well as in man-hours. Even better results would be obtainable if patients could be seen in the earlier stages of infection. Of the cases detailed in Table I, 65% needed immediate incision; in the three cases in Table II in which penicillin treatment was started less than three days after the onset of symptoms incision was unnecessary. This once again emphasizes the need for early diagnosis of the various infections of the hands and for appreciation of their serious nature.

Summary

A review is presented of 185 cases of infected hands seen in the surgery of Westminster Hospital—168 treated without, and 17 with, penicillin.

Cases treated without penicillin included 135 of paronychia and pulp-space infection, with an average loss of 21 days' duty; 16 cases of tenosynovitis, with an average loss of 17.5 days; and 17 cases of palmar-space infections, with an average loss of 16.5 days. The average loss of duty for these 168 cases was 20.3 days.

The penicillin-treated series comprised 7 cases of paronychia and pulp-space infection, with an average loss of 9.9 days' duty; 6 cases of tenosynovitis, with an average loss of 9.9 days (excluding Case 13, 7.4 days); and 4 cases of palmar-space infection, with an average loss of 17 days (excluding Case 16, 9.3 days). The average loss of duty in these 17 cases was 10.9 days. If the two unusual cases, Nos. 13 and 16, are excluded, the final average is reduced to 8.9 days—a saving of 56% compared with the non-penicillin series.

On the basis of these results it is suggested that the administration of penicillin in all severe infections of the hand, both in the early stages and after any necessary surgical intervention, should be adopted as a routine method of treatment.

In practice a dosage of 125,000 units in oil or 300,000 units in aqueous solution twice daily has been found adequate.

We are indebted to Mr. G. T. Mullally, Senior Surgeon, Westminster Hospital, whose enthusiasm and interest made this experiment possible; to Prof. R. J. V. Pulvertaft, who made penicillin in beeswax-oil available at a time when supplies were limited; and to the nursing staff of the surgery for their co-operation in carrying out treatment.

One of us (C. M. F.) is grateful to the Medical Director-General, Royal Navy, for permission to publish.

The President of the Medical Defence Union, Mr. St. J. D. Buxton, speaking at the Annual Meeting on Sept. 16, said that during the preparation by the General Medical Council of a Bill to amend the Medical Acts, representatives of the Medical Defence Union (together with those of the sister societies) had been received by the President of the G.M.C. and a subcommittee. Many of the recommendations made by the Union had been well received, but there was not agreement on all matters. In view of a clause in the Bill allowing appeal to a court from a decision by the G.M.C., the position of any member who had been defended by the Union was somewhat anomalous. In order that he should remain a member during the time allowed for decision as to whether an appeal should be lodged and during the hearing of such an appeal, the Articles of Association would be amended. Speaking of the National Health Service Act, he said that legal representation would be permitted on specified occasions, when practitioners were summoned regarding professional procedure and conduct. The Council periodically had to consider whether the Union could defend members against whom proceedings were being taken by patients in a matter not directly one of professional principle. Although the Council might wish to assist the member, the Union's Memorandum would not permit of the risk being covered by the Union. Hence it was recommended that members should take out a policy with a reputable insurance company to cover injury to patients, not connected with professional affairs, when on the practitioner's premises. Those who read the medico-legal matters in the medical and lay press would be aware of the importance of an action concerning assistants' agreements. He advised any members employing or seeking to employ an assistant, and all assistants who were members of the Union, to seek the Union's advice regarding the wording of the agreement, as the Council had obtained leading counsel's opinion and the Secretary was in a position to submit a suitable clause. With regard to swabs, it was noticeable that the retention of swabs in the pharynx after nose and throat operations had recently been more frequent than in the abdomen.

RELATION OF ABACTERIAL PYURIA TO REITER'S SYNDROME

BY

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Abacterial pyuria has been discussed in several recent papers. Its general features are now well recognized, but its aetiology remains obscure. Probably the most commonly held theory is that it is due to a virus infection.

