The differential diagnosis of a case of epileptiform convulsions / by Byrom Bramwell.

THE DIFFERENTIAL DIAGNOSIS OF A CASE OF EPILEPTIFORM CONVULSIONS.

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(*Being a Lecture delivered in the Extra-Academical School of Medicine, Edinburgh, during the Summer Session, 1880.*)

GENTLEMEN,—Having decided that the convulsions are general (*i.e.*, bilateral) and epileptiform in character, we have now to determine:—

I. If the attack is genuine.

II. If genuine, what is its cause.

The Differential Diagnosis of Genuine and Sham Epilepsy.

Impostors frequently feign epileptic fits in order to get admission to hospital, to obtain alms, or to give a confederate a convenient opportunity of abstracting the purses and watches of the sympathising crowd that gathers round them.

If the impostor is a clever artist, and well coached, the detection of the deceit is often very difficult; indeed, the detection of feigned or simulated disease is always difficult, and requires both acumen and a profound knowledge of the true affection.

The points of differential diagnosis are the following:—

Sham epilepsy is to be *suspected* when—

1. The fit occurs in a suitable place, and at a convenient time to collect a crowd of sympathising observers. In hospital practice a favourite time is the hour of visit of the physician or house surgeon. The occurrence of fits at such, and at such times only, is very suggestive of the feigned affection.

2. The patient receives no injury during the attack. Impostors do not hurt themselves as they fall; do not bite their tongues; do not soil their clothes by evacuating the contents of the bladder or rectum.*

If, in addition to these suspicious circumstances, any *positive* facts, inconsistent with the genuine affection, are detected, a positive diagnosis may then, *but then only*, be ventured upon.

* Genuine epileptics may not hurt themselves, and may not discharge urine and fæces. These negative facts, therefore, are not conclusive.

Attention is to be directed to:—

1. *The condition of the reflexes, especially of the pupil reflex.*—In genuine epilepsy the reflexes are abolished. The pupil, at the commencement of the fit, is widely dilated and insensible to light.* During the stage of tonic convulsions, the pupil is still dilated, but alternate contractions and dilatations occur. Snuff thrown up the nostrils produces no result. In sham epilepsy the pupil responds to light, and dilates under any powerful and unexpected sensory impression, such as the prick of a pin. Snuff injected up the nostril produces violent sneezing, and usually cuts short the attack.

2. *The character of the spasms.*—Sham fits are often exaggerated. Impostors may fail to hit off exactly the character of true spasms. They sometimes err in making the spasms of equal intensity on the two sides of the body. The duration of the attack is often unduly prolonged: &c.

3. *The state of consciousness.*—It is often difficult to detect feigned unconsciousness, but the acute observer will sometimes perceive that the supposed patient takes occasional sly glances at his surroundings. Other circumstances, too, may suggest that he knows what is going on. Where the Faradaic brush is available, as in hospital practice, it is an efficacious means of determining whether consciousness is really lost, and of arresting a sham fit.

4. *The initial pallor* is not observed in sham epilepsy. This is not, however, a point of much practical value, for it is sometimes absent, and may easily escape attention in the genuine disease.

5. *The presence of marked cyanosis* during the attack is in favour of the genuine disease. The fact that the skin is cold and (towards the end of the attack) bathed in a clammy perspiration is of value. In sham epilepsy the skin is often moist, but it is warm.

6. *The escape of frothy saliva, unless it be bloody*, is of no value. Impostors know full well the marked impression produced on the lay mind when a patient lies convulsed and foaming at the mouth. They take care, therefore, to secrete a piece of soap in the cheek, and to manufacture a copious supply of frothy saliva during the attack. The escape of *bloody* saliva is in favour of the genuine disease, for, as I have already mentioned, impostors do not care to bite their tongues during the attack.

7. *The condition on recovery.*—In the majority of cases of genuine epilepsy the patient is stupid and bewildered after

* In exceptional cases the pupil is not dilated.

the attack. Genuine epileptics, too, often feel ashamed of having been observed in a fit. This is not, of course, the case in the feigned disease.

8. *The facial appearance of the patient.*—Confirmed epileptics often have a characteristic facial appearance which is difficult to describe. The expression is dull and heavy. Their intellectual powers are generally impaired. When they are undergoing an active course of treatment, the face may be covered with the bromide rash.

9. *The effect of proposed energetic treatment.*—In sham cases, the proposed removal to the police station, the threatened application of the actual cautery, &c., are very efficacious means of arresting the attack, and of making a diagnosis.

