lence is very great the effect is a serous inflammation leading to spreading oedema.

Conclusions.

From these facts I draw the following conclusions:

1. If a disease is due to a specific causal agent it is not characterised by specific anatomical or functional lesions.
2. If a disease is characterised by specific lesions we must not expect to find a specific causal agent. From this it follows that from the characters, histological or macroscopical, of the lesions we cannot positively affirm that the disease is due to a specific causal agent. The only means by which we can affirm this is to find and isolate the causal agent itself.
3. Variations in the characters of the lesions in diseases due to specific causal agents depend not on the nature of the agent, but on the locality to which it is applied and on its relative virulence.

MYOKYMIA, OR PERSISTENT MUSCULAR QUIVERING.

By R. T. WILLIAMSON, M.D.LOND., F.R.C.P.,

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A SHORT time ago a patient came under my care at the Ancoats Hospital, suffering from a peculiar persistent quivering of the muscles of the limbs and trunk. The symptoms corresponded to those met with in the very rare affection to which Professor Schultze has given the name of "myokymie" (*μῦς*, a muscle, and *κύμα*, a wave), and Dr. Kny (who had previously reported two similar cases) that of "myoclonus fibrillaris multiplex." I have only been able to find records of eight cases in medical literature, but probably others have been reported.

The following are the notes of my case:

J. C., aged 21, clerk, was admitted as an in-patient at the Ancoats Hospital, August 29th, 1900, suffering from a peculiar persistent quivering of the muscles of the limbs, trunk, and face.

Previous History.

The left leg had been suddenly paralysed in infancy (probably owing to acute anterior poliomyelitis), and had remained useless. At the age of 16 years the left (paralysed) leg had been seriously injured, through an accident, and amputation had been necessary at the upper part of the left thigh.

Two years ago (at the age of 19) he had first noticed quivering of the muscles of the right leg. The onset was gradual, and the quivering spread in course of time to the muscles of the arms, trunk, face, and tongue. There had been no cessation or diminution of the symptom since its onset. After the amputation of the left leg he had been able to get about with the aid of a crutch, but recently he had found great difficulty in doing so, owing to the right leg frequently "giving way" through the muscular quivering.

He had always been a teetotaler, but had taken strong tea with each meal for many years. Three times a day for many years he had taken two cups (about one pint) of strong tea. He had only smoked about an ounce and a-half of tobacco weekly.

Condition on Examination.

The patient was fairly well nourished, and there was no definite wasting of any of the muscles of the arms, right leg, or trunk. There was a well-marked lateral curvature of the spine, which had gradually developed since the left leg had been amputated. The most striking symptom was a persistent rapid quivering of the muscles of the right leg, the arms, trunk, face, and tongue. Sometimes one small bundle of muscle fibres contracted rapidly, giving the appearance of fibrillary contraction; sometimes several bundles of muscle fibres contracted; at other times the whole muscle would contract rapidly. The symptom was best seen in the leg, and the quivering affected chiefly the thigh and calf muscles. At one moment a bundle or several bundles of muscle fibres would contract rapidly; directly afterwards a wave of contraction would pass over the whole muscle. This muscular quivering produced a very slight shaking or trembling of the whole leg, even when the patient was in bed. When the quivering was very violent, rapid and repeated slight flexion movements of the foot occurred at the ankle, owing to the quivering of the calf muscles. The arms were affected in a similar manner but not quite so markedly as the leg, the muscles of the shoulder, upper arm, and forearm being most affected, those of the hand very slightly. In the arms the quivering or contraction did not affect the whole of a muscle so frequent as in the leg. The rapid and repeated quivering of the muscles of the arms and leg continued more or less all day long; the muscles were affected in a most irregular manner, and the frequency of the quivering varied considerably.

The muscles of the chest and abdomen presented occasional fibrillary contractions. The muscles of the face and tongue, the masseters and neck muscles presented frequent fibrillary contractions. The eyes were not affected; there was no nystagmus. The muscular quivering and contractions in the limbs and trunk continued when the patient was at rest in bed, as well as when he was going about. When the arms were held out in front of the chest, the muscular quivering produced a slight tremulous movement of the whole limb resembling that so frequently

seen in Graves's disease; but there was not the flexion and extension movements of the fingers or wrist which are seen in paralysis agitans. The muscular quivering and slight tremor of the limbs were very little affected by voluntary movement; they were not diminished thereby; if any change occurred at all, they were slightly increased. The muscular quivering was increased by excitement, and when the patient was undergoing examination. The patient was able to write fairly well, and though the writing was slightly jerky, it was much better than in the various other forms of tremor, with the exception of paralysis agitans. He was also able to draw a straight line on paper fairly well.

The patient could perform all the movements of the arms and legs, and there was no localised wasting or paresis of muscles. There was no rigidity of the limbs. The knee-jerk was a little increased. There was no ankle clonus. On attempting to obtain ankle clonus occasionally a few jerks were obtained, but at other times this symptom was entirely absent. The plantar reflex was normal (flexor type). There was no loss of sensation to touch, pain, or temperature. The sensory cranial nerves were unaffected. There were no mental symptoms. The patient was not emotional or nervous. There was no affection of speech. The patient often complained of palpitation of the heart, and the pulse was often quick, 84 to 108 per minute. Examination of the heart, lungs, and abdomen revealed no evidence of organic disease. The patient complained of frequent sweating. There was no exophthalmos, no Graefe's symptom, and no thyroid enlargement. There was a little nausea after food; the bowels were regular.

