Now we do not find that the salicyl compounds have any influence on arthritis which is unconnected with rheumatism or the disorders allied to it, so I cannot accept the first view. Then, against the next alternative, we see that, however much the drug relieves the pain and reduces the fever, the rheumatic condition returns immediately the remedy is suspended, a fact which irresistibly suggests the conclusion that the drug operates by checking the outward manifestations rather than by curing it at its seat of origin. We are thus driven by a process of exclusion to the third hypothesis, namely, that the salicylates act by neutralising and preventing the chemical irritation of the joint. This, too, is the view which, as we shall find, covers known clinical facts best.

Dr. Latham, in the lectures already referred to, not only brings forward a mass of strong evidence in support of this doctrine, but is further able to suggest the probable nature of the chemical action of the salicyl compounds. Without entering into the clinical details with which his paper abounds, his view is that whatever salicyl compound is given, salicylic acid is set free in the system, which combines in the blood with glycocine and uric acid or their immediate antecedents, and so prevents all secondary manifestations traceable to this source.

We are now in a position to consider the circumstances under which these remedies are alleged to fail. Probably the most common source of failure consists in a faulty mode of administration. Either the drug is not given in sufficiently large doses nor at sufficiently short intervals. For a young adult, in a case of average severity, the dose should not be less than 20 grains, nor should the intervals between them exceed three hours. In cases of unusual severity, the dose should be repeated every two hours. Of course, it is not necessary to maintain this high dosage. As soon as the symptoms begin to yield, the quantity should be gradually diminished. If the drug be pure we need not fear its toxic effects. When they occur they are seldom alarming, and in any case quickly disappear on reducing the dose or temporarily suspending the drug. There seems good reason for believing that toxic symptoms are less liable to occur when these compounds are derived from willow-bark than when (as is most usual) they are prepared synthetically.

Failure in many cases may be explained by the fact that inflammatory changes have been set up in the joint which are too severe and deep-seated to subside immediately on the removal of the primary irritation. The salicylates remove the latter, but have no direct influence over the inflammatory change in the joint. Thus, we find that the salicyl compounds always answer best in the early stages of the disease, before the joint mischief has had time to make any headway. The longer the treatment is deferred after the invasion of the disease, the more disappointing are its results, other things being equal. Then, again, the treatment is always more satisfactory in first attacks than in subsequent ones, and this in direct proportion to the frequency of recurrence and the shortness of the intervals between consecutive attacks. Precisely similar arguments apply to cases of sub-acute and chronic rheumatism. In these instances it is not that the salicylates act otherwise than in early acute attacks; but, while in the latter the joint-inflammation, though sharp, is short, and quickly subsides on removal of the primary source of irritation, in the former, structural changes of an inflammatory origin have taken place which give rise to persistent pain, beyond the reach of salicyl influence, requiring time and careful local treatment to regain their normal condition.

A third important source of alleged failure is to be found in the association with the rheumatic condition, of more or less grave deterioration of the general health, with nervous debility and depression. It will be more convenient, however, to defer the consideration of this matter until the question of relapse has been discussed. No more frequent charge is brought against the value of the salicylates as a remedy for rheumatism than the indubitable tendency for the symptoms to return as soon as the drug is suspended.

I would explain the relation of the salicyl treatment to relapses in the following way: The cases treated by the expectant method show clearly that acute rheumatism tends to subside spontaneously after an average duration of about three weeks. As a rule, the more frequently the attacks recur, the longer their average duration, until they merge into chronic rheumatism. Now, it has already been shown that the salicylate treatment has no direct effect on the rheumatic process, but produces its influence by chemically repressing or preventing some of its earliest secondary manipulations.

Accordingly, if the drug be suspended during the early days of the fever, relapse is practically certain; later on, relapse is less likely to occur, until at last, when the metabolic disorder which constitutes the essence of the rheumatic process has naturally subsided, relapses

do not occur at all. Applying these facts to actual practice, it is necessary to continue the use of the drug, in reduced doses, for at least three weeks from the date of onset, and even then, on the slightest return of symptoms, to recommence its administration, and to continue it at least for another week or ten days. In this light, so far from the occurrence of relapses depreciating the practical value of the drug, they show up the specificity and certainty of its operation, within its own limits of activity, in a most striking manner.

