when an acid diminishes the excretion of uric acid in the urine, it does so by diminishing the solubility of uric acid and causing its retention, in accordance with Sir A. Garrod's observation, in the liver, spleen, and joints, which are naturally less alkaline than the blood and tissue fluids of the rest of the body; and my results are obviously in complete accordance with this suggestion.

I have examined upwards of sixty specimens of blood, some drawn during life, but most taken from the heart cavities after death, and all tend, I think, to show that there is excess of uric acid in the blood—some specimens containing as much as 0.07 to 0.08 per cent.—under exactly the same conditions in which there is excess of it in the urine, and recently I have drawn the urine from the bladder at the same time that I took the blood from the cavities of the heart, and my results seem to show that excess in the urine corresponds and coincides with excess in the blood, and scantiness in the urine with a very small quantity, or even none, in the blood.

Again, my results show that blood drawn during acute fever, as pneumonia, or at the seat of a local inflammation, contains almost no uric acid, and I have previously mentioned that Sir A. Garrod found none in the blood and serum drawn under similar conditions. On the other hand, blood obtained after death from chronic wasting disease, especially if there are urate deposits in the joints, may contain a very large amount, as 0.08 per cent.

From all these facts, I infer that it is safe to argue from the urine to the blood, and that an excess of uric acid in the urine above the relation to the urea of 1 to 33, comes from and is the index of some excess of uric acid in the blood—a fact which I was led to infer several years ago from the physiological and pathological results I can produce by influencing the excretion of uric acid by the urine. And results thus constant and in harmony with those previously obtained by others are, I submit, incompatible with the existence of errors in the process sufficient to vitiate it for clinical and comparative purposes; and, used with due care and sufficient knowledge and experience of the sources of error, I believe that Haycraft's is by far the best process for the clinical estimation of uric acid at present known.

TUBERCULOSIS IN THE SAUROPSIDA.

Read in the Section of Pathology at the Annual Meeting of the British Medical Association, held in Birmingham, July, 1890.

By WALTER K. SIBLEY, M.B.CANTAB.,
Assistant-Physician to the North-West London Hospital.

THE vertebrate kingdom of animals has been divided into three main divisions, namely, the ichthyopsida (fish and amphibians), the sauropsida (reptiles and birds), and the mammalia (mammals).

Tuberculosis, the commonest disease of domestication both in man and many of the lower animals, has not as yet been found in the ichthyopsida, and, with but very rare exceptions, in only one division of the sauropsida—that is to say, the birds. Until the last few months back no case had been described in the reptiles, but now we find isolated cases occurring in the ophidia when kept in confinement.

PART I. Birds.—The abdominal organs—namely, the spleen and liver—were the parts primarily affected; the abdomen often being bulged from the enormous size of the liver, whilst the spleen was usually enlarged many times its natural size. In some cases no deposits were seen in the intestines, at other times these organs were beset with hard nodules often as large as beans. Microscopically the deposits in an early stage consisted of small groups of epithelioid cells, which were often apparently lying in vessels and enclosed by the vessel wall. In a much more advanced stage, each deposit consisted of a central mass of caseation, which was very irregular in outline, and surrounded by a zone of giant and epithelioid cells undergoing degenerative changes. Surrounding this was a ring of granulation tissue, the whole being encapsuled by fibrous tissue. In a still more advanced stage there was a considerable development of fibromyxomatous tissue. In these last stages no bacilli were present in the newly-formed fibrous tissue, but they occurred in densely packed groups in the caseous masses. In the intestine the lesions varied from a

small ulcer with thickened edges to pouches occasionally as large as small oranges springing from the gut walls. The walls of these were formed chiefly of caseous tissue and fibrous tissue. Often the lungs presented no obvious lesion. In a few cases there were caseating lymphatic glands in the base of the neck. Tubercular disease of the bones was very rare. In one case of a fowl there was a caseous mass in the neck of the thigh bone, and in another case some caries of the vertebrae. The example of a peacock presented several tumours in connection with the bones and joints, such as the scapula, sternum, and knee-joint; several of these tumours were as large as walnuts. Microscopically, they were formed of granulation tissue, with groups of epithelioid cells, and also giant cells here and there. Tubercle bacilli occurred in parts of these growths.

The bacilli vary much in different classes of birds both as to their number and arrangement, and also their actual size. All of these I believe to be merely local varieties of the same bacillus, the result of the slight differences in the soil in which they are growing. In birds the masses of caseation are very firm; often a furrow appears around them, by the growth and contraction of the fibrous tissue, and so the deposits, which are close to the surface of an organ, may in this way become completely separated. In some birds I have found the nodes formed in the liver lying loose in the abdominal cavity. The deposits appear never to go beyond the stage of caseation; they do not of themselves break down and form cavities, nor do they become calcified. There is a tendency for the disease to extend by continuity and to a considerable new tissue formation.