Having decided that the spasms are genuine, we have next to ascertain their cause; for epileptiform convulsions may be due to a great number of different conditions, amongst which the following are the chief:—

1. Idiopathic (true or genuine) epilepsy.
2. A local "coarse" cerebral lesion; including under that term
   - (*a*) Cerebral tumour.
   - (*b*) Cerebral abscess.
   - (*c*) Local meningitis of the convexity.
3. General meningitis.
4. Cerebral hæmorrhage (central and meningeal.)
5. Arrest of the blood supply to the brain, as a whole, or of the motor area in particular.
   - (*a*) General bleeding.
   - (*b*) Embolism or thrombosis.
6. Reflex irritation, as worms, teething, &c.
7. The presence of certain poisons in the blood and brain; some of the more important being:—
   - (*a*) Retained urinary products.
   - (*b*) Alcohol.
   - (*c*) Lead.
   - (*d*) Malarial poison.
8. The onset of the exanthemata and other febrile conditions in children.

The diagnosis of the cause of an attack of epileptiform convulsions must obviously, in many cases, be a difficult and tedious operation.

The following are the steps in the enquiry which I am in the habit of following out in practice:—

PRACTICAL STEP NO. 1.

Ascertain from the patient's friends, or from himself, as soon as he becomes sufficiently conscious, whether he has had previous attacks, and for how long.

Positive information on this point enables us at once to divide cases of epileptiform convulsions into two great groups.

A. *Cases in which there have been repeated attacks of a similar character, which have not been attended with any serious disturbance of the general health.*

In such cases, the cause of the convulsions is, in all probability, the disease idiopathic or genuine epilepsy. Exceptions occasionally occur; in some cases of organic brain disease, especially syphilitic tumours pressing upon the motor area of the cortex, the grey matter gets into the habit of discharging, and may continue to discharge (*i.e.*, the patient may continue to take fits) for a long time after the original source of irritation (the syphilitic gumma) is removed. In such cases there may be no symptoms of coarse lesion, when the patient comes under observation; and, unless the history of the case, as to previous headache, vomiting, &c.; the age at which the fits commenced; and the exact character of the spasms, be carefully inquired into, the case may be thought to be one of the idiopathic or so-called genuine disease.*

B. *Cases in which there is no history of a previous attack* (either because this is the first fit, or because the patient cannot [from coma, stupidity, or other cause] give any information on the point) *or in which the fits have only commenced of late.*

The convulsions may in such cases be due to any of the causes mentioned above. (See page 182.)

In pursuing the inquiry the following circumstances must be taken into consideration:—

1. *The age of the patient.*—Some causes are undoubtedly more active at one period of life than at another. There are, however, so far as I am aware, no reliable statistics on the point.

In the following tables I have attempted to give my own impressions as to the relative frequency of the different causes at different ages, presuming in all cases that the first attack

* In some cases of plumbism the convulsions continue for some time after the other indications of lead poisoning (such as the blue line, colic, &c.), have disappeared; but in all the cases of this description, which have come under my notice, there has been anæmia, and some affection of the general health. In some cases too of alcoholic epilepsy, the convulsions continue after the other indications of alcohol have disappeared.

has occurred *at*, and not prior to, the age mentioned in the table. I am well aware that impressions of this sort are very often fallacious; but it is with the view of obtaining the opinion of more experienced observers than myself that I have ventured to draw up such a list.

Table I.

Supposed order of frequency of the different forms of epileptiform convulsions in children up to 10 years of age.

1. Reflex irritation, including many cases of rickets.
2. Meningeal inflammation.
3. Cerebral exhaustion and anæmia from diarrhœa.
4. Onset of the exanthemata and other febrile affections.
5. Idiopathic, including many of the so-called cases of infantile eclampsia.
6. Uræmia.
7. Cerebral tumours.
8. Meningeal hæmorrhage.

Table II.

Supposed order of frequency of the different forms of epileptiform convulsions, the first fit appearing between ages of 10 and 20.

1. Idiopathic.
2. Tubercular and other forms of meningitis.
3. Cerebral tumours (especially scrofulous growths.)
4. Uræmia.
5. Reflex.
6. Cerebral softening, the result of embolism.
7. Lead.

Table III.

Supposed order of frequency of the different forms of epileptiform convulsions, the first fit appearing between the ages of 30 and 45.