To return to the influence of debility as accounting for failure of the salicylates in certain cases. It is well known that a common cold lasts longer, and is thrown off with greater difficulty in proportion to the want of vigour and robustness in the person affected, other things being equal. It is precisely the same in acute rheumatism. In proportion as the patient is debilitated and in feeble general health at the onset of a rheumatic attack, the greater the difficulty in reacting from the disturbance which has been set up, the more tedious its progress, and the less complete the recovery. The rheumatic process once started in a weakly subject is certain to be much more protracted than usual, and sometimes exceedingly so. Hence it follows that, under these circumstances, the salicyl treatment would have to be continued much longer than usual, in order to prevent relapse. But there are practical difficulties in carrying this out owing to the toxic and debilitating effect of the drug when given in full doses, at short intervals, and for a long period. Thus, it can readily be seen why the results of salicyl treatment in such cases are so unsatisfactory. The dilemma is this: either the drug is suspended before the rheumatic process is over, in which cases relapses will certainly occur, or it is continued so long as to depress the vitality of the patient, and make it almost impossible for him to throw off the disorder.

With reference to heart-complications, I believe that they occupy exactly the same secondary relation to the chemical changes in the blood as does the joint-affection. Consequently, in the same manner as the joint-inflammation can be prevented by early and vigorous salicyl treatment, so I believe that cardiac inflammation may be prevented. I have never yet seen cardiac complications occur after the first twenty-four hours of salicyl treatment in a case which was known to be uncomplicated at the outset, and in which it was clear, from the rapid diminution of the joint-pains and fever, that the patient was properly under the influence of the drug. When such complications occur during the first day of treatment, it is probable that the inflammation had already begun when the patient first came under observation; and, as with the joint-inflammation, the salicylates have no direct influence of any kind on the inflammatory mischief *per se*.

I venture to hope that these observations, if carefully followed up, will do some little to place one of the most valuable therapeutic discoveries of modern times on a more rational and solid foundation, and enable us to employ the method more intelligently and with greater promise of success.

THE ETIOLOGY OF RHEUMATISM CONSIDERED FROM A BACTERIAL POINT OF VIEW.

By ALFRED MANTLE, M.D., Stanley, Durham.

AFTER demonstrating the bacteria which he had found present during the rheumatic process, Dr. Mantle remarked that although great advances had been made regarding the etiology of disease generally during the last decade, very little had been done to elucidate the causation of rheumatism.

The most common explanation given was that the disease originated from exposure to cold; this, indeed, might be a factor in the production of rheumatism and many other diseases; but until more was known of what this meant, why it was that one particular exposure, probably after many, brought about a certain train of symptoms, the cause assigned did not greatly help us. Pneumonia was frequently spoken of as being produced by cold, but it was now well known that certain micro-organisms were present during the course of the disease, which had (experimentally) caused the disease in the lower animals.

He had investigated, for bacteria, a number of cases of so-called "cold" or catarrh, which had usually been ushered in with chilliness or shivering, sometimes amounting to a rigor, and with a slight rise of temperature; lassitude and more or less aching of the body had been complained of, with or without increase in the lachrymal and nasal secretions. In all of these cases bacteria had been found to be present in the acute stage. This fact was mentioned to suggest that probably the commonly related story of having "caught cold," given as the cause of so many diseases, meant after all not merely a chilling of the body as generally understood, but in many cases the reception of some morbid germ which had found an environment

suitable for its development, the feeling of cold being the first intimation received of its advent.

In many cases of rheumatism, there was no history of exposure, and in the case of hospital patients suffering or convalescing from other diseases, the occurrence of rheumatism showed that the disease might originate without exposure to cold.

Dr. MacLagan, to whom we were indebted for having brought before the notice of the profession the action of salicine in the treatment of rheumatism, had described an analogy between rheumatism and malarial fever. Undoubtedly there were some points of resemblance, more especially in the course which the fever sometimes took in rheumatism, and the irregularity in duration of the disease. It was difficult, however, to believe that the poison was of a malarial nature when we called to mind the large amount of rheumatism seen in localities not at all favourable to the development of malarial fever. It was important also to remember that we never saw intermittent fever presenting any local lesions simulating rheumatism.

