tion owing to its tendency to cause vomiting when taken by the mouth.

Large quantities of fluids should be given (water, barley water, etc.). Lactogen, slightly sweetened, has been well tolerated where there has been much nausea, and Benger's food has also been easily taken.

The patient should be kept in bed for at least fourteen days even in the mildest cases. It is owing to patients failing to realize the importance of this that the prognosis of a mild case is so bad.

RESULTS.

Since assuming the post of district surgeon, Bindura, I have seen 36 cases of blackwater fever (29 males and 7 females); 33 recovered and 3 died. The percentage of mortality was thus 8.33. All the deaths occurred in males. Only one was actually due to blackwater fever, and in that one my warning not to move the patient had been disregarded.

Deaths.

Case 1.—A middle-aged man, living temporarily in a tent, developed blackwater fever. On the day of onset I attended him, and found that he did not present any alarming symptoms. He was put on the treatment I have mentioned, and instructions as to diet were given to his wife. She was also warned not to allow him to be moved. On the third day of the disease I had a message stating that his water was now quite clear and that he was much better. On the morning of the fourth day I received a very urgent message saying that the patient had been moved, for better nursing, to a cottage a quarter of a mile away, and was then in an extremely critical condition. I found the patient quite collapsed and very yellow. All attempts to improve matters by the use of hot-water bottles, subcutaneous and rectal saline, injections of camphor, strychnine, and pituitary extract, failed to produce any improvement, and he died twenty-seven hours after having been moved.

Case 2.—A middle-aged man who had no blackwater for ten days, and who had a history of a severe kick in the epigastrium from a mule three months before, developed what clinically appeared to be intussusception and died. He had complete stoppage, passed blood per anum, and had a well defined sausage-shaped tumour in the region of the transverse colon; the diagnosis, therefore, was probably correct. Operation was refused, and I did not urge it owing to the weak state of the patient; the relatives refused a *post-mortem* examination.

Case 3.—A young man developed severe blackwater fever, with scanty urine and retention, which necessitated catheterization during the whole of his illness. The urine became clear in two days, when bronchopneumonia developed; this cleared up in four days and the patient's condition seemed most hopeful. The patient then developed a very severe attack of mumps, and after another twenty-four hours an old appendicitis of five years' standing lit up. The patient was too ill to stand an operation, and died twenty-four hours later.

CONCLUSIONS.

A case of blackwater fever should never be moved if it can be avoided, and then only with the greatest care and for the shortest possible distance.

No case should be moved for at least fourteen days after the onset, however slight the case may be.

If possible a trained nurse should be obtained to see that the treatment is properly carried out.

If subcutaneous saline injections are given at once the urine will clear up more quickly.

I feel confident that quinine does not cause this disease, or even act as the fulminant, and think the reason this idea has been entertained is that, the premonitory symptoms being indistinguishable from malaria, quinine is taken, and the onset of blackwater is credited to it.

That blackwater fever is undoubtedly due to malignant tertian malaria has been conclusively proved by Professor J. G. Thomson of the London School of Tropical Medicine during his recent researches in Rhodesia.

The prevention of malaria, and therefore of blackwater, lies in the hands of the colonists, if they will only take the extremely valuable advice published for their benefit by Dr. A. M. Fleming, the medical director for Southern Rhodesia.

CONTACT CASES OF LEPROSY IN THE BRITISH ISLES.

BY

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At a recent meeting of the Dermatological Section of the Royal Society of Medicine a contact case of leprosy was shown in a boy, aged 14, who was born in England, had never been abroad, and who contracted the disease from his father, a leper, who died four years ago. This is the first case which has been exhibited at the Section in which the disease was contracted in this country.

Within the last few years I have seen three other similar cases, and in view of the impression, not only among laymen but even among certain members of the medical profession, that leprosy in this country is either non-contagious, or its contagiousity is so slight as to be negligible, I have considered it advisable to put these cases briefly on record.

CASE I.

A boy, aged 12, with nodular leprosy, was born in a southern county in Ireland, and was never out of Ireland until he was brought over to London for advice with regard to his disease. His father was a Russian, who had emigrated to Ireland, and who was in comparatively poor circumstances. When I saw the father he was suffering from nodular leprosy in an advanced stage, and had had the disease when he reached Ireland. As far as I could ascertain, the mother was not infected.