The urine was often pale, at other times of normal colour. The quantity was increased; it was measured for sixteen days, and found to vary in amount from 64 to 124 ounces daily. The specific gravity varied considerably; when the urine was pale it was often low—1005 to 1008; at other times it was 1012 to 1015. Albumen and sugar were absent. The reaction was sometimes alkaline, sometimes acid. The temperature was normal. Insomnia was a frequent symptom.

As the patient had taken strong tea at every meal for many years I allowed him no fluids except milk and water for two weeks, but without any result as regards his symptoms. During the four weeks he was in the hospital there was no definite change; some days the muscular quivering was a little less, but the diminution was never permanent. The muscular quivering continued more or less all day long, just as it had done for two years.

The symptoms in the case just recorded corresponded to those in the affection to which the name of "myokymia" has been given. Similar cases have been recorded by Kny, Schultze, Hoffmann, Biancone, Buber, Karcher, and Mayer. In the 8 cases of which I have been able to find records in medical literature, the ages were from 21 to 71. All the patients were males. Five of the eight were peasants or country labourers. One patient was a painter, another a lead solderer; both of these patients suffered also from symptoms of lead poisoning. One patient suffered from chronic double sciatica. In all of the 8 cases the legs were most affected. In 2 cases the legs only were affected; in 1 the legs and muscles of the back; in 2 the arms and legs; in 1 the legs and arms but not the hands; in 1 almost all the muscles of the body; in 1 almost all the muscles except those of the face and neck. [In the case which I have just reported, the muscles of the limbs and trunk, as well as those of the neck and face, were affected.] In 4 cases recovery occurred in the course of a few months; in 1 case there was distinct improvement. The treatment, in the cases in which recovery occurred, was by warm baths and galvanism (Kny), by bipolar faradic baths (Kny), by rest in bed and warm baths (Schultze). Buber's case improved distinctly under treatment with the galvanic current and warm baths. Biancone recommends complete rest in bed, warm baths, and sodium salicylate.

The cause and pathology of this peculiar affection are at present unknown.

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A BRIEF ACCOUNT OF THE RECENT EPIDEMIC OF CHOLERA IN KASHMIR.

By ERNEST F. NEVE, M.D., F.R.C.S.E.,
Kashmir.

ONCE more Kashmir has been visited by a sharp epidemic of cholera. Thus, in 13 years there have been three invasions. In 1888 there were probably 18,000 attacks, and a total mortality of nearly 10,000; in 1892 the official returns showed 16,845 cases, with 11,712 deaths; owing to the concealment then extensively practised by the people, it is probable that

there may have been hundreds of unregistered cases. This year there have been about 7,600 attacks and 4,256 deaths.

Kashmir, with its protecting belt of mountains, lies so far from the plains of India, and has so cold a winter, that cholera is not endemic. Indeed, a special train of circumstances is almost required if the valley is to become infected. In 1888 the hundreds of camp followers of the Maharajah brought it in. I remember, on the day of his arrival, meeting four men, on the bank of the river, carrying into the city an emaciated corpse-like form, a man in the collapse stage of cholera. A few days later the disease broke out in all directions. Since then improved roads and increased facilities for travel have tended to increase the risk of periodic outbreaks. Even now the long mountain roads, crossing passes and rivers at intervals, admit of the application of quarantine. And yet, even with such favourable conditions, this has nevertheless failed absolutely.

Cholera had been prevalent in the Punjab during the summer. A traveller from the Lyallpur District was taken ill and died of cholera on the Banihal road, one of the shortest routes to Kashmir, on July 18th. About 4,000 coolies were working on a new road close by. On July 21st the disease broke out amongst these, and 200 cases occurred, of which 176 died. Probably a large number of the milder cases were concealed. The survivors fled, many of them to Kashmir, carrying, no doubt, the infection with them. Hindu pilgrims, too, on their way to the sacred cave of Amarnath, at the north-east of Kashmir, traversing the infected districts, also suffered, and acted as carriers.

After a time of anxious expectation it was announced that cases of cholera were beginning to appear at the east end of the valley, the point at which the Banihal road enters. By September 3rd, 470 cases had occurred, 35 villages were infected, and, worse still, 18 cases had appeared in the city of Srinagar, which has a population of 125,000. The disease continued to spread, and on September 12th there were 93 new cases in Srinagar, and 143 in the district around. After this there was a gradual decline in the city. The maximum in the surrounding district was reached on September 28th, when the record of new cases was 250. From this time there was a steady improvement. On October 24th there were only 40 fresh attacks in the whole valley.

Compared with the last two epidemics this one has been comparatively small. The reasons are not far to seek: (1) A good supply of pipe water has been brought to the city; (2) in the early autumn the nights begin to get quite cold owing to the altitude; (3) a great deal has been effected in the way of sanitation during the past few years.