He hoped to show a much closer analogy, as well as relationship, between rheumatism and certain other diseases. In determining the nature of a morbid poison and its action upon the body after its introduction, we ought to note carefully: first, the symptoms produced, and secondly, the structures affected. By making such clinical observations and noting the uniform manner in which a poison operated upon the system in producing like symptoms, and altering the same structures, we were enabled to distinguish the poison of one disease from another. Different diseases had thus been separately diagnosed, and a name given to them based on some leading symptom or lesion observed during the course of the disease. If on comparing some diseases generally believed to be distinct, and caused by distinct poisons, we found a great similarity both in the symptoms presented and the structures affected, we should be led to think their respective poisons must be of a similar nature, and if these diseases were brought about by the action of organisms, a further thought would be suggested, namely, that these organisms might be generically related. Such a similarity was to be observed in the case of acute rheumatism, scarlatina, erythema nodosum, infectious and non-infectious sore throat. In all these diseases, the structures commonly affected were the throat, the skin and the serous membranes, particularly those of the joints and chest. These were seen involved in varying degrees, for while one of these structures was principally affected in one disease, the others being impaired only slightly or not at all, in another disease several or all were more or less affected. In acute rheumatism the throat was commonly affected, as also the joints, and in not a few cases the serous membranes of the chest. There was increased action of the skin, and an eruption of miliaria or erythema was not infrequently observed, followed in some cases by desquamation. In scarlatina the throat and skin always suffered, and the serous membranes of the joints and chest were frequently involved. Similar lesions were found, though in a minor degree, in some epidemics of infectious sore throat, a disease closely allied to scarlatina, the skin apparently escaping.¹

In erythema nodosum the skin was always affected, in most cases the joints also, the throat being usually complained of at the outset. Lastly, in acute tonsillitis, the throat was the principal seat of the disease, but the joints were commonly complained of too.

After dealing at length with rheumatic symptoms observed in the aforementioned diseases, based on numerous cases, in order to show the identity of such symptoms with those of true rheumatism, the relationship of these diseases to rheumatism was next pointed out. Rheumatism and scarlatina were not infrequently observed in the same house, scarlatina attacking the younger members of a family, rheumatic fever the older, scarlatina usually entering the house first. Erythema nodosum had also been observed in one member of a household, whilst two others suffered with acute rheumatism. Acute tonsillitis had frequently been observed under the same conditions. Moreover, scarlatina, erythema nodosum, or tonsillitis, might be developed during an attack of acute rheumatism.

There appeared to be certain conditions of the body similarly favourable to the development of rheumatism, scarlatina, and erythema nodosum. The frequency of the rheumatic manifestation, when the uterine function was either increased or interfered with, was very noticeable. During the latter months of pregnancy, after parturition and during lactation, as well as in younger patients with disordered menstruation, and women at the menopause, the poison of rheumatism seemed to find a suitable pabulum. The scarlatinal poison was also known to find a suitable pabulum during pregnancy and in the parturient state, inasmuch that old practitioners had said that puer-

peral fever was always caused by the infection of scarlatina. Erythema nodosum was frequently spoken of as being developed in those who were the subjects of amenorrhœa and dysmenorrhœa.

Referring to the question of communicability and epidemicity of rheumatism, Dr. Mantle considered there were as cogent reasons for believing acute rheumatism to be sometimes communicable and epidemic as there were in the case of pneumonia. Recently he had attended in one house a girl with erythema nodosum. During her convalescence the mother developed a first attack of acute rheumatism, and in another week the father, who had been nursing his wife, was likewise attacked with rheumatic fever for the first time. Scarlatina and erythema nodosum were prevalent in the neighbouring houses.

Four years ago, a lady who was nursing her husband in a third attack of acute rheumatism, took the disease. Dr. Robson, near Sheffield, had observed in his practice that year an unusually large number of cases of rheumatic fever, including two very interesting instances of apparent communicability of the disease. In a solitary farm-house, the father suffered with rheumatic fever; his two sons, who were living in the same house, and were frequently in contact with him, both took the disease, each having a severe attack of rheumatic fever.

In another instance, a young lady, who was suffering from an attack of acute rheumatism, was frequently visited by her sweetheart, who in a few days took the same disease. In the month of May he witnessed four cases of rheumatism in separate houses within a radius of fifty yards, also ten cases in a small village, in the space of three months.

Undoubtedly it was more usual to see sporadic cases of rheumatism, but he quite conceived the possibility of an organism having greater infective properties at one time than another, which was frequently observed with other fevers. He suggested that more facts concerning the communicability and epidemicity of rheumatism might be obtained through the Collective Investigation Committee.

The result of these analogies and clinical observations had led him to believe that all these diseases were brought about by poisons of a similar nature, though possibly specifically distinct, and that they were probably caused by the action of bacteria introduced into the system through the mucous membrane of the throat, for in nearly all of them the first signs of the disease were manifested in the throat. He felt, however, that valuable as these facts were, bacteria had yet to be found during the rheumatic process. Having, then, studied the methods usually employed in investigating disease for bacteria, he applied them in cases of rheumatism. He would give the results of his investigation so far as it had gone.