1. Intracranial tumours and other coarse cerebral lesions.
2. Basilar and other forms of meningitis.
3. Uræmia.
4. Idiopathic epilepsy.
5. Softening from thrombosis and embolism.
6. Alcohol, lead, &c.
7. General hæmorrhage.
8. Reflex irritation.
9. Cerebral hæmorrhage.

TABLE IV.

Supposed order of frequency of the different forms of epileptiform convulsions, the first fit appearing after the age of 45.

1. Intracranial tumours and other coarse cerebral lesions.
2. Uræmia.
3. Thrombosis and embolism.
4. Alcohol, lead, &c.
5. Hæmorrhagic apoplexy.
6. Idiopathic epilepsy.
7. General hæmorrhage.
8. Reflex irritation.

2. *The character of the attack itself.*—The most important points being:—

(*a*) *The mode of commencement,* whether bilaterally or locally, the latter being very suggestive of a "coarse" cerebral lesion.

(*b*) *The state of consciousness,* whether lost at the commencement of the attack or not. In epileptiform convulsions due to a "coarse" cerebral lesion there is usually no loss of consciousness at the commencement; and, indeed, consciousness may be retained throughout the stage of bilateral spasms. In other bilateral epileptiform convulsions, consciousness is lost.*

(*c*) *The duration of the subsequent coma* may give some information. In cases of cerebral hæmorrhage, for example, the spasms (when they occur) are followed by prolonged and deep coma. In cases of uræmia and lead encephalopathy, the patient usually remains in a comatose or semi-comatose condition for a considerable time. In ordinary (genuine) epilepsy, consciousness is, as a rule, soon regained, though the patient may be stupid and confused after the attack.

(*d*) *The recurrence of the spasms.*—In uræmia, plumbism, and in some cases of cerebral tumour, there are repeated fits at short intervals. In some cases of genuine epilepsy, the same fact is observed.

3rd, *and chiefly, The examination of the patient after the spasms have passed off.*—In this examination, attention is to be directed to the following points:—

(1.) *The present condition of the patient.*

(*a*) The presence of any symptoms or physical signs indi-

*This is the general rule; exceptions occasionally, though rarely, occur. Thus, in some cases of convulsions due to cerebral softening, embolism, and thrombosis, there is no loss of consciousness.

cative of a lesion which might cause the convulsions, especially the condition of the optic discs, heart, arteries, and kidneys.

(*b*) The presence of any obvious exciting cause, such as teething, worms, &c.

(2.) *The previous history of the patient.*

(*a*) As to the occurrence of previous attacks of spasms; their number; frequency; the age of the patient at the time of the first fit; the supposed cause of the attack, &c., &c.

(*b*) As to other symptoms and diseases, especially such as would result from nervous disease.

(*c*) As to his habits, surroundings, and mode of life.

(3.) *The family history* and hereditary tendencies, especially any tendency to nervous disorders.

(4.) *The progress of the case and the effects of treatment.*

In practice, I find the best method of following up the enquiry is:—

Practical Step No. 2.

Ascertain if there are any positive objective symptoms or physical signs of any of the causes of epileptiform convulsions mentioned on page 182.

In this way *Group B* is subdivided into two minor groups.

C.—*Cases of epileptiform convulsions due to lesions which are indicated by positive objective symptoms and physical signs.*

D.—*Cases of epileptiform convulsions in which the cause of the convulsions is not indicated by objective symptoms and physical signs.*

Class C.—Positive evidence in the form of objective symptoms and physical signs is generally present in the case of epileptiform convulsions, which result from:—

1. "Coarse" cerebral lesions, including under that term, tumour; abscess; and meningitis, limited to the convexity.
2. Basilar meningitis.
3. Cerebral hæmorrhage.
4. Embolism and thrombosis.
5. Lead.
6. Uræmia.
7. General hæmorrhage.

I shall therefore, in the next place, proceed to discuss the chief facts (negative and positive) in favour of each of these conditions.

Epileptiform convulsions resulting from a local "*coarse*"

cerebral lesion (including under that term tumours, chronic abscess, meningitis of the convexity).

A local coarse cerebral lesion is a frequent cause of epileptiform convulsions. The spasms are often limited in distribution. In the case, we are at present considering, (*general* epileptiform spasms due to a coarse lesion), the spasms usually commence locally, extend in a definite order, and finally become bilateral. Consciousness is retained in the earlier stages, and sometimes throughout the attack. There is often subsequent temporary (epileptiform) paralysis. The spasms tend to recur. In some cases the condition termed "epilepticism" is observed.