That Reiter's syndrome might be associated with a sterile cystitis has been known from the first. Later, Colby (1944) described three cases with renal symptoms. His second case is of particular interest, since it demonstrates the development and final resolution of a hydronephrosis. This has often been noticed as a renal complication in abacterial pyuria. In addition one kidney was removed because it was thought to be tuberculous. Section of this kidney showed a thin parenchyma with a dilated pelvis and calices and numerous scattered haemorrhages, but no evidence of tuberculosis. Unfortunately a report of the microscopical appearance is not given, but so far as it goes the condition seems to be similar to that described by Donovan (Moore, 1943) in a similar case later proved to be abacterial pyuria. Cystoscopy in Colby's case showed a diffuse reddening of the bladder. No specific treatment was given and the condition cleared up, but there was a relapse four years later. Prostatitis was present in two of Colby's cases, and an abscess formed and discharged in one. Hollander *et al.* (1945) noted a residual prostatitis in four of his cases.

Joint effusions have been reported in several cases of abacterial pyuria lately. If the cases reported below had come under observation at a time when the urethritis and cystitis were present but before the arthritis developed they would undoubtedly have been classified as abacterial pyuria, not as Reiter's syndrome. It would appear that there is nothing to distinguish the sterile urethritis, cystitis, prostatitis, and pyelitis found in Reiter's syndrome from abacterial pyuria. It has repeatedly been stressed that all cases of abacterial pyuria respond to N.A.B. (Baines, 1947; Moore, 1943; Vassallo, 1946). I have found the same response, so far as the urinary symptoms are concerned, in those cases of Reiter's syndrome that I have treated with N.A.B. The joint lesions did not appear to be greatly affected, though they all cleared up more quickly than might have been expected, and no new lesions were seen after the completion of the N.A.B. treatment. No relapses have been seen up to the time they were lost sight of (six to nine months). In so variable a disease this observation is not of much value. It should be noted that Reiter found arsenic, in the preparation used by him, ineffective, as also have some other workers. The failure of the joints to respond was not unexpected, and finds a parallel in the effect of arsenic on the genital tract lesions of abacterial pyuria.

I have suggested elsewhere (Baines, 1947) that abacterial pyuria is a coital, if not a venereal, disease. Vassallo (1946) came to the same conclusion. He assumed that the urethritis and cystitis were parts of an ascending infection. I have gone further and suggested that the whole urinary and genital tract may be infected by an organism gaining entrance through the urethra. It seems possible that the other symptoms seen in Reiter's syndrome may be a further extension of the same process. In the investigation into abacterial pyuria mentioned above it was thought that there was a characteristic cystoscopic picture in this condition. Two of the cases reported below underwent cystoscopic

examination in the acute stage and the findings were exactly similar to those seen in abacterial pyuria.

Lever and Crawford (1944) pointed out that the whole triad of Reiter's syndrome is not always present. This may be due to the urethritis or conjunctivitis not being severe enough to attract the patient's attention. Careful questioning will sometimes elicit a positive history that is not obtained by casual inquiry. However, at the present time many cases of large-joint arthritis of febrile non-rheumatic type are being seen in hospital and no cause found for them. So common were these in one military hospital that a special investigation was set on foot, the results of which are to be published elsewhere (G. Hearn and C. Tinker—personal communication). It is possible that some of these really belong to the same group.

I have not had the opportunity of examining large numbers of women, but the condition appears to be unknown in them. Hollander stresses the fact that although large groups of women were living under the same Service conditions as the men, he did not see a single female case. No undoubted case is reported in the literature. Dienes and Smith's (1942) series of women investigated for L organisms contained some with genito-urinary infections of a non-specific type associated with arthritis. This heavy preponderance of men is also seen in abacterial pyuria. This is a matter of some importance. If the view of the coital nature of the infection in both these diseases is upheld the organism should be found in both male and female tracts. It is possible that it is a normal inhabitant or common infector of the female, who is relatively immune to it. Urethritis in the female commonly goes unnoticed. Further investigation of the apparently minor lesions of the female genital and urinary tract would be interesting, though it is unlikely to progress far until a specific organism can be isolated. Women with arthritis should probably have a more detailed search made for associated lesions.

Aetiology of Reiter's Syndrome

Reiter himself attributed the condition to *Spirochaeta forans*. This has been a persistent suggestion, but there has never been any further evidence in favour of it.

