely if ever attack man, are found in pigeon-houses and dove-cotes, martins' nests, poultry-houses, and the homes of bats.

Fig. 1.—*Cimex lectularius*, male. × 15. (From Brumpt.)

The common bed-bug seems to have arrived in England about the same time as the cockroach—that is, some four hundred years ago. Apparently it came from the East, and was at first confined to seaports and harbours. It seems to have been first mentioned by play-writers early in the seventeenth century. How the insect got the name of "bug" is unknown. It has been suggested that the old English word "bug," meaning a ghost or phantom which walked by night, has been transferred to *Cimex*. This may be so, but the *Oxford Dictionary* tells us that proof is lacking.

The insect is some 5 mm. in length and about 3 mm. in breadth, and is of a reddish or brownish rusty colour,