The *positive* facts in favour of a local coarse cerebral lesion are :—

1. *Headache;* generally severe; often paroxysmal and intermittent; sometimes localised to one particular part of the head; and in such cases frequently attended with local tenderness on skull percussion.

2. *Vomiting* of a purposeless character; usually unattended by any feeling of nausea; generally accompanied by constipation; apt to occur on any sudden movement or alteration in the position of the patient (disturbance of the cerebral circulation). Cerebral vomiting often, therefore, occurs when the patient first rises from bed in the morning. It is often accompanied by marked vertigo. In cerebral vomiting the tongue may be quite clean, though it is not unfrequently furred.

3. *Double optic neuritis and optic atrophy.*—These conditions are present in the great majority of cases of cerebral tumour, irrespective of their seat; in some cases of chronic abscess; but not, as a rule, in cases of meningitis, limited to the convexity.

4. *The presence of associated lesions.*—The presence of external tumours (cancerous, sarcomatous, &c.); of enlarged glands; of associated diseased conditions, such as occur in connection with special forms of intracranial growths (syphilitic disease of the bones, throat, &c.; scrofulous disease of glands, joints, lungs) in a supposed case of intracranial tumour. The presence of ear disease in a supposed case of cerebral abscess. The presence of lung disease in a supposed case of limited meningitis, &c., &c.

In addition to these symptoms, which may be present in local "coarse" cerebral lesions irrespective of their seat, there may be—

5. Special symptoms due to the special position of the lesion. (Localising symptoms.) Amongst which I may mention:

(*a*) Local spasms, which are usually of much more frequent occurrence than the general epileptiform convulsions in which they terminate; and with which we are at present concerned.

(*b*) Affections of individual *cranial nerves.* Local paralyses; local alterations in the sensibility of the face; disturbances of the special senses.

(*c*) Symptoms which result from lesions of special parts of the encephalon. Such as alternate hemiplegia in tumours of the pons varolii, &c., &c.—

6. *A previous history of*—

(*a*) Symptoms of cerebral tumour.

(*b*) A head injury *—(a frequent exciting cause of intracranial growths)—a chronic ear discharge, &c.

7. *A family history* of cancer, scrofula, &c., may be of some corroborative value.

The negative facts in favour of a local "coarse" cerebral lesion are:

1. The absence of cardiac and arterial disease,—the great causes of embolism, thrombosis, and of cerebral hæmorrhage.

2. The absence of the symptoms and signs of kidney disease —the cause of uræmia.

3. The absence of any indications of lead impregnation; in which condition symptoms and signs identical with those of a "coarse" cerebral lesion occur.

4. The absence of marked pyrexia and delirium; conditions which are usually associated with diffuse (not local) meningitis or cerebritis.†

Having decided in favour of a local "coarse" cerebral lesion we have next to determine whether it is a tumour, chronic abscess, or limited meningitis.

The differential diagnosis of intracranial tumour and chronic cerebral abscess.

A cerebral abscess—so long as it remains chronic (*i. e.*, does not give rise to meningitis or cerebritis) may be regarded simply as an intracranial tumour.

In some cases there are no symptoms (it is entirely latent). Any diagnosis is then of course out of the question. In other cases the symptoms resemble more or less closely those of

* Genuine epilepsy sometimes follows a head injury. This and the other facts mentioned above are of course not absolute. In estimating their importance all the facts of the case must be taken into consideration, and due allowance be made to the relative value of each.

† The abscess of pyrexia and delirium is chiefly valuable as a negative diagnostic of intracranial tumours. In the case of abscess of the brain intercurrent attacks, characterised by these symptoms, not unfrequently occur.

solid intracranial growths. The points of distinction to which attention is to be directed are—

1. *The presence of bone disease* (running from the ear, &c.) the great cause of cerebral abscess.

2. *The exact characters of the symptoms.* In cerebral abscess the symptoms are often very stationary and chronic. There is less frequently optic neuritis. Local paralyses and spasms do not so frequently occur.

Acute exacerbations, characterised by violent symptoms, headache, vomiting, delirium, mania, convulsions, high temperature, &c.; and depending upon meningitis or cerebritis, may occur, and cause a rapid and fatal termination.

