of the tongue was noticed the day after the bite.

Taken in connection with Cunningham's experiments of the action of the viperine poisons, the severity of the blood changes in this case is very interesting.

1. The peculiar brick-dust colour of the blood from the wound near the affected part.

2. The hæmaturia, hæmoptysis, and ecchymosis of the tongue.

3. The non-coagulability of the blood for more than forty-eight hours after the bite was inflicted, shown by the continual bleeding from the wound on the removal of pressure.

The absence of any signs of the action of the poison on the central nervous system is also notable.

If a ligature is applied at once, as in this case, I think there should be more chance of averting a fatal result, if a free incision is made round the wound, and hæmorrhage encouraged until the part below the ligature is practically bloodless than in mere scarification of the wound and subsequent suction.

## A CASE OF POST HEMIPLEGIC ATHETOSIS.

UNDER THE CARE OF G. H. A. HARRIS,
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*1st Physician, Medical College Hospital, Calcutta.*

REPORTED BY U. N. BRAHMACHARI, M.A., M.B.,
*House Physician.*

THE patient named Khetter, *æt.* 25, a Hindu male, belonging to the railway mail-service, states that on 17th July 1899, twelve hours after a fatiguing swim, he began to suffer from giddiness and weakness in the left side of his body. Two or three days later he was admitted into Lt.-Col. Harris's ward for treatment of hemiplegia with the following symptoms :—

(1) Left facial palsy.

(2) Complete motor paralysis of the left upper and lower extremities without any sensory disturbance.

(3.) Exaggeration of the left knee-jerk. There was no history of syphilis but history of occasional transient fits of unconsciousness for about a year. The patient left hospital almost recovered. One night about a month after leaving hospital, he was awakened from sleep by an involuntary flexion of the left middle finger which, since then has more or less persisted in a series of mobile spasmodic movements. This condition gradually increased and affected the other fingers, interfering very materially with his work, and it was on this account that he again sought relief on 13th December 1899. The symptoms on admission were :

(1) Slight disorder of sensation in the fingers of the left hand. The following was the table of tactile sensibility of both the upper estremities as measured with the æsthesiometer.

| | *Right Side* | *Left Side.* |
|---|---|---|
| (*a*) Dorsum of 1st phalanges of fingers. | $\frac{7}{12}''$ | 2″ in middle finger.<br>$1\frac{3}{8}''$ in index finger.<br>1″ in the thumb. |
| (*b*) Palm of hand ... | $\frac{5}{12}''$ | $1\frac{1}{2}''$ |
| (*c*) Back of hand ... | $1\frac{1}{6}''$ | 2″ |
| (*d*) Forearm | $1\frac{1}{2}''$ | $1\frac{1}{2}''$ |
| (*e*) Dorsum of feet ... | $1\frac{1}{2}''$ | $1\frac{1}{2}''$ |

(2) Strength of the grasp of the hands as measured with the dynamometer:

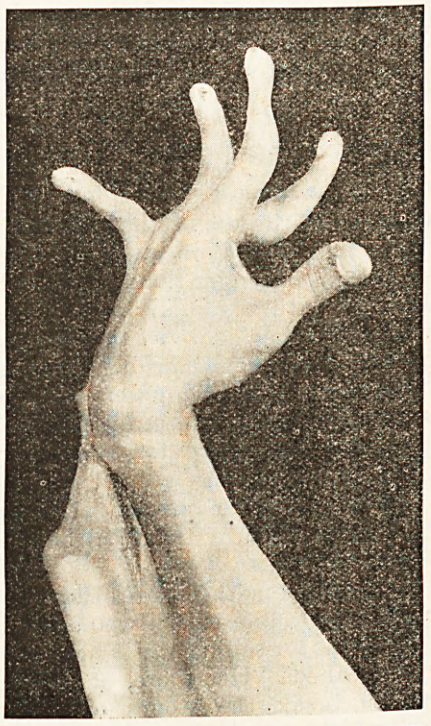
(*a*) Left hand ... 20 lbs.
(*b*) Right hand .. 70 lbs.

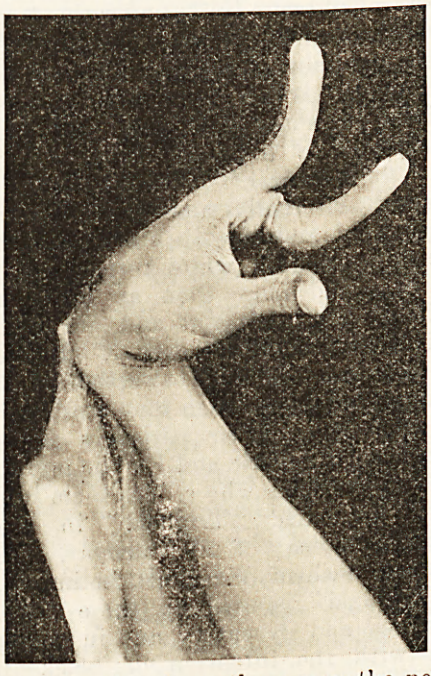
(3) Marked increase of the left knee-jerk.