More recently it has been suggested that it is due to a virus-like organism. This is probably based on Collier's (1938) work. He found a spontaneous transmissible arthritis in *Rattus norvegicus*, and was able to isolate inclusion bodies from them. He was also able to produce a similar disease in rats by injecting fluid from the joints or blood from a case of human acute rheumatism. This work was confirmed by Beeuwkes and Collier (1942), who gave warning that the results should be regarded with caution until similar inclusion bodies could be demonstrated in humans without transmission through rats, since spontaneous virus disease is very common in these animals. Findlay and co-workers (1939) confirmed these results and were able to show that the organisms resembled the virus of pleuropneumonia. Preston (1942) agreed with this, and incidentally pointed out that the reaction was a pyogenic one resembling human pyogenic arthritis rather than rheumatoid arthritis. Abscesses were sometimes found throughout the body of experimental animals, especially at the site of needling for injection purposes. This is of special interest, since one of the objections raised to a virus aetiology for Reiter's syndrome and abacterial pyuria is the fact that they are essentially purulent diseases; whereas virus diseases are not commonly so. Dienes (1940) and Dienes and Smith (1942) isolated pleuropneumonia-like organisms (L organisms) from the cervical, vaginal, and urethral secretions of women, and from the urethra and prostatic secretions of men. Some of these women had

gonorrhoea, non-specific infections, or arthritis, but some appeared to be normal. All the men had symptoms of prostatitis. Beveridge (1943) found L organisms in four out of 24 cases of non-specific urethritis. Klieneberger-Nobel (1945) found L organisms in 40% of patients under treatment for venereal diseases, 33% of patients attending with non-venereal gynaecological conditions, and in only 14% of normal ante-partum women. Salaman and others (1946) found L organisms in 6% of normal women and 14% of normal men. The incidence was no higher in men with non-specific urethritis, but was 44% in women with non-specific cervicitis, possibly due to the greater difficulty in excluding gonorrhoea in women. If there was associated gonorrhoea (or *Trichomonas* infection) the incidence rose to 34% in men and 60% in women. If both gonorrhoea and *Trichomonas* were present it was 100%.

This fits in very well with the findings of Dienes and Smith (1942) quoted above, and of Coutts (1942). Denfield's (1946) observations in Nigeria may be of some interest in this connexion. He regards Reiter's syndrome as an allergic reaction to repeated attacks of gonorrhoea. It seems more probable that there is some sort of commensalism and that the L organisms (if indeed they have anything to do with the disease) grow more freely in the presence of other organisms.

It is perhaps permissible to speculate somewhat at this point. In my paper on abacterial pyuria I came to the conclusion that it was almost certainly acquired by coitus, though not necessarily by extramarital infection. This was based on the almost constant history of recent intercourse, both marital and otherwise, in a series of cases occurring in an army stationed abroad. Urethritis was found in almost every case, though it varied from frankly gonococcal, through post-gonococcal and non-specific ones, to a mild mucoid discharge. In some cases there was no shred of evidence that there had ever been any extramarital intercourse, and the urethritis developed after a home leave. One was driven to the conclusion that infection might be due to a normal inhabitant of the vagina. These cases were by no means common compared with those that were frankly venereal. This parallels the state of affairs found by Salaman (1946) and by Klieneberger and Smiles (1942), with L organisms present in about 10% of normal women and about 60% of those with gonorrhoea. Kristjansen's (1930) case is of some interest in this connexion. His patient developed a non-specific urethritis and arthritis a week after intercourse with a girl known to have a vaginal discharge, which was repeatedly found to be negative for gonorrhoea. Another man subsequently developed urethritis without arthritis following intercourse with her. There is thus a recurrent suggestion that there is some vague connexion with gonorrhoea; but that cases do occur without it is not to be doubted. In this the clinical and pathological findings agree. Salaman has made the alternative suggestion that L organisms are stages in the life cycle of the gonococcus.

In view of the suggested mode of infection another organism which must be considered is *Trichomonas vaginalis*. This was strongly pressed as a likely cause of abacterial pyuria by Lydon (1945), but other workers have failed to find it in this condition, nor are the symptoms the usual ones of known *Trichomonas* infection in the male. Incidentally this organism does not usually respond to N.A.B. It is more probable that *Trichomonas*, when present, is a co-infector. Its relation to L organisms is mentioned above.