Cover-glass preparation of Blood: Acute rheumatism. ($\frac{1}{2}$ oil-immersion. No. 2 ocular.) Klönne and Müller's.

A typical case of acute rheumatism being under his care, under strict antiseptic precautions, and by means of a sterilised hypodermic syringe, about a drachm of serum was withdrawn from the knee-joint. Several sterilised tubes of gelatinised meat-infusion were at once

¹ See author's paper, Infectious Sore Throat, in which Rheumatism Played a Prominent Part, JOURNAL, Vol. II, 1885, page 960.

inoculated, and in each tube a copious growth took place. In six other cases serum had been withdrawn in the same way, with like results. Serum being not always available, whilst blood was, and feeling it to be of equal importance to discover if organisms were present also in the blood, in sixteen cases of acute rheumatism, including some which would be called subacute, blood had been taken from the general circulation, with every precaution to prevent contamination from without, when in every case bacteria had been found.

Cover-glass preparation of Serum. ($\frac{1}{2}$ oil-immersion. No 5 ocular.)
Klönne and Müller's.

In all of these cases there was considerable fever, joint-inflammation, and sweating, and in most the usual complications. His attention, however, had been directed to the less acute and chronic forms of the disease, both in children and adults. He believed as much rheumatism remained undetected in children, and boys and girls about the age of puberty, as was detected. The importance of marking the most insignificant joint-pains in children could not be over-estimated. Such children, if brought to us, were said to be suffering from "growing-pains." There was no proof physiologically that normal growth of the joint-structures caused pain. The course the disease here took was most insidious, so much so that he would call it "insidious rheumatism." Its progress, however, was in most cases very sure, and sooner or later we saw an acute outburst of the disease, either in the form of rheumatic fever, tonsillitis, erythema, pericarditis, endocarditis, or chorea, or the heart might become slowly involved, and the joints slowly disorganised, producing the so-called rheumatoid arthritis of the young. The blood of ten such cases had been examined by him for bacteria, with the same results as in the acute form of the disease.

Chronic rheumatism in adults, both with and without deformity, had also been investigated. In some of these the disease had come on insidiously, being apparently located in one or two joints, though the changes which had taken place in the valves of the heart showed the disease had not been so limited. In other cases a history of an acute attack was found, the patients stating they had never been free from pains for long together since their first seizure. This he thought opened out a very important fact, that in some cases, when once a child or adult has had rheumatism, a "status rheumaticus" was produced, which showed itself by a recurrence of pains of greater or less severity, usually more manifest when the body was in a depressed condition.

In all of these, as well as in that other form of rheumatism known as rheumatoid arthritis, he had found bacteria. Though there was much difference of opinion, he believed that rheumatoid arthritis was only a manifestation of the one disease rheumatism, for a careful history generally disclosed rheumatic pains or fever having been suffered from when young, or an inheritance of rheumatism. He had only had the opportunity of investigating one case of gonorrhoeal rheumatism, in which he found similar organisms in the blood as in other forms of rheumatism. He believed rheumatism occurring during gonorrhoea was not directly dependent upon this disease for its causation, or, instead of seeing an odd case now and then develop rheumatism, we should meet with it much more frequently. It was probable that those in whom it did occur were predisposed to rheumatism by inheritance, or by a peculiar condition of body resulting from an exhausting discharge, making a suitable nidus for the rheumatic poison. The obstinate and destructive character of the

joint-affection was explainable by the neglect which, for obvious reasons, this form of rheumatism received, for good results were obtainable when the patient at once took to his bed.

Before describing the bacteria that he had discovered in rheumatism, Dr. Mantle wished it to be understood that he did not bring forward this subject as completed; it was merely a preliminary report.

He had found two kinds of bacteria, a micrococcus and a small bacillus. Cover-glass preparations of blood and serum showed micrococci as single cocci or pairs (diplococci) and in acute cases zoogloea masses; in addition, small short thick bacilli were also seen either singly, in pairs, or in colonies. Plate-cultivations had been made both of serum and blood, and both kinds of bacteria had been observed under a low power of the microscope, and pure cultivations of each had been made in gelatinised meat-infusion and nutrient agar-agar. He had found that in an acid medium these bacteria grew best. At a temperature of 30° C., a rapid growth took place chiefly on the surface, which presented a prettily reticulated brown appearance. The macroscopical appearances differed in a neutral or alkaline medium; the growth was not so rapid, and its surface had a greyish-white appearance. The bacilli had been observed to become longer and more rod-shaped in an acid medium, and sporulation had been noticed.

