RESPIRATORY FAILURE AS A SEIZURE PHENOMENON

A case report

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SUMMARY

Recurrent acute respiratory failure in an epileptic subject is described. The first episode of respiratory failure occurred while the patient was having frequent epileptic fits and was probably secondary to cerebral oedema with temporal herniation. The second occurred suddenly after 27 days during which the patient had been free from seizures. It is suggested that this episode of acute respiratory failure was the result of an epileptic seizure without any motor symptoms.

The common central causes of spontaneous respiratory failure include central nervous system depression by drugs as well as various brain stem and spinal cord lesions. In the case described here respiratory arrest was observed in an epileptic patient on two occasions, when the airway was completely patent, the ventilatory capacity adequate and respiratory mechanics undisturbed. Both incidents of respiratory failure were considered central in origin. The first occurred during status epilepticus, the second suddenly and unexpectedly after 4 weeks of freedom from epileptic fits, presumably as a consequence of epileptic activity spreading to and interfering with the function of brain stem respiratory centres.

CASE REPORT

A 57-year-old white male was admitted to the Clinic of Neurology at Academician Dérer's Hospital in Bratislava. He had suffered two severe head injuries: the first in 1945, and the second in 1952 which resulted in unconsciousness lasting for 3 weeks and a residual, slight right hemiparesis. The seizures started 2 months after the second injury and were manifested by rotation of the head and eyes to the right, occasionally by oral automatisms and by tonic and clonic movements on the right side which spread in varying degrees to the left. At the end of the fit, restlessness with depressed consciousness persisted, sometimes for up to 30 min.

Five days before admission, serial seizures developed and during the 24 hr before admission to hospital they recurred at intervals of less than 10 min and lasted about 30 sec. On admission, the patient was drowsy with a right-sided hemiparesis and aphasia. He was treated with phenobarbitone 110 mg i.m. thrice daily and diazepam 10 mg i.v. or more as required. Within the 24-hr period before admission the patient had been given diazepam 90 mg in all (70 mg i.v. and 20 mg i.m.) and, in addition, mannitol as an intravenous infusion.

Two days after admission sudden respiratory failure occurred, manifested by marked cyanosis and gasping respiration with extreme bradycardia. Manually controlled respiration (with 100% oxygen) was started immediately by means of a self-inflating bag and face mask. The trachea was subsequently intubated. Spontaneous respiration was soon restored, but the endotracheal tube was left in place as the patient remained unconscious. The fits recurred shortly afterwards at approximately 45-min intervals. The anti-epileptic therapy remained unchanged. In the next 24 hr the response to painful stimuli disappeared, the pupils ceased to react to light, the corneal reflex was absent on the right and decreased on the left. Five days after admission the patient was still unconscious but breathing spontaneously, and a tracheostomy was performed. The frequency of epileptic fits began to decrease and ceased on the 10th day after admission. Simultaneously, the patient regained consciousness, with only a slight right-sided hemiparesis.

Left carotid angiography 7 days after admission showed narrowing of vessels, possibly as a result of cerebral oedema with signs of temporal herniation. The electroencephalogram (e.e.g.) on admission showed an epileptic focus in the fronto-centro-temporal region on the left (figs. 1, 2). However, the e.e.g. pattern improved in parallel with the improvement in the clinical state of the patient such that 3 days after the respiratory arrest the e.e.g. showed a complete disappearance of focal signs and persistence of a slight diffuse abnormality.

Twenty-seven days after the seizures had ceased,
FIG. 1. At the outset of a typical clinical seizure, the e.e.g. pattern showed spike activity over the left hemisphere with a successive generalization.

while the patient was receiving oral diazepam 15 mg thrice daily and phenobarbitone 100 mg thrice daily, a second incident of acute respiratory failure occurred. This happened at 6.40 a.m. when the patient was turned on his side for nursing purposes. The nurse observed rattling and gasping irregular breaths (5–6 b.p.m.), the lips were blue, the face pale, and there was tachycardia. The patient did not respond to painful stimuli. Both the corneal reflex and the light reflex of pupils were absent and there was muscular hypotonicity with loss of tendon and peristomal reflexes. Within the first minute the nurse administered oxygen, 10 litre/min, by nasopharyngeal catheter. During the next minute, controlled respiration with a self-inflating bag, face mask and pure oxygen was instituted. The trachea was intubated and the patient was ventilated by means of a Bird Mark 8 ventilator. The cyanosis disappeared promptly, the corneal reflex reappeared symmetrically and the patient began to respond to painful stimuli by movements. At 9.00 a.m. he regained consciousness, responded adequately to commands, but remained drowsy. Assisted ventilation was continued. The heart rate was 76 beats/min, the arterial pressure was 140/90 mm Hg and the electrocardiogram showed sinus rhythm without signs of myocardial damage. On e.e.g. examination only moderate diffuse slowing of the background activity was seen, without focal changes or signs of epilepsy. At 3.00 p.m. the patient was disconnected from the ventilator, and following an hour of adequate spontaneous breathing the endotracheal tube was removed.

Two days later the patient was able to walk unaided and his neurologic status was similar to that existing before this second episode of respiratory failure.

DISCUSSION
Alterations in respiratory function associated with epileptic discharges have been described frequently. In petit mal Bogacz and Yanicelli (1962) almost always found respiratory changes which included short periods of apnoea. Whether the respiratory arrest occurred in expiration or in inspiration depended on the phase of the respiratory cycle during which the epileptic discharge arose.