(The occurrence of an acute attack of this discription, especially in an adult, should suggest the presence of purulent meningitis. In all such cases a careful local examination, more especially of the ear; and a rigid inquiry into the history and symptoms is necessitated. Such cases often pass unrecognised, and are returned as deaths from acute mania, typhoid, &c.)

The differential diagnosis of cerebral tumour and a limited meningitis of the convexity is often very difficult. (In some cases it may be impossible owing to the fact that the two conditions are combined, as in some scrofulous cases.) Both conditions may give rise to headache, vomiting, and limited or general epileptiform convulsions.

The points to which attention is to be directed in making the distinction are—

1. *The temperature.* There is generally some pyrexia in cases of meningitis. The temperature is not increased, and in some cases is subnormal, in uncomplicated cases of cerebral tumour.*

2. *The condition of the optic discs.*—As I have already more than once remarked, there is double optic neuritis or optic atrophy in the great majority of cases of intra-cranial tumour. In the great majority of cases of meningitis, so long as the lesion is limited to the convexity, optic neuritis is not observed.

3. *The associated pathological symptoms.*—Limited meningitis (when not traumatic, including under that term meningitis due to external injury, extension of ear disease, &c.) is nearly always tubercular. The presence, therefore, of associated disease in the lungs, peritoneum, or glands, is of some value, as corroborative evidence. Or rather, the absence of

* It is quite exceptional to find a normal temperature of continued duration, in cases of meningitis. Such cases do however occasionally occur.

such associated pathological conditions is against limited meningitis.*

4. *The presence of paralyses of individual cranial nerves.*—In cases of intracranial tumour affections of individual cranial nerves, such as result from pressure on the nerve trunk, are common. In cases of meningitis, limited to the convexity (the condition which we are at present considering), local paralyses of cranial nerves do not occur.

5. *The course of the case.*—The course of local meningitis of tubercular origin is usually much more rapid than that of intracranial tumour. Characteristic symptoms of meningitis—viz., pyrexia, delirium, coma, &c.—soon appear and place the diagnosis beyond doubt.

6. *The general state of nutrition.*—In cases of tubercular meningitis, emaciation is a more prominent symptom; indeed, in many cases of cerebral tumour the general nutrition is well preserved.

7. *The effects of treatment.*—Temporary improvement often occurs in the case of intracranial tumours. In syphilitic cases the improvement is not unfrequently permanent, and directly attributable to this treatment. In tubercular meningitis the case usually goes on from bad to worse, in spite of all treatment.

8. *The previous history.*—In cases of cerebral tumour there may be, and often is, a history of long continued headache, attacks of vomiting, convulsions, paralyses, &c. In cases of meningitis the paralysis (hemiplegia or monoplegia), when present, is of recent date.

(*To be Continued.*)

CASE OF HÆMATOMA OF THE PERICRANIUM IN A CHILD SIMULATING HYDROCEPHALUS AND ASSOCIATED WITH CONSTITUTIONAL SYPHILIS.

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A boy, 8 years of age, was admitted to the Western Infirmary on 9th October, 1879. His head was enormously enlarged,

* The positive evidence is not of so much value; for a scrofulous tumour may be associated with the same lesions as a tubercular meningitis.

Dr. Gairdner, from his experience as a pathologist, was interested in the remarks of Dr. Coats. In 1851 he was greatly disabled by the pathological virus. He had at that time a succession of boils—150 or 200, at different parts of his body. After going abroad without much benefit, he was led to give up pathology as a special branch of study. While before that period he was only occasionally and temporarily subject to such attacks, it is remarkable that ever since then, if he merely handled pathological products, he was liable to an occurrence of pathological spots. So that here a special susceptibility to the virus had been induced by the virus itself.

18

ORIGINAL ARTICLE.

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(*Being a Lecture delivered in the Extra-Academical School of Medicine, Edinburgh, during the Summer Session, 1880.*)

(*Concluded from page 190.*)

Convulsions Resulting from (General) Meningitis.

For practical purposes cases of general meningitis may be divided into three great groups:—

1. Cases of meningitis resulting from traumatic injury, including under this head the suppurative meningitis, which follows bone disease.
2. Cases of typical, tubercular (basilar) meningitis.
3. Cases of meningitis which arise idiopathically, including cases of epidemic cerebro-spinal meningitis.

In all of these epileptiform convulsions may occur.

The *positive* facts in favour of general meningitis are:—

1. *The character of the symptoms.*—In many cases two