the meatus and middle ear mechanism is possible. Even if no middle ear exists, the provision of a skin-lined meatus permits the wearing of a hearing aid insert so that amplified sound may be delivered to a functional cochlea.

These operations are difficult because the anatomy may be very abnormal and the facial nerve may be encountered in an unexpected situation. The meatus needs to be made as wide as possible and lined with a free skin graft to reduce the inevitable contraction. A new tympanic membrane may need to be fashioned and the ossicle chain reconstructed. This is all done under the operating microscope. If one ear is normal there is no urgency to operate, as postoperative management is much easier in the older child. But in bilateral cases early operation within the first 2 years of life is essential to restore hearing so that speech development may proceed normally.

Unfortunately, the cases with inner ear defects are much more common, and for these surgery can offer no help. The use of the now available prophylactic inoculation against rubella may, however, cause a striking reduction in the incidence of this type of deafness. Here the small residual hearing that is usually present must be used and stimulated at the earliest possible time, for without such stimulus the auditory area in the brain will not develop and use of it will be lost for ever. Hearing aids and parent guidance in the principles of auditory rehabilitation form the basis of management in the first few years, followed by the selection of a school suited to the severity of the defect. This may mean a school for the deaf, or in less severe cases who have some speech development, one for the hard of hearing. In mild cases, or after a time in a hard-of-hearing school it may be possible for the child to attend a normal school using a hearing aid. In such situations the understanding and co-operation of the class teacher are important.

Mention has not been made of congenital abnormalities not associated with deafness such as accessory auricles and pre-auricular fistulae. These are minor problems compared with the handicap of the deaf child.

It is hoped that this brief review has shown that much can be done for some of these little patients, and that something of value can be done for all.

I wish to thank Dr J. G. Burger, Medical Superintendent of Groote Schuur Hospital, for permission to publish.

REFERENCES


A Case of Television-Induced Epilepsy with Repetitive Head Movements During the Seizure*

FRANCES R. AMES, M.D., M.MED., D.P.M., AND MARTIN PIETERSEN, Department of Neurology, Groote Schuur Hospital and University of Cape Town

SUMMARY

A case of 'television-epilepsy' is reported. An unusual clinical feature was repetitive head movements occurring during the seizure and the pathogenesis of these is discussed.


In this article we report the case of an adult female in Britain whose only generalized seizure occurred while she was adjusting a faulty television set. The main purpose in reporting the case is to draw the attention of South African doctors to the entity of 'television epilepsy'. An additional reason is that this observant and articulate patient described unusual repetitive head movements during the seizure; the pathogenesis of these requires analysis.

CASE REPORT

A 24-year-old White female related that 4 years ago, while adjusting a faulty black-and-white television set, she became uncomfortably aware of the movement of the images on the screen. These hurt her eyes so that she screwed them up. She then saw pale blue yellow flashes on the screen, which were visual hallucinations not seen by her companion. She turned her head to the left to avoid looking at the screen but her head moved back 'involuntarily' to its original position. She again looked to the left but her head was again 'pulled back, as if it was on a
Some patients, however, are sensitive to lower frequencies than the rate of 2 waves a second in front of the patient's eyes, and was followed within 4 seconds by a burst of slow waves of increased amplitude (Fig. 3).

DISCUSSION

It is well known that in photosensitive epileptic patients seizures can be evoked by exposure to light, especially if it is flickering between 15 and 25 frequencies per second. Some patients, however, are sensitive to lower frequencies or even to single transition from light to darkness or vice versa. Our patient clearly had photosensitive epilepsy. She not only developed a photoconvulsive EEG response and myoclonic jerking to stroboscopic stimulation at 20 flashes per second, but also manifested EEG abnormality on intermittent interruption of sunlight by a hand passing in front of the patient's eyes. During hyperventilation irregular theta activity appeared in the posterior leads. Binocular intermittent photic stimulation with the eyes closed did not provoke any abnormality until the frequency was increased to 20 flashes a second. At this stage a generalized burst of high amplitude polyspike and slow wave activity appeared (Fig. 1). This persisted for about a second after the stroboscope had been switched off. During this period the patient blinked, jerked, screwed up her eyes and complained of a 'sort of painful tetanus of my eye muscles'. During subsequent EEG recordings it was established that EEG abnormality was enhanced by eye closure, abolished by active or passive opening of the eyes, and that she was insensitive to monocular stimulation. At times during intermittent photic stimulation it tended to be more marked on the left (Fig. 2). When exposed to bright sunlight no abnormality appeared until one of us (F.A.) waved a hand (with fingers abducted) at the rate of 2 waves a second in front of the patient's closed eyes. Irregular generalized slowing then appeared and was followed within 4 seconds by a burst of slow waves of increased amplitude (Fig. 3).
The entity of 'television epilepsy' is now generally accepted. Daube\(^1\) stated that 100 cases were reported between 1952 and 1956. Pantelakis \(\text{et al.}^5\) estimated the incidence as being 5.7\% among epileptic children. Seizures while watching television occur mainly but not exclusively in the second half of childhood; they are often grand mala type fits and are particularly likely to occur if the patient is close to the set, in dimly-lit surroundings or looking at a faulty set. The most potent factor in pathogenesis is thought to be flicker. Vulnerability to particular frequencies of flicker has been invoked to explain the fact that significantly more cases have been reported from Europe than from the USA. Charlton and Hoefer\(^6\) stated that 'of the adequately reported cases 55 are in the European literature and only 3 in the United States'. They ascribed this to the fact that in European television the scanning rate is about 50 per second at the usual viewing distance, but this rate is halved if the viewer is close to the set. This brings the flicker stimulus to 25 per second, a frequency to which photosensitive epileptic patients are particularly vulnerable. By contrast, in the USA the scanning rate is 60 per second. Also in the USA a greater number of lines is scanned (525) than in England (405), and this enhances image fusion, thus reducing the flicker effect.

There is a discrepancy between the ease with which EEG abnormality and myoclonic jerking can be evoked by stroboscopic stimulation in the laboratory and the difficulty in evoking these by exposure to television. Gastaut \(\text{et al.}^3\) working with the French radio-television service was unable to do so, though Daube\(^4\) mentions two continental writers who were successful. This suggests that other factors, apart from flicker, may be important in pathogenesis. These include excitement, fatigue, eye closure,\(^9\) the type of retinal stimulation,\(^9\) hypersensitivity of oculomotor muscles,\(^10\) intensity of light, and so on.

**Management**

It is impracticable to forbid patients, especially children, to watch television. The frequency of attacks can be diminished if they are warned to look away if the set becomes faulty, to avoid changing and adjusting channels and to sit in a well lighted room at least 1.5 m from the screen. Anticonvulsant medication is advisable if the patient has seizures or myoclonic jerks when not exposed to television.

**REFERENCES**