are rife in other parts, where the inhabitants are more or less inured to them. In this way, it is possible that these agents of disease are innocuous unless a chill, damp, or other condition inimical to health predisposes the individual to their attack. Not so in St. Kilda. This inoculation of the inhabitants does not take place, consequently they suffer as a rule when they are exposed to their influence. Is it not also possible that consanguinity may be a factor in the predisposition of this disease as well as of the infantile affection?

As far as I could learn, there never has been a *bond fide* case of phthisis pulmonalis. Some people were said to have died of "cait-heabh," which, in their language, means a wasting; but, considering their ages and other circumstances, I am led to believe that chronic bronchitis was the cause of death, and not phthisis. As to the other diseases existing there, I can fully confirm what Dr. Acheson, R.N., of H.M.S. *Jackal*, states in his notes of a visit to St. Kilda, which appeared recently.

As no medical man has stayed longer on the island than during the short periods boats usually anchor in its somewhat unsafe bay, all our information regarding the trismus neonatorum and the "strangers' cold," must still be considered unsatisfactory. The accounts we have, however, of the diseases which are peculiar to the natives of St. Kilda, are sufficient to show that here is a field where scientific observations might throw much light on some obscure points in the etiology and pathology of disease; while, from an ethnological, as well as from a medical point of view, this small community, isolated for many centuries from the outer world, forms a subject for much interesting study.

SATURNINE GOUT, AND ITS DISTINGUISHING MARKS.

By G. LORIMER, M.A., M.D. EDIN., Buxton.

LEAD-IMPREGNATION is a predisposing cause of gout. In gout so caused, two morbid influences must be considered: 1. Lead-impregnation, with the blood-changes and cachexia which it induces; 2. Gout, with its complications and tendency to tissue-degeneration. It is proposed to inquire if their mixture affects the course of the latter, and confers upon it any special distinguishing characters.

It may be premised that, in the initial stages in some cases, no deviation from the appearances of ordinary gout is observed; that the course and character of the disease may be affected by the relative predominance of one or other morbid state; and further, that there is no one and special sign which marks saturnine gout as a distinct morbid entity. The appearances observed in the fully developed and more advanced stages are such as *a priori* might be expected to occur, but, taken in the aggregate, constitute a group of distinguishing marks.

The conclusions arrived at are based upon an analysis of 107 cases of gout due to plumbism, which have occurred in the writer's experience, and the subsequent remarks constitute a record of facts so observed.

Briefly to summarise, the distinctive marks are as follows.

1. *Age*.—The first attack of gout due to lead-impregnation occurs at an earlier age than when independent of it. Of 107 cases, the first attack occurred prior to 35 in 70 cases; and, in the remaining 37 cases, occurred for the first time between the ages of 35 and 65. This contrasts with the history of non-saturnine gout; for the first attack of the latter usually occurs from 35 to 40, while acquired gout is frequently a disease of later life.

2. *Hereditary Tendency*.—In saturnine gout, the influence of direct hereditary predisposition is less marked. Of 107 cases, it was noted in 9; and, even admitting that deductions drawn from negative proofs are only partially conclusive evidence of its absence, still, this percentage contrasts with non-saturnine gout, where hereditary predisposition is found to occur in at least 50 per cent. When hereditary tendency includes the arthritic group of diseases, the predisposition is increased from 9 to 30 per cent.

3. *Anæmia*.—In gout, the blood-corpuscles undergo no change in number and quality. The anæmia of saturnine gout results from the lead-cachexia, and is probably due to the action of lead on the blood. The red corpuscles are diminished in number, the white sometimes increased, and the red colouring matter is reduced. Anæmia was noted in 75 cases; in the remaining 32 cases, it was less marked or absent. The anæmia of saturnine gout not only contrasts with the sanguineous arthritic diathesis, but affects and modifies the type and progress of the disease.

