Review Article

Behavioural treatment of epilepsy

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Summary: There is abundant evidence of the close interrelation between the induction and inhibition of seizure activity and the patient's thoughts, feelings and behaviour. The detailed knowledge that we now have of the epileptic focus and the way that it is connected to the surrounding cerebral mechanisms, makes it possible for many patients with focal epilepsy to establish a significant degree of seizure control by altering their thinking and behaviour.

Introduction

The accepted model of seizure genesis is that seizures arise as a result of abnormal brain discharges, usually caused by a change in brain excitability arising from genetic causes. The excitability may also be caused by a damaged area of brain. More recent models of seizure genesis make it possible to relate the long acknowledged link between the genesis of seizure activity, normal brain function and the psychic life of the individual.

Seizure genesis

Lockard,¹ in an animal model of focal epilepsy, has defined two populations of epileptogenic cells. Group 1 neurones are at the centre of the focus. They are partially damaged and always fire in an epileptic, bursting mode. They fire continually and their activity is not modified to any significant extent by surrounding brain activity. Group 2 cells are partially damaged neurones surrounding the focus. They can fire in both the bursting, epileptic mode, and in a normal mode. Their activity can therefore be modified by surrounding brain activity.

A focal seizure occurs when the continually discharging group 1 cells recruit group 2 cells into the seizure discharge. If group 2 cells recruit cells in the surrounding normal brain, the focal seizure spreads to become secondarily generalized. This model shows that there are two points in the

evolution of a seizure when ongoing brain activity can either increase or decrease the likelihood of the seizure developing. The first is between group 1 and group 2 neurones, and the second between group 2 and normal brain neurones.

There are also brain mechanisms which may modify the likelihood of generalized seizures. Musgrave and Gloor² and Avoli and Gloor³ have suggested that reticular formation activity may have a direct effect on generalized spike-wave seizures. Reticular activity, like cortical activity, varies as a function of behaviour, and so behaviour could be expected to have a direct effect on generalized seizure frequency.

Lockard⁴ has also shown that the numbers of spikes occurring at any one time can be modified by, amongst other things, psychosocial processes. Thus, both spike frequency and seizure frequency may vary as a function of behaviour.

Evoked seizures

Seizures which are precipitated by specific external stimuli such as reading, eating, movement, sounds or smells, are called 'evoked seizures'. They are said to occur in about 5% of people with epilepsy,⁵ though a study of patients attending the Maudsley Hospital indicates a rate of nearer 25%.⁶ The current concept is that peripheral stimulation raises the level of activity within a damaged area of the cortex, by rhythmic driving of the group 2 cells, and so allows seizure discharge to spread within the focus. The opposite may also be true: an alteration in the level of excitation in the area of damaged cortex may prevent seizure activity from arising and spreading.

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Psychogenic epileptic seizures

Psychogenic epileptic seizures are those which arise as a consequence of mental activity. Primary psychogenic epileptic seizures are produced by a deliberate mental attempt to induce a seizure. The Maudsley hospital survey indicated that between a quarter and a third of patients attending a psychiatric epilepsy clinic are able to generate their own seizures at will.

Secondary psychogenic epileptic seizures (also called the 'thinking epilepsies') are those which occur when the subject is thinking, calculating, or 'feeling', but not trying to induce a seizure.⁶⁻¹² It is well known that patients describe having more seizures in certain situations. In the Maudsley survey, over 50% of the patients said that they had seizures when they were tense, depressed, or tired, and over 30% had them when they were angry, excited, or bored. Only 4% of patients said they had seizures when they were happy. Temkin and Davis¹³ also found that high stress levels are associated with more frequent seizures. Happiness is clearly a very powerful anticonvulsant.