Third Cultivation of Bacilli in Acid medium. ($\frac{1}{2}$ oil-immersion No. 5 ocular.)
Klönne and Müller's.

Second Cultivation of Bacilli in neutral medium. ($\frac{1}{2}$ oil-immersion. No. 5 ocular.) Klönne and Müller's.

These bacteria were easily stained with methyl-violet, fuchsine, or by Gram's method. The utmost caution had been exercised to prevent contamination from without. In two cases of purpura rheumatica, he had found no bacilli. In the one case of gonorrhoeal rheumatism, only bacilli had been found in the blood. He had observed bacilli in the desquamating cuticle of one case of acute rheumatism in which desquamation was marked. He thought micrococci were more noticeable in the acute stage of the disease, but had observed both kinds in chronic cases, examined at frequent intervals. Experimentation only could decide if these organisms were the cause of the disease. One thing was certain, that it was not the organisms in that or any other

disease which directly caused the symptoms, but the chemical products the result of their action upon the tissues. This, he thought, opened out the interesting question, might not lactic acid, which had been said to play such an important rôle in the production of rheumatism, be the chief ptomaine of this disease? The well-known experiments of Dr. Richardson, and the observations by Sir Walter Foster of the development of rheumatism during the administration of lactic acid, suggested its being an important factor in the production of the disease. The manifestation of similar symptoms in other diseases might be accounted for by the ptomaines being of a similar nature. He had found that cultivations of bacteria found respectively in rheumatism, tonsillitis, erythema nodosum, and scarlatina produced lactic acid fermentation in sterilised milk.

In conclusion, Dr. Mantle stated that he hoped to give the results of further research into this interesting question at a future time. He felt, however, convinced that rheumatism was produced by the agency of a bacterium, which he thought in most cases entered the system through the lymphoid structure of the tonsil. The necessary pabulum being found, the organism multiplied, its morbid action was confined chiefly to the lymphatic system, the serous membranes being affected in preference to other structures. The fact of salicine—a potent germicide—being so successful in the treatment of the disease strengthened this theory.

NOTE ON SOME POINTS IN THE NATURAL HISTORY OF ACUTE RHEUMATISM.

By ARTHUR DOWNES, M.D.,

Medical Officer of Health to the Chelmsford Combined Districts.

In the admirable memorandum, and in the not less excellent card on acute rheumatism issued by the Collective Investigation Committee of this Association, I do not observe any reference to the question of heredity as a possible factor in the causation of this disease. It may be that the compilers were anxious to curtail the field of their inquiry, and in this they would be wise, for in every investigation of Nature we must be content to seek and to learn a little at a time, to crawl before we walk, to walk before we attempt to run. One is loth to criticise even the details of an enterprise endowed with such promise of success, and having such possibilities before it; yet the fear presents itself that some of the inquiry-cards are over-freighted by their load of questioning.

Still, in any systematic inquiry, it seems to me that the problem of heredity must of necessity be among the first for solution. Apart from its intrinsic importance to the community, to say nothing of its interest as a question of pure science, the influence of heredity constitutes one of the two great branches into which etiology divides. Its general tendency is opposed to that of the environment—the influence *des milieux* of the French—and I fail to see how we may scientifically gauge the effect of the one while we remain ignorant of the other.

The men who can throw most and the best light on this matter are the general practitioners, to whose abilities Dr. Stephen Mackenzie lately paid in these columns a well-merited tribute of acknowledgment, and whose opportunities for acquiring trustworthy knowledge of family tendencies to disease are well summed up by him.

I do not remember that the text-books give much information on heredity in regard to acute rheumatism; systematic writers, indeed, commonly contrast gout in this respect. From time to time, however, one meets, both in practice and in medical literature, with instances of marked family predisposition to rheumatic fever. I came across such an one while lately inquiring into a little epidemic of erysipelas.

S. P., aged 51, an agricultural labourer, was attacked by facial erysipelas early in August, followed shortly by acute rheumatism, from which he had previously suffered eleven years ago. F. P., brother of the above, aged 47, living in a cottage hard by, came in to lift his brother up in bed. On August 20th he, too, sickened with erysipelas, which in its turn was likewise followed, in about fourteen days, by acute rheumatism, with pericarditis, from which he died on October 1st. I did not inquire exhaustively into the family history, but I learned that two other brothers, W. P. and J. P., have had rheumatism of some kind; that last Easter, two young women, daughters of S. P. and F. P. respectively, had come home almost simultaneously from different situations ill with acute rheumatism, one of these being a second attack; that another daughter of S. P. had had it two years ago, and a daughter of F. P. had died of the same disease nine years back. Influences of locality appeared to me quite insufficient to explain this exceptional incidence on a particular family.