In a large number of cases which occurred in a farmyard among the fowls there was very strong evidence that the disease was hereditary in its nature, in that the birds became affected during consecutive years when about the same age—that is, young adult life. Also the disease was limited to certain broods, others which lived along with these remaining free from the affection. So, also, pigeons and wild birds feeding in the same yard never became subject to the affection, and the disease was not present in the neighbouring farms.

I have thus far found tuberculosis in the following birds dying naturally in confinement, the diagnosis being in every case made from the microscopical appearances of granulomata undergoing central degeneration, together with the presence of the bacilli: canaries, finches, fowls, geese, guan, owls, prafowls, pheasants, pigeons, swans and vultures.

PART II. Snakes.—The first case I met with of tuberculosis in snakes occurred in an Italian snake (*Tropidonotus matrix*), which died in the Zoological Society's Gardens; it is described in full in *Virchow's Archiv* for 1889.¹ This case was marked by the number of tumours in various regions of the body together with general visceral tuberculosis, tubercle bacilli being abundantly present in most of the lesions. Here the seat of the deposits appeared to be in the lymphatic vessels, there being a complete tubercular thrombosis of the main lymphatic channels. The microscopical structure of the lymphatic deposits was the same as that of the visceral deposits.

In addition to this well-marked case of tuberculosis I have examined a few specimens of what appeared to be the same disease, in which the lesions were confined to the subcutaneous tissues. A viper had a large tumour on the side of the neck, which had been watched for over two years without showing any appreciable change in its size. This tumour was composed of very dry caseous tissue lying in definite areas of fibrous tissue.

The study of tuberculosis in reptiles is of great interest, in that the disease can be studied with a complete control of the animal's temperature, as the temperature of a snake's body does not differ much from that of the surrounding medium, so they may be kept at a given temperature and the disease under its various aspects so studied. With this object I have kept various members of the reptile class (inoculated with tubercle) in incubators regulated to definite temperatures. After considerable difficulty I have succeeded in producing tuberculosis in snakes by the inoculation of pure cultures of the bacilli and keeping the reptile at about a temperature of 35° C.

¹ Translated in the *Journal of Anatomy and Physiology*, 1889.

The first successful case died 52 days after the primary inoculation, with miliary tubercles in the liver and lungs, etc. The deposits appeared to be of a caseous nature, most of them containing groups of tubercle bacilli. A full description of inoculated tubercle in reptiles I hope to publish shortly.

In the cases occurring in the London gardens it is seen how the disease is arrested by reduction of the temperature. Thus the average temperature of the reptile house is decidedly lower than that at which the tubercle bacilli can grow, that is to say, it is only during the summer months that the disease can be active, and so far I have only found cases of the disease in those snakes which are kept in cages exposed to the full rays of the sun.

PROTECTIVE AND ANTAGONISTIC INOCULATION IN LEPROSY.

Read in the Section of Pathology, at the Annual Meeting of the British Medical Association, held at Birmingham, July, 1890.

By BEAVEN RAKE, M.D.LOND.,

Medical Superintendent of the Trinidad Leper Asylum.

MANY diseases have been described as retarding the progress of leprosy. Thus Danielssen and Boeck,¹ speaking of the effects of an outbreak of variola in the St. George Leper Asylum in Bergen, says: "Après la guérison de ces ulcères et l'entière destruction de ces tubercules, et à l'exception de cicatrices, tantôt petites, tantôt grandes, mais enfoncées, la place ne laissait sentir aucune trace de tubercules aux endroits qui s'en trouvait affranchis." I have noticed similar swelling of the skin and subsequent disappearance of tubercles at the Trinidad Asylum in patients whom I have vaccinated. The following is a typical case:

E. A., negress, aged 17, suffering from mixed leprosy, admitted February 12th, 1889.

February 15th. Vaccinated on left arm.

February 19th. Face swollen; arm beginning to rise, but no inflammation at the site of vaccination.

February 21st. Four successful insertions.

March 1st. Swelling has gone down.

March 2nd. Tubercles almost gone from face; skin dry and wrinkled; fever gone.

March 15th. Face and extremities desquamating; hardly any tuberculation to be seen.

March 18th. No tuberculation; some swelling of fingers and toes.

Hardy mentions pleurisy, pneumonia and variola as acting similarly. Leloir² also describes erysipelas as retarding leprosy. This I have noticed in the asylum here. He also mentions a disappearance of cutaneous tubercles when the patient is attacked with phthisis. This is very marked towards the end of old tuberculated cases in Trinidad, and on *post-mortem* examination I have found tubercles not only in the lungs, but often also in the viscera. The connection between leprosy and tuberculosis I hope to discuss in a future report.

All these instances of the retarding effect of other diseases on leprosy seem to point to the possibility of antagonistic inoculation; for variola, pneumonia, erysipelas, and phthisis are each associated with a distinct micro-organism.