CASE II.

A boy, aged 15, was admitted to the St. Giles's Homes for British Lepers in 1920, suffering from nodular leprosy, with ulcerated lesions. He was born in a town in Lancashire and had never been out of England. His parents were healthy and came from British Guiana, bringing an elder son who had nodular leprosy. The boy was born a fortnight after their arrival in England. During his childhood he had been in close association with his infected brother, with whom he had slept for five years, and from whom he contracted the disease. He died in the St. Giles's Homes after being there seven months, two years after the death of his leper brother.

CASE III.

A married woman, born in Belgium, who suffered from leprosy of the mixed type, with both nodular and anaesthetic lesions. She was married in England and had lived here since then. She contracted the disease from her husband, who had nodular leprosy, and died in the St. Giles's Homes in 1916. Down to the time of her husband's admission to the Homes, she had taken no precautions to avoid infection, and had lived with him.

It is only comparatively of late years that the contagiousity of leprosy has been recognized, and it was not until 1897, at the first Leprosy Congress held in Berlin, that leprosy was definitely agreed to be a contagious disease, and resolutions were passed recommending compulsory notification and segregation. Since then numerous instances have been recorded of the transmission of leprosy by contact in countries where the disease prevails.

In Great Britain, largely owing to good hygienic conditions, leprosy has come to be a rarity, though at one time it was common, and the cases which exist here, with few exceptions, are exotic lepers who have come home from different parts of the empire where they had become infected with the disease.

The last place where a leper settlement of any importance existed in this country was in the Shetland Islands. There the disease seemed to have been rife, possibly because the natives were of Scandinavian origin, and were in more or less frequent communication with Norway and Iceland, where leprosy was prevalent. The value of segregation was early appreciated by the Shetlanders, and several centuries ago a leper colony was established in the island of Papa Stour, to which all suspected cases were sent. This is a remote island situated to the west of the Shetland group, and separated from the mainland by a stormy sound, which effectively isolates it in anything but fine weather. The last case of leprosy in Papa Stour—as far as I have been able to ascertain—died more than a century ago, and the

disease seems to have been exterminated there, probably on account of isolation and acquired immunity. At the present time the natives of the island are a hardy race, and show no trace of the scourge which prevailed amongst their ancestors, in spite of the fact that in winter their diet consists largely of dried, partially cured, and imperfectly cooked fish, a diet which was once believed to be responsible for the propagation of leprosy.

The experience in Papa Stour cannot but suggest that an island is the ideal site for a leper colony, and should it be found necessary to adopt measures of segregation here it might be possible to find some such island where a colony could be established.

The contact cases here recorded show the danger of belittling the contagiousity of the disease in this country, and of gainsaying the risk connected with intimate association with lepers; it may not be amiss to quote the resolutions of the second Leprosy Congress at Bergen in 1909—namely:

(1) "That leprosy is a disease that is contagious from man to man, whatever may be the method by which this contagion is effected."

(2) "That every country, in whatever latitude it is situated, is within the range of possible infection by leprosy, and may, therefore, usefully undertake measures to protect itself."

By means of a modified segregation, which was introduced in Norway in 1885, the number of cases was reduced in twenty years by about 50 per cent., and similar results have followed methods of isolation in Iceland and other countries. It does not seem inopportune at the moment to reconsider the advisability of putting our own house more in order, lest further cases should occur.

A CASE OF MYASTHENIA GRAVIS.

BY

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WORTHING.

THIS case, I think, merits publication because the disease is comparatively rare, and the case also presents certain uncommon features.

A man, aged 57, sought my advice in March, 1924, on account of choking attacks occurring towards the end of meals. He had one healthy child. His mother had died of diabetes and his father of rectal carcinoma. Apart from scarlet fever at the age of 8 his health had always been good.

History of Illness.

In 1913 he noticed that on tapping the extensor surface of either forearm or thigh the underlying muscle rose up in a wheal. His eyes happened to be examined at this time on account of errors of refraction; no ophthalmoplegia was then present. In 1914 he had a transient attack of abdominal pain and nausea accompanied by bilateral ptosis. This was attributed to his having eaten bad fish. Later in this year the muscles of the thigh and gluteal regions felt stiff and an unusual effort was required to rise from a stooping posture. Transitory diplopia also occurred about this time.