One other organism has been mentioned. Coutts (1947) attributes Reiter's syndrome to the virus of lymphogranuloma venereum, and states that he found positive

Frei tests in the condition. In the majority of his cases there was an associated gonococcal urethritis. As Coutts (1943) points out, double infections are not uncommon in venereology, and it is possible that yet another organism was acquired at the same time as his two other common infectors. He also found a similar condition in women (Coutts, 1944), and in the so-called Behcet's syndrome. Frei tests are not infrequently reported as positive in Reiter's syndrome. This may be due to the fact that it is probably not entirely specific for lymphogranuloma. Some of the viruses of atypical pneumonia, and possibly psittacosis, appear to have antigenic affinity with that of lymphogranuloma (Sargent, 1945).

Case I

The patient, a male aged 40, five years previously developed a non-gonococcal urethritis one month after exposure. This responded to sulphonamides. Shortly afterwards he had a dry pleurisy which lasted only a fortnight. He remained well for two years, until he again developed pleurisy. This cleared in six weeks, but twice relapsed at intervals of a month. Full investigation revealed no cause for this. Shortly afterwards he had a recurrence of his urethritis, and at the same time developed a painful arthritis of his left knee. Tuberculosis was suspected. X-ray examination revealed an area of translucency at the upper end of the tibia which was not thought to be responsible, and the nature of which was not known. Lymph-gland biopsy was carried out and showed only "chronic inflammatory change." Guinea-pig inoculation with gland extract was negative. He also had a mild conjunctivitis at this time. Both this and the urethritis cleared up and the knee settled completely in about three months.

He had no further trouble until two weeks before his present admission, when his discharge recurred. Ten days later he developed a bilateral conjunctivitis and arthritis of the left shoulder and ankle and right elbow. Most of the inflammation was periarticular. There was some frequency of micturition and occasional "stammering" but no pain.

Investigations.—Urethral discharge and prostatic bead: pus +++++; no gonococci; Gram-positive cocci; culture of *Staphylococcus albus*. Midstream urine: normal; dark-ground, no spirochaetes. Swab from eye: diphtheroids. Blood count: normal. E.S.R.: 29 mm. in 1 hour. Serum agglutinations for dysentery: negative. X-ray examination of joints revealed nothing abnormal. Cystoscopy: the bladder contained a large amount of pus which, in the absence of any sign of inflammation and a clear efflux from both ureters, must have come from the posterior urethra, which was markedly inflamed.

Once again the condition settled down without specific treatment, and at the end of a month he was nearly normal.

Comment.—This appears to be a classical case of Reiter's syndrome following on a non-specific urethritis. There have been three relapses in five years, with complete recovery on each occasion. The association of pleurisy may be of interest. It has not previously been reported as part of the syndrome, and may in fact have nothing to do with it. I have seen one other case in which a severe fibrinous pleurisy with marked x-ray changes was associated with a very acute abacterial pyuria. Further observations are required.

Case II

A man aged 25, with a healthy history; no dysentery. Six weeks before admission he developed a watery discharge after several extramarital exposures. This was non-gonococcal (smears and culture), and cleared up with sulphonamides. Two weeks later he began to have haematuria, dysuria, and frequency (D/N=30/20). A fortnight before admission he developed bilateral conjunctivitis, and four days later his left ankle became swollen. During the following week the other ankle and both knees were involved.

On admission he had a mild conjunctivitis, and a peri- and intra-articular arthritis of the joints mentioned. Rectal examination showed a tender boggy prostate. Urethral smears were

repeatedly negative for gonococci, but contained a lot of organisms and grew staphylococci and *Ps. pyocyanea* on culture. Urine: pus +++++; culture sterile. Cystoscopy: fluffy oedematous cystitis with multiple discrete ulcers closely resembling those seen in abacterial pyuria. The gonococcal flocculation test and Wassermann reaction were negative, as was the Frei test. Stools and agglutinations were negative for dysentery. The blood count was normal, and the E.S.R. was 18 mm. in 1 hour.