It was not long, therefore, before such inoculations were attempted. Cornil injected jequirity into leprosy patches without effect. Campana inoculated lepers with erysipelas with the result that nearly all the patients in the ward got erysipelas, and the ward had to be closed. No effect was produced on the progress of the leprosy.³

In view of this apparent antagonism, I have been making during the year some experiments on the treatment of leprosy by inoculation of cultures. As we are not yet certain that the bacillus lepræ can be grown on the ordinary media, protective or antagonistic inoculation may appear somewhat premature. Still I thought it worth while to take a working hypothesis, and go a little ahead of our actual knowledge, for the cultures from fragments of tubercles or viscera might contain either spores or some secretion of the bacillus, and so set up some recognisable changes when injected beneath the skin.

Even if the growths are not leprosy they may be of value in destroying leprosy bacilli, for Campana has described relief in phthisis from the inoculation of bacterium termo.

There are three conditions in which such inoculations might be of use:

1. In anæsthetic leprosy, to arrest further growth in the nerves.

2. In tuberculated leprosy, to cause local destruction of tubercles, or to protect against further growth.

3. In leprotic fever, to arrest the outbreak of tubercles.

In the first series of cases I have found temporary swelling, redness, and sometimes suppuration, but no permanent effect. I may mention here that I have never known an anæsthetic leper develop tubercles. Leloir,⁴ however, mentions three cases in which pure anæsthetic leprosy afterwards became mixed from the development of tubercles. In one of these cases the tubercles disappeared by suppuration, and the case became anæsthetic again. One or two old anæsthetic patients at Cocorite have told me that they were formerly tuberculated. The alleged disappearance of tubercles was, however, before my time, so that I cannot vouch for its truth, nor can I be sure whether they were anæsthetic before they became tuberculated.

In the second series I have noticed in some cases a certain amount of local ulceration and destruction of tubercles, but not to any marked extent. The ulcers soon healed, leaving the tubercles nearly as large as before.

In the third series I thought that the receptivity of the patient might be increased by the leprotic fever, and that possibly the leprosy bacilli, being more active at that time, might be more amenable to the influence of antagonistic cultures. The same local effects were, however, observed in these cases also.

[A detailed account of the various inoculations was given in a table which was shown at the meeting.]

FURTHER PROCEEDINGS OF THE SECTION OF PATHOLOGY.

The Tongue in Disease.—Dr. W. HOWSHIP DICKINSON exhibited a series of macroscopical and microscopical drawings:—A. The changes represented were chiefly those produced in the organ by diseases external to itself. (Macroscopical and microscopical appearances), namely: 1. Healthy tongue, an average state—epithelial covering, vegetable growths; 2. Stippled tongue; 4. Coated white moist tongue; 6. Plastered tongue; 7. Furred and dry tongue; 8. Encrusted dry tongue; 9. Denuded tongue; 10. Red and dry tongue; 11. Red, smooth, covered with membrane; 12. Cyanosis. B. Some of the morbid states proper to the tongue itself were also shown, namely: 1. A form of glossitis affecting children; 2. Annulus migrans; 3. Leucoplakia in a smoker.

Alcohol as an Antiseptic.—Dr. J. J. RIDGE read a paper on the Action of Alcohol on Micro-organisms and Cell Protoplasm. He described the effect of minute quantities on cell protoplasm, taking first its effect on the growth of vegetable cells, chlorophyll corpuscles, etc., and secondly on animal protoplasm as exemplified by its action on the eggs of the blowfly. He next described its effect on micro-organisms, the bacilli of certain fevers, of septicæmia.

Tubercular Diseases of Bones and Joints.—Mr. WATSON CHEYNE exhibited specimens illustrating the Pathology of Tubercular Diseases of Bones and Joints: 1. Specimens showing the chief types of synovial disease. 2. Specimens showing soft deposits in bones, their mode of growth, and their progress. 3. Specimens illustrating the formation of tubercular sequestra. 4. Specimens showing the destruction of cartilage, and caries of the ends of bones. 5. Specimens of tubercular periostitis and tubercular osteomyelitis. 6. Specimens illustrating the pathology of chronic abscess.

Comparative Pathology of Tuberculosis.—Professor J. McFADYEN (Edinburgh) discussed the Comparative Study of Tuberculosis in Man and the Common Domesticated Animals (ox, horse, sheep, pig, dog, cat, and fowl). The principal points touched on were: The relative frequency of the disease in the above-mentioned animals; the character of tubercular lesions, and the differences in their distribution and histology in different animals. The distribution of tubercle bacilli, their relative abundance, and the question of their identity

¹ *Traité de la Spédaleskhed*, p. 324.

² *Op. cit.*, p. 224.

³ Leloir, *op. cit.*, p. 3.6.

⁴ *Op. cit.*, p. 209.