From then until May, 1923, the patient felt well, but the irritability of his muscles persisted. Since 1923 he had had frequent attacks of diplopia, which at first lasted from one to three days. From January, 1924, the condition became permanent, but was always less noticeable in the early morning. In March he noticed weakness of the jaw towards the end of a meal, and found that if he assisted it with his hand he was unable to close his mouth for half an hour afterwards. Being informed that he was suffering from a nervous breakdown associated with high blood pressure, he was advised to go to the seaside. His condition here became steadily worse, and marked ptosis in both eyes developed. For this he wore glasses, but found that the more he attempted to keep his eyes open the more complete was their closure. On the evening of March 27th fluid suddenly regurgitated through his nose whilst he was drinking, and a choking attack ensued.

Condition on Examination.

When giving me the above history a remarkable change passed over him. Whereas at the commencement of the recital his voice was clear and strong, yet, as he talked, a nasal twang developed to a pronounced degree. The forehead, previously wrinkled, became smooth, and bilateral ptosis developed coincidentally. From time to time he stopped to support his jaw, then with a further effort struggled to the completion of the sentence. The main points discovered on examination were:

1. Pronounced hippus when a light was thrown on the eye.
2. Normal reflexes and sensations.
3. Blood pressure 162 and 105.
4. All except the muscular system normal. A skiagram of the chest revealed no suggestion of thymic shadow.
5. Muscular system: Weak right external rectus. Ptosis, which became more pronounced if the patient made a sustained effort to look upwards. Inability to roll r's when speaking, and to whistle. The masseters were rapidly fatigued by faradism, recovered after rest, and were again fatigued after the passage of the current for one minute forty seconds. They were then capable of weak voluntary movement and of contraction to the galvanic current. The "nasal smile" or "myasthenic snarl" was exhibited in an attempt to smile after conversation. The upper lip was drawn upwards in the effort, movement at the corners of the mouth being absent. Movement of the soft palate was defective, with slight deviation to the right. No weakness of the limb muscles was demonstrable, but they all showed the remarkable excitability to direct mechanical stimulation already referred to. This was chiefly marked in the extensors, a tap of the finger producing a wheal-like swelling of the muscle, which persisted in one case for three minutes. The reaction was precisely such as one finds in Thomsen's disease, of which, however, there was no further sign.

Choking Attack.

On the evening of April 19th I received an urgent summons as the patient was choking. I found him in a prostrate condition, filled with fear of impending death. The respiration was both stertorous and stridulous, the soft palate flapping to and fro with each breath. At the time it was impossible to examine the condition of the vocal cords, but on the following day a definite weakness of abduction could be detected. Apparently, previous to this attack, he had been talking more than was his wont, but the actual onset was precipitated by his taking some bismuth. Feeling the powder cling to the back of his throat, he made violent but largely ineffectual efforts to clear it, thus rapidly exhausting the pharyngeal muscles. Rhonci and râles could be heard over both lungs at this time, and the patient complained of inability to "clear his chest." The condition improved during the following day, and in thirty-six hours he suddenly coughed up ten ounces of viscid mucoid sputum, expressing himself much relieved thereby.

Treatment.

Treatment was mainly directed towards securing as complete rest as possible for the affected muscles. Visitors were forbidden. All food was minced so as to reduce mastication to a minimum. Both eyes were covered by shades for the greater part of the day. The masseters and larynx were daily subjected to diathermy, which appeared to produce a definite amelioration of the condition. Strychnine was administered by the mouth.

Gradual improvement followed, so that when last seen in June there had been no further choking attacks; nasal intonation of the voice was very slight; diplopia was often absent during the greater part of the day. The patient was thus able to return home and resume light work.

Presumably the ultimate prognosis is thoroughly bad.

The main point of interest in this case is to be found in the muscular excitability to mechanical stimulation. Those muscles which were clearly affected by the myasthenia—for example, the masseters—failed to react abnormally to tapping. So far as I know this condition has not been previously described in myasthenia gravis, and I should be interested to hear opinions as to whether its occurrence here should perhaps be regarded rather as a coincidence than an unusual manifestation of the disease.