The introduction of a supply of pure water has saved hundreds of lives in Srinagar. In spite of this, however, in some of the more crowded and insanitary quarters, the disease appeared to obtain at any rate a temporary footing.

The mortality as a whole was very constant, being about 56 per cent. The records of some districts are much higher. This may be due partly to the intensity of the poison, but is more probably owing to the concealment of non-fatal cases. As time went on there was a tendency for the attacks to assume a milder character. The cold no doubt checked the growth of the bacilli outside the body. Rain appeared to favour the spread of the disease. Continuous rain, however, which ushered in colder weather, proved very beneficial.

Prophylactic treatment with sulphuric acid is, I believe, of value in averting the disease. At the Mission Hospital all cases of chronic diarrhoea were treated in this way. Only one was attacked, and in spite of debility (due to traumatic discharging empyema, the result of a severe gunshot wound, for which he was under treatment), he made an excellent recovery.

The immense importance of early treatment with some preparation of opium cannot be too strongly insisted upon; 70 per cent. of those treated by us in this way recovered. The proportion would have been larger, but our returns included bad cases to which we were called too late. In two cases on the second day I found an appearance of spores in the bacilli after prolonged staining with carbol-fuchsin, a point of some interest, as their existence is denied by high authorities.

During the epidemic excellent work was done by Major Roberts, Acting Residency Surgeon, and by Dr. A. Mitra, the Chief Medical Officer to the Kashmir State.

QUININE HÆMOGLOBINURIA.

By A. G. WELSFORD, M.D., F.R.C.S.ENG.,
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WITH regard to the theory that the so-called blackwater fever of the tropics is a peculiar condition caused by the administration of quinine in malarious patients, few (if any) practitioners who are experienced in tropical practice believe that this is a common cause, because while they are in the habit of prescribing quinine in large doses for all varieties of the malarial fevers, they do not practically find that blackwater fever results, and they also know that blackwater fever is a strictly localised disease, and that there are some malarious districts Africa which are free from it.

It is a fact that quinine can in certain very rare cases cause hæmoglobinuria, but so rare are they that there are few observations on record. The following case is interesting as being one of these rare cases.

CASE I.

A.B., a white prospector, was admitted into the Beira Railway Hospital in June, 1899. He had been in the country nine years, and had just returned from a trip into the interior, which had lasted nine months. He was suffering from a high degree of malarial cachexia. He had had frequent slight attacks of blackwater fever during the previous three years. He rarely, if ever, took quinine. On admission he objected to take quinine, alleging that a single dose invariably made his urine black, but he eventually consented to take two 10-grain doses, which were administered at 8 A.M. and 2 P.M. At 6 P.M. the patient had a rigor; the temperature, which before was normal, rose to 105° F. with vomiting and pain. At 7 P.M. he passed 10 ounces of black urine. He improved during the night, and next morning his urine was clear, but still contained a little albumen.

Before taking the quinine the patient had been up and about, and there was therefore a possibility of the hæmoglobinuria having been due to chill. To obviate this source of error the patient, who took a most intelligent interest in his case, after his temperature had remained normal for a period of seven days was put to bed for two days. On the third day two 10-grain doses of quinine were given with the same result; the temperature rose in the evening with rigor, vomiting, and pain, and 12 ounces of black urine were passed. The following morning the urine was clear, and in two days there was no albumen.

CASE II.

A more doubtful case of quinine hæmoglobinuria occurred in the case of a man who was admitted with a somewhat severe attack of blackwater fever. This was treated without quinine and the patient recovered. Three weeks later his temperature rose to 100° F. and 10 grs. of quinine were ordered three times a day. Two days later his temperature suddenly rose to 105° F. and his urine became black and continued so for two days. As in this case the symptoms were well marked; this attack may have been a relapse.

The exact relationship between blackwater fever and malaria is not yet made out, but the symptoms of blackwater fever are so definite that the fever can be readily distinguished from the transient hæmoglobinuria which in the first-mentioned case followed the administration of quinine.

Hæmoglobinuria may be produced by several poisons, among which are potassium chlorate, carbolic acid, and arseniuretted hydrogen. It is in this category that quinine hæmoglobinuria must be placed.

Blackwater fever, if not due to a specific organism, is due to the toxin of malaria, and falls in line with the hæmoglobinuria sometimes produced by the toxins of syphilis, yellow fever, enteric, and scarlet fevers.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

A FATAL CASE OF POISONING WITH ZINC SULPHATE: NECROPSY.

I WAS called to see A. C., a widow, aged 53 years, at about 5 P.M. on July 5th, 1900. She was said to have swallowed a large packet of sulphate of zinc at some time between 1 and 3 P.M. She was suffering from severe pain in the stomach and bowels, paleness of the countenance, coldness of the limbs, irregular pulse, and cold sweats, with purging, but had only vomited about a teaspoonful. I administered a large amount of carbonate of soda diluted freely with tepid water. Copious vomiting of a white fluid ensued (showing presence of carbonate of zinc). After which I directed that she should be frequently given white of egg and milk, and later on, when signs of collapse appeared, whisky with the latter. I injected