(4) Athetoid movements in the fingers of the left hand. There is a slow mobile spasm affecting mostly the interossei and the lumbricales of the left hand. The amount of spasm in these muscles varies from time to time and thus slow, successive, incoordinate movements of flexion, extension, pronation and supination of the fingers are produced. The movements have been compared (Gowers*) to those of the tentacles of the cuttlefish. During the execution of some of the movements the interphalangeal joints pass into a state of subluxation. Sometimes the distal phalanges are extended, shewing that the long extensor also partakes of the spasm. Sometimes the 1st phalangeal joint of the middle finger is strongly flexed with the thumb closely pressing against it, and sometimes there is flexion of the last phalanges of one or more fingers, showing that the long flexors of the fingers also partake of the spasm. This implication of the long flexors is noteworthy, as Gowers says this never takes place. Rarely the fingers assume a position which has been compared (Charcot†) to that of the contracture of the type of extension in chronic articular rheumatism. Occasionally, there is a simulation of some of the above movements by the fingers of the right hand, though they are hardly so marked as to justify the case being regarded as one of double athetosis. (Two of the positions assumed by the fingers are given in the accompanying photographs.) Rarely, there is an involuntary hyperextension of the left big toe especially during walking. The movements disappear during sleep and can be controlled and limited only for a short time by position and extraordinary effort of the will. The phenomena, as pointed out by Von Zemissen, seem to have partially the character of associated movements,‡ for while the fingers move, the arm becomes rigid and hard.

* Gowers' Diseases of the Nervous System
† Charcot Le'on de Mardi 1887.
‡ Charcot's Diseases of the Nervous System (New Sydenham Society 1888).

The spasm disappears when the wrist or the fingers are rigidly flexed, but after a time it starts again, sometime sslowly and sometimes suddenly. The spasm markedly interferes with a voluntary act, so that the patient has to wait till it passes off. There is not the slightest contracture in any of the fingers.





(5) Besides the above there were the negative symptoms of absence of motor paralysis of any cranial or spinal nerves and of wasting of any groups of muscles.

*Remarks.*—The diagnosis of a case of athetosis as a post-hemiplegic state is simple when one is alive to the character and significance of its symptoms. It might possibly be thought due to disseminated sclerosis and paralysis agitans; to paramyoclossus multiplex when it attacks the muscles of the arm and face which it very rarely does after hemiplegia; to convulsive tic, chorea spastica, tetany and localized chorea of Sydenham; in none of these conditions, however, are the movements truly athetoid. The possibility of hysterical simulation of the movements should be borne in mind. The purposeless nature of the movements and the absence of hysterical stigmata should determine the diagnosis. The condition, as Drewry* says, is also impossible to be imitated even by the most skilful malingerer. Some authors, including among others Hammond and Gray,† seem to make an unfortunate confusion by including post-hemiplegic athetosis under post-hemiplegic chorea; but the latter, properly speaking, refers to chorea-like movements sometimes occurring in the affected limbs after hemiplegia, and should have nothing to do with the slow and peculiarly characteristic movements of athetosis.

The seat and nature of the lesion in athetosis have not as yet been satisfactorily worked out. Hammond, who first described the condition, supposed the seat of the affection to be in the intracranial ganglia or in the upper regions of the spinal ganglia. He also states that one probable seat of lesion is the corpus striatum. Von Zemissen supposed it to be due to changes, partially circumscribed, of the centres of motor innervation; while Hitzig supposed it to be due to irritation of these centres.‡ Beevor considers it to be due to lesions in the motor regions of the cortex,§ and in favour of this view might be cited his case in the *British Medical Journal* for 1890. Osler, however, says that in cases of athetosis occurring in adults, the lesion is not in the cortex. Gowers‖ says that a distinction must be made between the cases occurring in adults and those in childhood. He thinks that in the former the disease is situated in or outside the optic thalamus, or in some cases in the posterior of the internal capsule. In Gould's Yearbook of Medicine and Surgery for 1899, it is stated that Von Kunu collected fourteen cases from literature, in seven of which there was localized disease of the corpus striatum, in four of the optic thalamus, in two of the pores and in one a softening involving the optic thalamus and corpus striatum.

