

## Treatment of Status Epilepticus with Diazepam (Valium)\*

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Valium has been shown (4) to be effective orally in the control of chronic epilepsy, and when used parenterally, we were pleased to find Valium remarkably effective for treating "status epilepticus". We, therefore, decided to carry out an extensive study of its use in such conditions.

### MATERIALS AND METHODS

Over a period of 6 months, we have studied the effects of slow intravenous or intramuscular injection of one or more ampoules (10 mg in 2 ml) of Valium in all the in-patients for whom an emergency EEG has been necessary because of status epilepticus. This study has been carried out in the Civil Hospitals in Marseilles and the Centre Saint-Paul (H.G., R.N. and C.A.T.), and in the Centre Psychothérapeutique at Nancy (R.P.).

From the large number of patients and in this short time, we have had the opportunity of studying 27 attacks of status in 23 patients, whose EEG's were suitably clear.

We have based our classification on the definitions proposed at the *10th Colloquium at Marseilles* (3), held in October, 1962, which gave special consideration to this matter. Accordingly we use the term *status epilepticus* to describe an epileptic attack, which is so prolonged or so frequently repeated as to constitute a definite lasting condition.

### CLASSIFICATION OF SEIZURES

The above definition covers such an extensive range that further classification is desirable.

#### *I. Generalized Status Epilepticus*

##### *A. Generalized convulsive status*

(i) *Tonic-clonic status epilepticus* (grand mal status), or the classic status epilepticus of Calmeil (2) and Bourneville (1), with generalized tonic-clonic paroxysms repeated

\* Valium Ro 5-2807/B-36 F has been supplied by Laboratoires Roche (Paris).

1–5 times per hour, coma, progressive autonomic disturbances, and a fatal prognosis if not stopped quickly. We distinguish between primary tonic-clonic status with fits generalized from the start, and secondary tonic-clonic status with partial seizures secondarily generalized.

(ii) *Tonic status* usually occurring in childhood. Such attacks occur 4–20 times in an hour and may disappear or fade away until they can only be perceived on the EEG. Autonomic disturbances may increase and lead to death from asphyxia and circulatory collapse.

(iii) *Clonic status* only occurring in infancy as prolonged clonic attacks, lasting several hours or days and accompanied by coma.

(iv) *Myoclonic status* also occurring in childhood as continuously repeated fragmentary or massive myoclonus, lasting several hours or days with impaired consciousness of varying degree from subject to subject.

#### *B. Non-convulsive generalized status epilepticus*

This absence status is characterized by clouding of consciousness, which may be slight, with simple slowing of ideation and means of expression; or more marked and accompanied by confusion; or severe with drowsiness or even stupor. They may last for hours or days. These states correspond to what Lennox (7) suggests should be named *petit mal status*.

### *II. Unilateral Convulsive Status Epilepticus*

These occur in young children, as hemiclonic attacks and may last for hours or days. They are always followed by hemiplegia, which fortunately usually passes off but may be permanent (H.H. syndrome), and sometimes may be accompanied by secondary epileptic attacks (H.H.E. syndrome).

### *III. Partial Status Epilepticus*

This form is confined to adults with repetition at varying intervals of any type of partial epileptic seizures and usually presenting itself as Jacksonian motor status or jerks incessantly repeated in the same part of the body [epilepsia partialis continua of Kojewnikoff (5)].

We would like to emphasize that an exact diagnosis cannot usually be made without the help of an EEG. This is particularly the case in certain varieties of unilateral status and also of absence status.

#### RESULTS

Owing to the great diversity of the material, we are not presenting our results massed together. It seems to us clearer and more logical to separate them according to the variety of status. In all our patients simultaneous EEG's and ECG's were recorded during their fits and treatment with Valium and monitoring of respiration was

also often done. Every time we mention a type of seizure (e.g. generalized tonic-clonic status), it may be assumed that we observed both its clinical and EEG characteristics. We shall only mention special details when necessary.

### *I. Valium in Generalized Status Epilepticus*

#### *A. Generalized convulsive status (15 cases)*

##### *(i) Tonic-clonic variety (5 cases)*

###### *Case 1*

*History.* LI., aged 28 years, has suffered from psychomotor epilepsy with left temporal focus in EEG since the age of 14, inadequately controlled by drugs. At 15 years, secondary generalized tonic-clonic attacks were seen; often of sufficient duration and frequency to constitute status epilepticus.