He was treated with penicillin, 1,000,000 units, and sulphathiazole, 30 g., without effect. He was then given N.A.B., 0.3 g., three doses at four-day intervals. Improvement in the urinary symptoms was rapid, and he was normal by the tenth day. There was a slight flare-up of the joints after the second injection, and at this time the E.S.R. was 30 mm. in 1 hour. From this time improvement was rapid and he was able to get up in a fortnight, being discharged a few days later. Cystoscopy sixteen days after N.A.B. was started showed a dramatic change in the bladder, which was now normal apart from slight congestion of the vessels.

Comment.—The urinary picture is that of abacterial pyuria, and is associated with conjunctivitis and arthritis. The joints were responding to rest, but the urinary lesions were untouched till N.A.B. was given, when the expected dramatic response was found. Thereafter the joints rapidly settled. It is not possible to say if the N.A.B. had any effect on the joints since they vary so much spontaneously. The flora of the urethra was not regarded as being of much importance in view of the sterile urine and characteristic cystoscopic findings.

Case III

Two months previously the patient, a man aged 22, had been treated with penicillin for acute gonorrhoea. The urethritis never cleared and he began to have haematuria, dysuria, and frequency of micturition. He was given a course of sulphathiazole, which stopped his discharge, but this recurred a week before admission. Four days later his shoulder swelled and became painful. Two days after this the left knee was also involved.

On admission he had an irregular temperature of 100°–103° F. (37.8°–39.4° C.). There was a mixed intra- and extra-articular arthritis of the shoulder and knee. The leucocytes numbered 17,000; E.S.R. 30 mm. in 1 hour. Urine: pus +++++; culture sterile. Repeated urethral smears were negative for gonococci. Mild conjunctivitis appeared on the following day; this lasted for four days, but recurred three weeks later and again disappeared. During the succeeding days various other joints were involved. The pain and swelling flitted from one to the other and recurred.

Cystoscopy showed severe patchy cystitis—parts of the apex and lateral walls being clear—maximal round the ureteric orifices; multiple plum-coloured small ulcers; the general appearance being one of pink fluffiness characteristic of abacterial pyuria. In view of this he was treated with N.A.B. in the usual way. The urinary symptoms cleared up within a week.

The E.S.R. rose to 95 mm. in 1 hour on the eighteenth day and thereafter fell. His joints slowly recovered, and he was able to get up at the end of two months, being discharged a month later.

During the course of his illness he had three attacks of nose-bleeding. It is possible that these were due to involvement of the nasal mucosa, but no ulceration could be seen in the nares.

Comment.—This case was actually the first one seen. The patient was examined at a time when abacterial pyuria was being investigated, and it was clear that so far as his urinary lesion was concerned there was no difference from the other cases we were seeing. (The original presence of gonorrhoea we regard as of no importance, since it is a not uncommon co-infecter in cases of abacterial pyuria. The reasons for thinking that this is so are given in full in the paper (Baines, 1947) on this subject.) This is why we started treating these cases with N.A.B. in spite of the

bad reports from the earlier workers. We found that the urinary lesions clear up in Reiter's syndrome, just as they do in abacterial pyuria. It is difficult to assess the effect on the joints, since these are so variable in any case, but we are inclined to think that, at any rate, no further involvement occurs after the use of N.A.B. An adequate opinion cannot be given until much further trial has been made. One wonders whether the joints may not be an allergic reaction to the primary urethral infection. The effect of N.A.B. on the urethral lesion is in any case obscure (in contrast to the effect on the bladder).

Case IV

A man aged 43 had been treated with penicillin for "urethritis" six weeks previously. Since the dose given was not the standard dose for gonorrhoea it is probable that a firm diagnosis was not made. The urethritis cleared up but relapsed a few weeks later, and at this time he had a mild conjunctivitis which lasted about a week. Shortly after this he began to have painful and frequent micturition. Within a few days he developed an arthritis of his left knee. During the next ten days various other joints became involved, including the right knee, ankles, big toe, shoulder, elbow, and spine.

On admission he was febrile—100°–101° F. (37.8°–38.3° C.) and in great pain. All the accessible joints had the same appearance, being hot, tender, and with intra- and extra-articular swelling. Urine: pus +++; culture sterile. Repeated urethral smears and prostatic beads were negative for gonorrhoea. The gonococcal flocculation test and Wassermann reaction were negative.