4. *Asthenic Type of Arthritis*.—From the early age at which saturnine

gout occurs, when functional activity is more vigorous, asthenic and acute type of arthritis might be expected to prevail. In the early attacks in many cases, and in subsequent attacks in a minority of cases, the arthritis assumes this type; but, in the majority of cases, the type of arthritis is asthenic or adynamic. The local and constitutional phenomena are less intense, but more persistent, lingering, and obstinate, and show a tendency to pass insidiously into a chronic form. The asthenic type of arthritis appears due to impaired vital activity, resulting from the cachexia, and to organic renal changes which subsequently arise.

5. *Albuminuria*.—The experiments of Ollivier, denied by Rosenstein, but repeated with affirmative results by Charcot and Gumbolt, show that albuminuria occurs in lead-impregnation. On the other hand, Dr. Garrod has found that in gout, especially in its chronic form, albuminuria is present in 26.5 per cent. of cases. *A fortiori*, it follows that, in saturnine gout, albuminuria would occur more frequently. In the 107 cases, it was present in 89, either as an intermittent or permanent condition. In 18 cases it was not noted. In the early history it may be absent, in subsequent stages it may be intermittent, but it may be doubted whether it is ever entirely absent during the whole progress of the disease. A daily examination of the urine has, in the writer's experience, seldom failed to discover its presence, and there is no sign in saturnine gout more constant, certain, and characteristic. Further, the specific gravity of the urine is diminished, averaging 1012. The quantity of uric acid is decreased, and, in the final stages of the disease, is absent altogether.

6. *Arterial Thickening and Degeneration*.—This condition, noted in sixty-nine cases, consists of a sclerosis of the arterial coats, along with atheromatous changes. It is, in fact, a premature ageing of the arterial system. *a*. It may be due to the action of lead, which causes contraction of the muscular walls of the arteries, and raises arterial tension. *b*. It may be connected with the renal changes which arise in saturnine arthritis. *c*. It may depend on the condition of the blood in gout, which gives rise to increased arterial tension, and predisposes to atheroma. Cardiac hypertrophy is observed in saturnine gout, especially at the advanced period of the disease. The arterial changes, however, may occur independently of the cardiac. Pericarditis has been noted by Charcot and Gumbolt. One instance only was noted by the writer in the cases referred to.

7. *Cutaneous manifestations* of gout are seldom present in saturnine arthritis. Of 107 cases, one was associated with psoriasis and one with eczema, and in the latter instance there was a hereditary history of gout, contrasting with non-saturnine gout, where eczema occurs in 18 per cent. of cases.

The blue line upon the gums, due to formation of lead-sulphide, was noted in eighty-seven cases. It is a sign which may indicate the cause of the gouty phenomena.

Gouty affections of the eyes were of infrequent occurrence. One case of iritis was noted. Exception, however, may be made to neuro-retinitis, in the causation of which gout and plumbism appear both to participate.

Joints affected in Saturnine Gout.—In twenty-eight cases, the joints of the feet were involved, in seven of which the great toe only was implicated. In twenty cases, the joints of the hands. In thirty-four cases, the joints of the hands and feet were both affected. In the remaining twenty-five, the knees also were implicated, in four of which the elbows also were affected. In twenty-three cases, tophaceous deposits were noted on the helix of the ear. Gout associated with lead-impregnation, however, seems to attack the kidneys. Gout associated with alcoholic excess generally attacks the joints. Lancereaux has observed that, in saturnine gout, uratic deposits are less abundant, and with this opinion the writer's experience is in some measure in harmony. It is, however, a question of degree, not of kind. Their presence is conclusive evidence of gout.

The experience of some observers is that rheumatism, and not gout, is associated with plumbism, and the difference is ascribed to the absence of previous habits of alcoholic intemperance. The explanation is incomplete. Gout may supersede rheumatism. The writer has observed more than one instance in which the question of alcoholic intemperance might be dismissed, where plumbism was originally associated with rheumatism, and in which uratic deposits occurring twelve years subsequently, showed unequivocally the gouty character of the disease. Such cases seem to lend some countenance to the doctrine of a "basic arthritic diathesis," out of which are evolved, under special causes, the several arthritic diseases.

How far saturnine gout may be due to the action of lead on the pneumogastric nerves, leading to imperfect metabolism, and on the trophic centres of the joints in the spinal cord, are subjects at present in the region of conjecture.