*Observed status.* Tonic-clonic status with fits secondarily generalized, spreading from an initial focus; 14 attacks in four hours, followed by coma. Temperature 38°C (100.4°F).

Following an intravenous injection of 10 mg Valium, the attacks ceased for one hour. Four attacks occurred in the second hour. The patient was given 20 mg Valium i.v., and 10 mg i.m. This controlled the attacks and an hour later the patient regained consciousness. There were no more seizure discharges on the EEG, only an interseizure focus in the left temporal area. The basic rhythms were fast.

###### *Case 2*

*History.* LA., aged 26 years, has suffered from psychomotor epilepsy since the age of 14, with an irritative focus in the left temporal area, often becoming secondarily generalized with several attacks of status epilepticus.

*Observed status.* Tonic-clonic status with fits secondarily generalized, spreading from a focus. Rapid progressive loss of consciousness. Valium 10 mg i.v. stopped the attack with no recurrence. The interseizure EEG became normal. Half an hour later the patient walked to his room.

###### *Case 3*

*History.* MU., aged 74 years, got a right hemiplegia of vascular origin the preceding year with a left temporal focus of  $\delta$ -waves.

*Observed status.* Tonic-clonic status with fits secondarily generalized, spreading from a focal discharge. Seven attacks in 40 min followed by coma. Serious autonomic disturbances endangering life. Valium 10 mg i.v. immediately stopped the clinical and EEG signs of the fits, and the interseizure EEG tracings rapidly became normal. The patient regained consciousness and the disturbances of the autonomic, cardiovascular, and respiratory systems disappeared.

###### *Case 4*

*History.* KU., aged 30 years, suffered, from the age of 13 on, from numerous gener-

alized tonic-clonic fits. The EEG between attacks showed generalized bilateral, synchronous, symmetrical, spike and wave complexes.

*Observed status.* Tonic-clonic attacks with generalized features from the start, occurring from 5–10 times an hour followed by coma. Temperature 38°C (100.4°F). Serious autonomic disturbances.

Phenobarbitone sodium 0.4 g i.m. had no effect. Valium 10 mg i.v. had no effect. Three further injections of 10 mg Valium i.v. were given in the next hour. The attack stopped a few minutes after the last injection. The patient regained consciousness and the autonomic disturbances ceased. Five hours later 10 mg Valium i.m. was given prophylactically.

#### *Case 5*

*History.* LOH., aged 47 years, with asthma. Rare generalized tonic-clonic seizures. EEG showing generalized bilateral, synchronous and symmetrical interseizure spike and wave complexes.

*Observed status.* Three generalized tonic-clonic fits in quick succession. Marked disturbance of consciousness, and dyspnoea. EEG showed abnormal  $\delta$ -rhythm with interseizure spike and wave complexes. Photic stimulation induced another tonic-clonic attack, thought without doubt to be the start of a generalized tonic-clonic status epilepticus. Valium 10 mg i.v. stopped the attack and considerably improved the interseizure EEG. Consciousness quickly returned.

#### *(ii) Tonic variety (3 cases)*

##### *Case 6*

*History.* RUE., aged 8 years, suffered from tonic attacks affecting the trunk only (axial), or the trunk and limbs (axorhizomelic), and accompanied by atypical absences. EEG showed interseizure generalized slow spike and wave complexes (petit mal variant). Has presented many previous tonic status lasting some days.

*Observed status.* Axial tonic status with paroxysms every 10–20 sec. EEG recorded 600 discharges in two hours. Marked clouding of consciousness. Valium 10 mg i.m. stopped the attack both clinically and on the EEG within half a minute, and consciousness returned quickly.

##### *Case 7*

*History.* JAU., aged 14 years, suffered from generalized tonic seizures. EEG showed interseizure slow generalized spike and wave complexes (petit mal variant). No previous history of status epilepticus.

*Observed status.* Axial tonic status with numerous paroxysms, amounting to several hundreds per hour, and accompanied by apnoea. Marked clouding of consciousness. Valium 10 mg i.m. had no appreciable effect. As this was one of our early cases and the safe action of Valium was not fully appreciated, the physician in charge preferred to use 2500 mg Eunocet, which lessened the frequency of paroxysms. After a night's sleep, the status epilepticus stopped.