Similar changes have been observed in focal epilepsy, especially in temporal lobe epilepsy, which has been induced with leptazol (Van Buren and Ajmone-Marsan, 1960). Penfield and Jasper (1954) described respiratory arrest in man following the stimulation of the anterior part of the cingulate gyrus or deep temporal structures, and occasionally of the inferior parts of the sensorimotor region. Umbach (1966), also studying man, observed respiratory changes including arrest with stimulation of many limbic structures. Animal studies established the alteration of respiration by discharges from the limbic system and diencephalon, but the technique of anaesthesia...
and the type of stimulation affect the response (Oberholzer and Tofani, 1960; Kaada, 1960). Respiratory changes only rarely appear to be the principal and important components of a seizure, but such patients have been described. Nelson and Ray (1968) reported a patient with temporal lobe epilepsy who developed nine episodes of severe respiratory failure, explicable only as seizure phenomena. Another case was that of a child with digitalis intoxication in whom the epileptic seizure discharges in the e.e.g. were constantly associated with respiratory arrest (Feuerstein et al., 1973). The sudden and unexpected death of an epileptic, unexplained by autopsy, is relatively frequent. Such deaths have been attributed to acute functional disturbances in cardiorespiratory centres as a result of seizure discharge (Hirsch and Martin, 1971).

The first occurrence of respiratory failure in our patient was during status epilepticus and several factors which were present simultaneously could have influenced the normal functioning of the respiratory centres. These included cerebral oedema, high doses of barbiturates and diazepam, together with the temporal herniation shown by carotid angiography.

Before the second incident, the treatment consisted of low doses of anti-epileptic drugs. All common causes of acute respiratory failure could be excluded and a central origin seemed likely. From the outset of the acute respiratory disturbance no motor signs of an epileptic fit were visible, but this cannot eliminate a seizure discharge in neuronal circuits controlling respiratory function. Such explanation conforms with the observations of Nelson and Ray (1968) and Hirsch and Martin (1971), that motor manifestations of the seizure were absent before the respiratory arrest, although some of the patients had previously shown widespread tonic and clonic convulsions. An acute cerebrovascular accident manifested exclusively by respiratory failure is unlikely because of the rapid recovery and absence of any other residual symptoms.

Thus, it is probable that in the present patient acute respiratory failure developed as a consequence of propagation of seizure discharges leading to dysfunction of brain stem respiratory centres. There is no simple theory to explain why seizure discharges involve, occasionally, the respiratory centres or why this arises so rarely.

The e.e.g. from scalp electrodes does not provide satisfactory information on deep subcortical structures. The e.e.g. recorded in our patient, after the incident, showed only an irregular slow activity probably resulting from unconsciousness and cerebral hypoxia. The e.e.g. pattern recorded before the event revealed a frontotemporal focus and a tendency to rapid spreading of the focal epileptic activity which fluctuated markedly in the course of time. Van Buren and Ajmone-Marsan (1960) failed to find any direct relationship between e.e.g. changes and respiratory alterations. With equal discharges in e.e.g. record from the scalp surface, changes in respiration may not be constant, but only occasional. Thus, it could be postulated that the spread to subcortical structures may vary also. Beyond this, extensive involvement of subcortical structures at a distance from the region of an experimental focus may not be evident even when depth macroelectrodes are being used; diffuse changes of neuronal unitary activity are reliably demonstrable only when using microelectrodes (Jasper, 1964).

The literature and the present case show that epileptic seizures occasionally can be manifested clinically by sudden respiratory failure without any motor symptoms.

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REFERENCES


INSUFFISANCE RESPIRATOIRE COMME PHENOMENE DE CRISE

RESUME
On décrit dans cet article un cas d’insuffisance respiratoire rechutante chez un sujet épileptique. Le premier épisode d’insuffisance respiratoire s’est produit lorsque le malade avait de fréquentes crises d’épilepsie lesquelles provenaient probablement d’un oedème cérébral ayant une distension temporaire. La seconde s’est produite soudainement, après qu’une période de 27 jours se soit écoulée sans qu’il y ait eu de crise. On suggère que cet épisode d’insuffisance respiratoire aiguë provenait d’une crise épileptique sans aucun symptôme moteur.

ATMUNGSVERSAGEN ALS ERGEBNIS VON ANFALLSERSCHEINUNGEN

ZUSAMMENFASSUNG
Beschrieben wird wiederholtes akutes Atmungsversagen bei einem Epileptiker. Der erste Fall von Atmungsversagen trat ein, während der Patient häufige epileptische Anfälle hatte, und war wahrscheinlich Folge eines vorangegangenen zerebralen Ödems mit temporärer Herniation. Der zweite Fall trat plötzlich nach 27 Tagen ein, während welcher Zeit der Patient frei von Anfällen gewesen war. Es wird angenommen, daß dieser zweite Fall von akutem Atmungsversagen das Ergebnis eines epileptischen Anfalles war, bei dem keine motorischen Symptome aufgetreten waren.

FALLO RESPIRATORIO COMO FENOMENO DE ATAQUE

SUMARIO
Se describe un fallo respiratorio agudo repetido en un paciente epiléptico. El primer episodio de fallo respiratorio se produjo mientras el paciente tenía ataques epilépticos frecuentes y era probablemente con efecto secundario el edema cerebral con hernia temporal. El segundo se produjo de repente después de 27 días sin ataques. Se sugiere que este episodio de fallo respiratorio agudo fue el resultado de un ataque epiléptico sin ningún síntoma motor.