Seizure inhibition

Certain types of activity tend to increase the likelihood of seizure occurrence, while others tend to decrease it. Seizure inhibition, like seizure generation, consists of both primary and secondary components. Primary seizure inhibition is the direct inhibition of seizures by an act of will. The main mechanism of action is the alerting of the patient in some way, so altering the level of cortical excitability non-specifically. Patients whose seizures have focal onsets will commonly either say 'no' to themselves, or try to attend to something different at the onset of the aura. Secondary inhibition occurs when a patient produces a reduction in seizure frequency by an action of mind or behaviour, without deliberately intending to do so. Maintenance of interest or alertness are such measures. The final common pathway for all such activity in those patients who have focal seizures is the alteration in activity of group 2 neurones.

Psychological methods for treatment of epilepsy

Behavioural methods for the treatment of epilepsy include reward management, which gives positive reinforcement to seizure-free periods; self-control procedures which allow the patient, by cognitive processes, to gain control of his seizure activity (for example methods which involve relaxation and covert desensitization), and psychophysiological treatments. However, it must be recognized that

there is likely to be more than one therapeutic factor in any conditioning programme.

Individual or group psychotherapy has also been used to give the patient a better understanding of himself and the relationship of his seizures to life-events.^{14,15} However, this is not always successful.¹⁶

Induction and inhibition of seizures

Most patients will admit to having a mental mechanism which they use to try to inhibit their seizures. A study at the Maudsley Hospital of 70 outpatients showed that over a third claimed that they could sometimes stop their seizures from happening, or prevent them from spreading. One 42 year old woman had had feelings of *déjà vu* since the age of 6, which had at times been associated with feelings of guilt. Her therapy, in part, consisted of helping her deal with her guilt, which she described as the most powerful anticonvulsant that she had ever been given. This patient had a right temporal lesion, with a right temporal focus on electroencephalogram (EEG). Thus the group 1 neurones were probably situated in the hippocampus and amygdala, and these areas were activated when she felt guilty.

Dahl *et al.*,¹⁷ in a study of 18 children, found that all 18 were able to predict their seizures; three could elicit them on demand, and 12 could identify low-risk situations for seizures. The study showed that the children were able to discover pre-seizure cues, and that using a relaxation technique significantly reduced seizure frequency. Non-specific attention did not have this effect.

In 1987, Dahl *et al.*¹⁸ looked at 18 adults with refractory epileptic seizures. They found that patients who were given relaxation therapy showed a significant seizure reduction. They were able to determine the early onset of their seizures, and abort them with the relaxation procedure. However, not all seizures could be stopped this way, and the authors could find no clear predictors as to those seizures which could be stopped.

Dahl *et al.*¹⁹ have also used intervention techniques to reduce both seizure frequency and paroxysmal EEG activity. The patient is taught to use a counter-measure at seizure onset, such as the moving of an arm in the direction opposite to that caused by seizure onset. They found that it is not enough to identify pre-seizure behaviour: there must be an active intervention to stop seizures spreading and reduce seizure frequency.

Conditioning of seizures

Epileptic seizures can be conditioned in a classical Pavlovian paradigm. The conditioning of seizures is a fragile process and can be obliterated by one

grand mal seizure. Forster²⁰ has applied classical methods of habituation and extinction to the control of epilepsy. Abnormal cortical discharges evoked by a specific stimulus are habituated by continual exposure to the stimulus. This has been tried in the visual, auditory and sensory modalities, with some success.²¹⁻²⁵

Biofeedback

Several workers have used biofeedback training to reduce seizure frequency, with varying degrees of success. Sterman²⁶ was the first to investigate the anticonvulsant properties of the 12-16 Hz sen-

sorimotor rhythm (SMR) in man, by using biofeedback training. This is a rhythm which arises over the sensorimotor area of the brain, and whose amplitude biofeedback sets out deliberately to enhance. Although many of these early experiments show the effectiveness of biofeedback procedures, compared to relaxation and placebo, they did not demonstrate the specificity of the SMR.⁶ However, current opinion now supports the view that the SMR change may contain components specific to the reduction of epileptic seizures. Other investigators have tried the effect of conditioning different background cerebral rhythms with some success.²⁷⁻²⁹

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