As regards the nature of the lesion it has been supposed to follow mostly cerebral softening causing "extensive slight damage." James

* Zemissen's Cyclopædia of the Practice of Medicine.
† Beevor's Diseases of the Nervous System.
‡ Gould and Pyle's Anomalies and Curiosities of Medicine.
§ Hammond's Diseases of the Nervous System (1873.)
‖ Osler's Medicine.

Taylor in his article on cerebral palsies of children states that cases of paralysis followed by athetosis in adults are mostly the results of accident.* In the present case no history of accident can be discovered. The history of hemiplegia following over-exertion, might be suggestive of embolism, in which case, however, the paralysis would more immediately follow the over-exertion; nor are there at present any signs of heart lesion which might be suggestive of a possible source of emboli. On the other hand, the excited action of the heart and the increase in blood pressure consequent upon the over-exertion in swimming might have led to rupture of some diseased vessels in the motor tract of the brain. The diseased condition of the vessels might have been the result of syphilis of which the patient, however, denies any history. Besides, extreme headache which forms one of the characteristics of syphilitic disease of the vessels of the brain is absent in the present case. It might be that the association of the over-exertion with the hemiplegia was an accidental one, and that the occasional transient fits of unconsciousness might represent the minor form of epilepsy (petit mal), with which the athetosis was connected, as many of the recorded cases have been.

The present case is of interest as it is a case of athetosis in an adult unassociated with hemianæsthesia and vasomotor change, and following an attack of marked hemiplegia after it was completely recovered from. Another point of interest in the present case is the fact that the long flexor and extensor muscles of the fingers partook of the spasm to some extent, which Drewry says are rarely affected; while Gowers says, as already mentioned, the long flexors are never affected.

I am indebted to Lieutenant-Colonel G. H. A. Harris for permission to publish this case from his wards.

* Allbutt's System of Medicine, Vol. VII.

## "BAHEDA" POISONING.

By BABU BHONDOO LAL,

*Civil Hospital Assistant.*

A boy, named Phakiria Gond, nine years of age, ate fresh kernels of "bahédá" seeds in excess on the afternoon of 4th February 1900, and was brought by his mother to Múl Branch Dispensary, in the Chanda District, on the forenoon of 5th February 1900 for treatment.

The following abnormal symptoms were observed in him at that time, such as—Countenance anxious, eyes shut, pupils normal, unconsciousness, retardation and slowness of respiration, pulse feeble, head and neck hung on shoulder, body warm, no answer on questions, occasional sighs and stoppage of breath, fore-arms flexed on arms, rigidity of the upper extremities; lower extremities were stretched and rigid; passed one normal stool at his house as his mother said. There was no vomiting—languor. These threatening symptoms did not allow me to count the beats of the pulse and to take temperature of his body.

The patient was immediately placed under artificial respiration, and the following mixture was given:—

℞.

| | | |
|---|---|---|
| Spirit. Ammon. Aromat. | ... | m xv |
| Aqua ... ... | .. | ad ℥i |

Mix. Every one hour.

After the third dose of the mixture he opened his eyes and turned on his other side, and his pulse became stronger than before. He began to breathe naturally. His mother then took him away to her house as this is an out-door dispensary, and next morning, that is, on the 6th February 1900, the patient was better as I sent the Kotwar to inform his mother to bring him to this dispensary for further treatment, but she sent word she will get him treated at his house by native quacks. The boy might have been treated by them but he died on the same date at 4 P.M.

The patient's mother states that she went in company with him to the wood and began to cut dry grass for selling while her son, the sick, saw a "bahédá" tree there, sat under it, and began to break the stones of the "bahédá" fruits, which were lying under the tree and to eat their kernels as much as he liked which caused giddiness at the very time as she said.

"Bahédá" tree is akin to the tree of Bassia-latifolia in shape and size, and is common throughout all parts of India, the fruits of which are conical. Their pericarps are ash-coloured externally and green internally, in which hard and smooth stones are enveloped. The stones contain white kernels. Some fruits are oval and more than an inch long. The pericarps are astringent and are used by the natives as a colouring matter, but the kernels are nauseous although sweetish in taste and produce giddiness when they are fresh and not so when dry. Their pericarps are generally mixed with Belleric and Emblic myrobalans by native physicians who reduce them into powder which is called "Triphalá" in the Indian Materia Medica of all authors, such as:—Charack, Sushrut, Wagchutt, Lolimbráj, etc.

"Triphalá" is used by these physicians in diarrhœa and dysentery, and in people of grey hair with the intention of blackening hair-follicles so that they may not grow grey again.

The "Bahédá" poisoning is not mentioned in any of our works, hence it seems to me a new poison which ought to be published in the *Indian Medical Gazette* for the information of the public.