*Case 8*

*History.* SCH., aged 34 years, alcoholic.

*Observed status.* During a drinking episode the patient developed status epilepticus with severe generalized tonic spasms and coma. Severe autonomic disturbances. Intramuscular barbiturates were given without effect. Valium 10 mg i.v. quickly stopped the paroxysms but 40 minutes later they started again. Coma and autonomic disturbances continued in spite of further injections of barbiturates. Twelve hours later, 10 mg Valium i.v. stopped the attack suddenly and completely. Consciousness returned and the autonomic disturbances ceased within a few hours.

*(iii) Clonic variety (5 cases)**Case 9*

*History.* FOU., aged 14 years, had suffered from violent myoclonus, causing falls, from the age of 1–3 years. Symptoms then stopped until the age of eight, since when there have been several clonic attacks lasting an hour on the average.

*Observed status.* (February 1964). Clonic type of status epilepticus with generalized frequent muscular twitchings of low amplitude (*véritable danse musculaire*), and severely impaired consciousness. Considerable autonomic disturbances. One minute after 10 mg Valium i.v. had been given all signs stopped both clinically and on the EEG. Thirty minutes later, myoclonus returned accompanied by bilateral synchronous spike and wave complexes on the EEG. Valium 10 mg i.v. stopped the attack within 20 sec for one hour only, so 2500 mg Eunocet was injected. One and a half hours later the attack was definitely suppressed.

*Case 10*

*History.* Same subject as in Case No. 9, readmitted in status epilepticus.

*Observed status.* As in Case No. 9. The patient had already received 2500 mg Eunocet without effect. Valium 10 mg i.v. completely suppressed all signs in 40 sec.

*Case 11*

*History.* The same subject again in a third attack of status epilepticus.

*Observed status* was identical to the two preceding attacks. Valium 10 mg i.v. stopped the status in 30 sec.

*Case 12*

*History.* BAT., aged 12 years, had suffered from the age of three, from right hemiclonic attacks, and occasional generalized tonic-clonic seizures; generalized bilateral synchronous interseizure spike and wave discharges.

*Observed status.* Repeated hemiclonic attacks followed by generalized clonic status epilepticus. Impaired consciousness. A few minutes after 10 mg Valium i.v. all paroxysms progressively decreased and the electrical spike and wave discharge disappeared.