In view of the possibility of gonorrhoea, even in the absence of any positive findings (but after primary penicillin), he was given 1,000,000 units of penicillin and 30 g. of sulphathiazole concurrently. This had not the slightest effect. His urethritis and haematuria got worse. The joints varied from day to day. He then had a course of N.A.B. The urinary lesions cleared up in a few days and did not relapse. The joints continued to vary but improved slowly from this time, and he was able to get up in six weeks. When discharged to light duty a month later there was still some residual stiffness, and a little thickening round the ankles. A skiagram of the big toe showed a destructive arthritis of the metatarso-phalangeal joint; the other joints were normal.

Comment.—This case is somewhat similar to the previous one. It is obviously not absolutely possible to rule out gonorrhoea, but we do not regard this as essential. The presence of abacterial lesions responding to N.A.B. makes it pretty certain that even if gonorrhoea was originally present it was a dual infection. It certainly illustrates the difficulties of diagnosis. However, the remarkable recovery of these cases suggests that the condition is not gonorrhoeal in origin.

Case V

This patient, a man aged 26, had had a non-gonococcal urethritis and epididymitis three months before admission. Two weeks before admission he had a sore throat, and 12 days later he developed a mixed peri- and intra-articular arthritis of both knees and one ankle. There was a mild irregular fever. The urine was normal, and prostatic bead, W.R., and G.F.T. were all negative. E.S.R., 36 mm. in 1 hour. With rest in bed there was some improvement, but a week later he had an acute flare-up with pyrexia to 101° F. (38.3° C.). The leucocytes numbered 8,900. E.S.R., 80 mm. The joints continued to vary, the arthritis flitting from one to another and also involving the shoulder. The E.S.R. slowly sank. Since resolution seemed to be very slow he was given a course of N.A.B. After this the temperature remained steady, and the E.S.R. fell to 8 mm. Convalescence was uninterrupted.

Comment.—This case is probably not one of Reiter's syndrome. It is included to illustrate the difficulty in knowing what should be classed under this heading, and as an illustrative case of a type now common. The arthritic

lesions were typical enough of the variety seen in Reiter's syndrome, and it followed a non-specific urethral infection. Conjunctivitis was not seen, but this is often absent. N.A.B. was given to discover if it could be used in any way as a therapeutic test; but it was impossible to say what effect, if any, it had in this case. Certainly the condition settled rapidly after it had been given, but it was already beginning to do so before treatment was started. Until some specific test is evolved or an organism found, this difficulty will continue. All that one can hope is that in queer cases of arthritis the diagnosis will be considered and adequate investigations be made.

Summary

Reiter's syndrome is a convenient diagnosis for cases of arthritis associated with lesions of the mucosal surfaces. Whether it is a single and real entity will not be proved till a causative organism can be identified and Koch's postulates fulfilled.

There is enough evidence to show that it is neither gonococcal nor dysenteric in origin. The reasons for regarding it as being due to a virus are attractive, but at the moment there is little real evidence on this point.

I have tried to show that there is some reason for believing that the primary lesion is probably urethral. The importance of this has not been adequately stressed in the past. There is no apparent difference between the genito-urinary lesions of Reiter's syndrome and those of abacterial pyuria. It has been suggested that the latter is sometimes associated with other lesions of the genital tract, and this may be but another facet of a widely invasive disease.

Treatment is most unsatisfactory. It is exceedingly difficult to assess. The only thing which seems certain is that the urinary lesions when present in the florid state clear up with N.A.B. just as they do in bacterial pyuria. How far this affects the joints is open to doubt, and we must await the passage of time and a careful follow-up of many cases. It has been condemned in the past.

It is perhaps worth calling attention to the not uncommon cases which resemble Reiter's in one or more respects but can hardly be included with certainty. Many of these are associated with non-specific lesions of the genito-urinary tract. Too frequently it has been assumed that these are all gonococcal. This is probably not true, and they should repay a more careful study. Certainly the urethra and prostate must be searched in every doubtful case of arthritis. Greater knowledge will surely follow better investigation.

I am greatly indebted to Prof. A. P. Thompson for permission to use Cases I and II, which were under his care; also to his residents, Drs. Hayward and Fisher, who undertook most of the tests and investigations in these two cases.

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