There is another point of interest in this history. Both in S. P. and F. P. an attack of erysipelas appeared to act as the exciting cause of the rheumatism supervening in individuals already specially predisposed to it. Trousseau (*Sydenham Society's Transactions*, iv, 442) describes a similar sequence, and compares erysipelas in this relation with scarlet fever. There is, indeed, a curious connection between a number of pathological states which group themselves around rheumatic fever, for the unravelling of which we look hopefully to collective investigation.

Urticaria, for example, is well-known as an occasional concomitant of acute rheumatism, and Hebra (*Sydenham Society*, i, 310) asserts that urticaria is more common when erysipelas is prevalent than at other times. The same author (*Ibid.*, p. 291) considers erythema nodosum to be allied to the erysipelatous diseases, and I once received (*apropos* of some note I had published) a communication from a practitioner in the Black Country, drawing my attention to a sequence which he believed he had observed between erythema nodosum and scarlatina; in fact, to use his own comparison, he regarded the former as certain a harbinger of the latter as the cuckoo is supposed to be of spring. I am bound to say that I have only once, during the eight years which have since gone by, observed a concomitance of these two diseases, but then erythema nodosum does not often come to my notice as a non-practising official.

This leads me to speak of another feature in the clinical relations of acute rheumatism. It is half a century since Rayer described the occurrence of a papular rash (*E. papulatum*) in this disease, and nearly as much since Bouilland and Schönlein respectively pointed out its relations to erythema nodosum, and to a form of purpura. The latter, indeed, under the names of purpura rheumatica (Reder), peliosis rheumatica (Schönlein), and rheumatocelas (Fuchs) has been described as an independent disease. I do not pretend to have studied the pathology of this affection, but in what I have personally observed of it, I have seen nothing but acute rheumatism *plus* certain cutaneous and other results of multiple thrombosis and embolism. This was Trousseau's view. Acute rheumatism, he argued, is the morbid state in which the blood contains the greatest proportion of fibrin, and that is not a condition predisposing to hæmorrhage. May it not be, then, that the petechiæ and ecchymoses which one meets with in acute rheumatism depend on numerous obliterations of veins and consequent extravasations of blood? (*loc. cit.*, iv, 448-9).

I believe that these irregular forms of acute rheumatism—at least so I regard them—are more common than is generally supposed, and I think that not seldom they are relegated to the vague and unsatisfactory category of "blood-poisoning." In one of my annual reports about three years ago, I gave a short description of six cases, one of which I saw myself, occurring in the practice of my friend, Mr. Arthur Goodchild. The chief features of these cases, as observed by him, were: 1. Articular rheumatism, with possibly cardiac complications; 2. Erythema nodosum; 3. An eruption, sometimes occurring in crops, papular, measles-like, or purpuric; 4. Orchitis in two; and 5. Albuminuria, intense and persistent, or, in two cases, intermittent. These symptoms were not necessarily all present in the same case. I have not been able to find any previous notice of the occurrence of orchitis or of albuminuria—both highly important observations, it seems to me. These cases were all males, and all young, some, indeed, small children. I mention this because the writer of the article on Peliosis Rheumatica in Hebra (*loc. cit.*, iv, 422-3) says that it had never been seen in children. On the other hand, an article by Aug. Schmitt, "On Peliosis Rheumatica of Children," is referred to in *Syd. Soc. Year-Book*, 1860, p. 403. Unless this article happens to resemble the famous chapter on "Snakes in Iceland," it should, with Mr. Goodchild's experience and my own, modify Hebra's assertion. A similar train of irregular symptoms accompanying acute rheumatism was described by Trousseau under the misleading name of erythema papulatum.

TINCTURE OF SIEGESBECKIA ORIENTALIS IN RINGWORM.

By J. HUTCHISON, M.D.,

Physician to Anderson's College Dispensary, Glasgow.

Of the drugs now generally prescribed in the treatment of tinea in its several varieties some are exhibited in the form of ointments, and ointments are always more or less disagreeable to use; others free from greasy annoyances, give the patient more or less pain in the application. The preparation I have been using for some time past is devoid of both these drawbacks, and at the same time is speedy and reliable means of curing the disease.