CASE REPORT : REFLEX EPILEPSY AND PERIPHERAL INJURY

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In discussing the significance of reflex epilepsy Critchley (1933) stated that an incoming stimulus would hardly provoke convulsive phenomena unless there was a state of supercharge in the central nervous system. Holmes (1927) described cases in which a cortical lesion apparently caused the disturbed peripheral response to a specific stimulus, and he considered that the appropriate external stimulus may excite epileptic symptoms if the corresponding part of the cortex is in an unstable state. A number of the cases in Critchley's series were associated with severe structural changes in the brain, and he also quotes a case of Rosenhain's in which attacks were precipitated by irritating the stump of an amputated finger, a necropsy eventually showing a tumour of the parietal lobe of the opposite hemisphere. Dawson (1947) has since shown experimentally that a peripheral stimulus can produce an electrical response over the contralateral cortex.

The association between the epileptic response and peripheral injury in some cases of reflex epilepsy has been reviewed recently by Parsons-Smith (1948) who added a case of his own. This was the case of a man who had cerebral cysticercosis and subsequently sustained a peripheral nerve injury, after which focal and generalized fits were precipitated by the passage of a stimulus through the injured peripheral nerve.

The following case also shows evidence of abnormal cortical function and peripheral nerve injury but differs from Parsons-Smith's case in that the patient suffered from traumatic epilepsy for many years before he sustained the peripheral injury which led to the development of reflex fits.

Case Report

The patient was a widower, aged 62, who came of healthy stock, and from whom no family history of epilepsy or other nervous disorder could be obtained. He was in good health until 1917 when he sustained a penetrating wound of the right frontal region while on active service in France. The right orbit was also injured and this resulted in a permanent squint and almost complete loss of vision in the right eye. He

made a satisfactory recovery from the immediate effects of the wound but about 12 months later he began to suffer from fits. These were typical major epileptic convulsions and at first occurred three or four times a week. The attacks gradually became less frequent and in recent years have occurred at intervals of about six months. They have always been preceded by an aura of slight giddiness. In 1927, i.e. 10 years after the head injury, he injured his left foot. He was rather vague about the details of this injury, but it evidently resulted in swelling and ulceration over the dorsum of the foot and took a long time to heal. After this he became aware that his fits were sometimes related to the peripheral injury, in that painful stimulation of the left foot might precipitate an attack.

He remained in good general health and had been free from attacks for six months, when, on March 5, 1949, he fell and again injured the left foot and ankle. He was taken to hospital and found to have marked swelling and ecchymosis over both malleoli, especially on the lateral side where there was acute tenderness. A radiograph showed a simple fracture of the lower third of the left fibula. A plaster cast was applied and he was allowed to return home. He attended the fracture clinic a week later when it was decided to renew the plaster. The old cast was being cut with plaster shears when he had a fit. The attack began with clonic movements of the left foot and leg, spreading to the left arm and face, and followed rapidly by a generalized convulsion and loss of consciousness. When he was examined about 10 minutes later, he had recovered consciousness but was still confused and unable to give a history. A brief neurological examination was now carried out, and while the left plantar response was being tested he had another fit. After making a further attempt to examine him, it was realized that the stimulus applied to the outer border of the sole of the left foot in eliciting the plantar response was precipitating the fits. Several further attacks were produced in rapid succession by this means. He was then admitted to hospital for a few days until the pain and swelling of the ankle had subsided, when he was discharged home in a walking plaster. No further fits occurred in hospital but he was now taking $\frac{1}{2}$ gr. phenobarbitone three times a day. Further progress at the fracture clinic was uneventful and the plaster was eventually removed without incident.

He was re-examined as an out-patient after an interval of seven months when a detailed neurological examination was made. He was a heavily built, elderly man

who walked with a slight limp. There was a depressed scar over the right frontal region extending slightly upwards and laterally from the medial third of the supra-orbital ridge to a point two inches above the pinna. An underlying bony defect could be felt. There was a marked external squint and only hand movements could be detected with the right eye. The visual acuity of the left eye was 6/12 (uncorrected). There was marked atrophy of the right optic disc and gross pallor of the surrounding retina. Numerous areas of deep pigmentation at the sites of old haemorrhages were also seen. The left fundus appeared normal. The scar, and the skin over the greater part of the distribution of the first division of the right fifth nerve, were anaesthetic to pin-prick. Both corneal reflexes were brisk and sensation was normal over the remainder of the face and head. The other cranial nerves were normal. There was no weakness of the upper or lower limbs or trunk. Coordination was normal. The deep reflexes of the upper limbs were brisk and equal on both sides. The left knee and ankle jerks were slightly more active than those on the right and the lower left abdominal reflex was absent. Both plantar responses were flexor. Appreciation of all forms of sensation on the left side of the body was slightly impaired except over the lateral aspect of the lower third of the left leg and the outer border of the left foot, where there was marked hyperaesthesia. The hyperaesthetic area corresponded to the skin distribution of the sural nerve. There was a circular, depressed scar, $\frac{3}{4}$ in. in diameter, on the medial side of the left foot, and an irregular scar, $2\frac{1}{2}$ in. $\times$ 1 in., over the lateral aspect of the dorsum of the foot. Both scars were anaesthetic.

There were no abnormal physical signs in the cardiovascular, respiratory, or digestive systems. The blood pressure was 160/100. The urine contained no abnormality.

A radiograph of the skull was reported on as follows:—

“A bony defect is shown at the right frontal region, presumably due to an old injury. A dense opacity is shown in the mid-line just above the floor of the anterior fossa and anterior to the anterior clinoid processes. ? Metallic foreign body from old injury. There appears to be some reaction round it.”

He had had three fits since his discharge from hospital seven months before, each attack having been precipitated by excitement. An attack occurred soon after he arrived at the hospital for re-examination. He was in an excited state on arrival and was very apprehensive about the unusual interest which was being taken in his case, and these emotional factors probably precipitated the fit. This attack began with clonic movements of the left foot, as in the previous attacks, but consciousness was not lost and he recovered completely within two minutes. The neurological examination was then carried out without incident until the left plantar response was tested. The firm stroking stimulus applied to the outer border of the sole of the foot at once precipitated a typical Jacksonian fit which began in the part stimulated. The attack was confined to the left side of the body and consciousness was not lost. Further

identical attacks were readily produced by the same stimulus and by the application of a painful stimulus (pin-prick) but light touch and a tickling stimulus had no effect. Neither was there any response to thermal stimulation. The trigger zone was confined to the hyperaesthetic area on the outer margin of the foot. Stimulation of the remainder of the sole was without effect, and there was likewise no response to stimulation of the area of hyperaesthesia on the lateral aspect of the lower third of the leg.

The result differed from that seen seven months earlier in that the attacks were now localized to the left side, they were brief, and consciousness was not lost. The patient had taken no drugs for several months so that it would appear that the cortex was either in a less irritable state, or that the threshold to the appropriate peripheral stimulus had increased.

Discussion

This case confirms the view of Holmes that peripheral stimulation may excite an epileptic response if the corresponding part of the cerebral cortex is in an irritable state. In discussing the mechanism of reflex epilepsy, the writer of an annotation in the *Lancet* (1948) states that a study of all the effective stimuli makes it clear that in most instances there is an abnormality in the pathway responsible for the afferent impulse. This abnormality may be in the receptor organ, in the peripheral nerve or spinal cord, or, more often, in the central receiving area in the cerebral hemispheres. Parsons-Smith (1948) considers that in those cases associated with peripheral injury there may be some abnormality in the reflex arc on the afferent side between the periphery and the cortex. The evidence of the present case supports this view.

In Parsons-Smith's case the actual nerve injury did not serve as a noxious stimulus to the reflex, but it seemed that the stimulus of light touch was altered as the result of the injury and served to detonate a focal cortical outburst when applied to the area supplied by the injured nerve. In the case described above, the patient had been subject to traumatic epilepsy for 10 years before he sustained a peripheral nerve injury. The actual injury did not precipitate an epileptic attack, but he became aware that subsequent fits were sometimes related to painful stimulation of the site of the injury. When the same limb was injured again after a further interval of 12 years, a painful stimulus to the injured part precipitated a generalized convulsion. This occurred within a week of the injury, when the foot and ankle were still swollen and painful. Further generalized fits were readily produced by the application of a painful or pressure stimulus to the foot. Seven months later it was still possible to produce focal fits by applying the appropriate

stimulus to an area of hyperaesthesia corresponding to part of the distribution of the injured peripheral nerve. The history that, following the first injury to the foot, fits were sometimes precipitated by painful local stimulation, together with the presence of hyperaesthesia, suggests that the threshold to pain was lowered in the area supplied by the injured peripheral nerve, and that this was sufficient to cause a focal discharge from the corresponding area of cortex which was already in an unstable state, when the appropriate stimulus was applied. The second peripheral injury probably resulted in a further lowering of the threshold, with a corresponding increase in the ease with which the fits could be precipitated. When the patient had made a complete recovery from the second injury stimulation of the hyperaesthetic area precipitated focal fits, without loss of consciousness. As no drugs had been taken for several months, the reduction in the severity of the reflex fits was probably due to recovery from the second injury with consequent raising of the threshold to painful stimulation to its previous level. This case differs from Parsons-Smith's and from those described by Holmes in that a painful or deep pressure stimulus was the specific trigger, whereas they found that a light tickling touch was effective, and the sensations of pressure and pain did not act as excitants. In one of

Critchley's early cases, however, focal fits were precipitated by scratching the sole of the foot when testing the reflexes, as in the present case.

Summary

A case of traumatic epilepsy is described, in which focal and generalized fits were precipitated by applying a specific sensory stimulus to the site of a peripheral nerve injury.

This case confirms the view that peripheral stimulation may excite an epileptic response if the corresponding part of the cerebral cortex is in an unstable state.

The probable effect of peripheral injury on the threshold to local sensory stimulation is discussed.

I wish to thank Dr. L. W. Smith for permission to report this case; Dr. Macdonald Critchley for his criticism; and Miss D. D. Hayes for her help in tracing the patient.

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