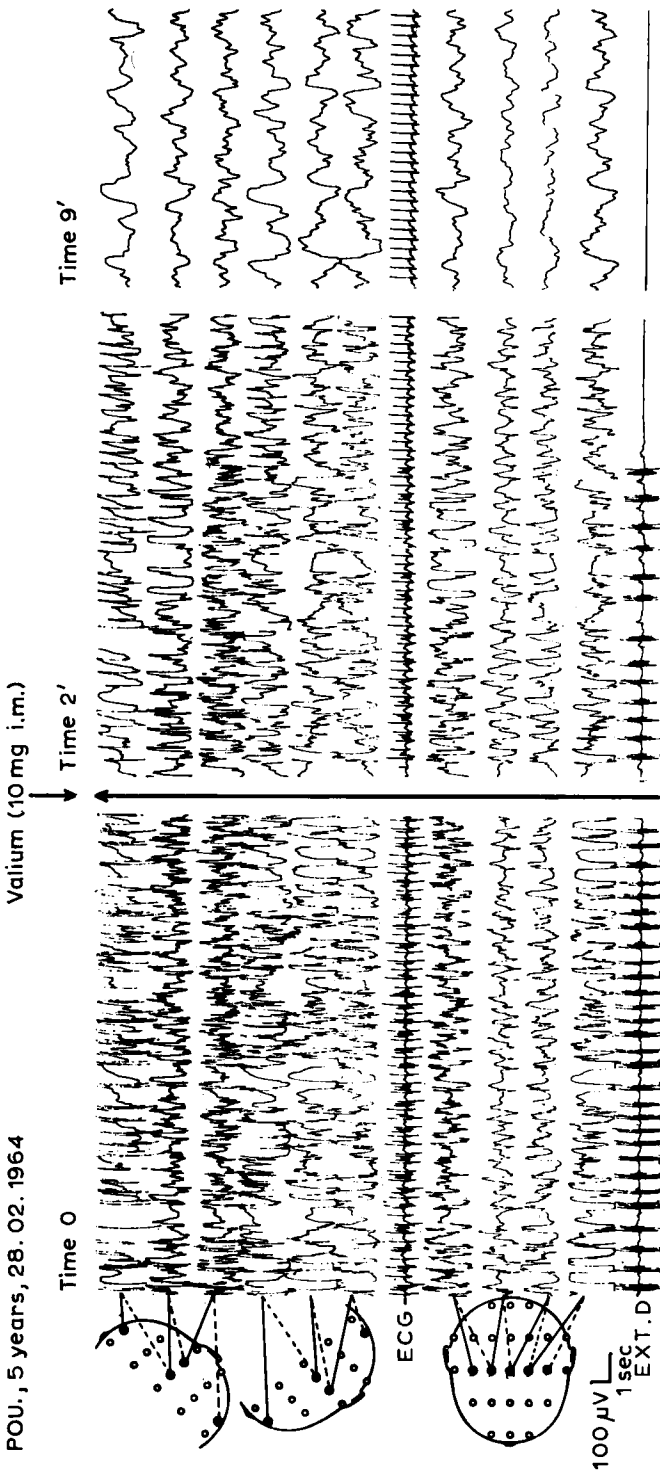


Fig. 1. POU., 5 years old (Case 13). Simultaneous recording of the EEG (in bipolar), of the electrocardiogram (ECG), and also of the EMG of the common extensor muscles of the right fingers (Ext.D). Generalized clonic seizure, as it is shown in the left part of the figure (Time 0). Two minutes (Time 2') after an intramuscular injection of 10 mg of Valium, the frequency of the muscle clonic discharge (Ext. D) becomes progressively slow and suddenly disappears. The EEG record is still very abnormal, with slow waves mixed with important components of spike activity. Nine minutes (Time 9') after the injection, the EEG shows only slow waves. All the irritative elements have disappeared completely.

*Case 13*

*History.* POU., aged 5 years, had from the age of nine months, numerous clonic attacks, often brought on by pyrexia. In one severe status epilepticus, tracheotomy was necessary.

*Observed status.* Clonic status epilepticus with continuous spike and wave complexes shown on the EEG. The paroxysms stopped a few minutes after 10 mg Valium i.m. had been given. The EEG returned to normal more slowly (Fig. 1).

*(iv) Myoclonic or myoclonic-amyotonic status (2 cases)**Case 14*

*History.* BAR., aged 14 years, had suffered from sporadic generalized myoclonic attacks during infancy becoming more frequent. Slight progressive mental deterioration. At the age of 13, a cerebello-spinal syndrome developed. EEG showed generalized, bilateral, symmetrical, synchronous polyspikes and spike and wave complexes, occurring both spontaneously and as a result of photic stimulation. Diagnosis: Ramsay Hunt's myoclonic cerebellar dyssynergy.

*Observed status.* Increasingly frequent myoclonic paroxysms preventing all activity. On the EEG, the multiple spike and wave complexes ran into each other. Strictly speaking, this case should not be regarded as true status epilepticus. The attack lasted several days, during which time two intramuscular and two intravenous injections of 10 mg Valium were given. Twenty sec after both intravenous injections and 10–15 min after both intramuscular injections, the muscular jerks and spike and wave complexes in the EEG disappeared for 1–3 hours, depending on the route of injection. Each time they reappeared, they were less intense and allowed some rest for 24 hours. At the same time, sensitivity to photic stimulation was reduced.

*Case 15*

*History.* RAC., aged 8 years. Extreme sensitivity to light necessitated the patient being kept in the dark, as otherwise myoclonus and generalized amyotonic fits might occur several hundred times a day.

*Observed status.* A sudden deterioration of condition in spite of being kept in the dark. Incessant myoclonus and amyotonic fits prevented the patient from standing. Valium 10 mg i.m. suppressed the status epilepticus in approximately 3 min. The next day there were only occasional attacks of myoclonus and amyotonia.

*B. Generalized non-convulsive status (absence status) (6 cases)**Case 16*

*History.* MAT., aged 50 years, had suffered from typical absences several times a day in infancy, becoming rare since adolescence. At the age of 30, three generalized tonic-clonic seizures, since when he has taken 200 mg phenobarbitone sodium daily and had only occasional absences.

*Observed status.* When admitted to hospital in a marked state of confusion, his EEG showed tracings characteristic of absence status (Fig. 2). Valium 10 mg was given

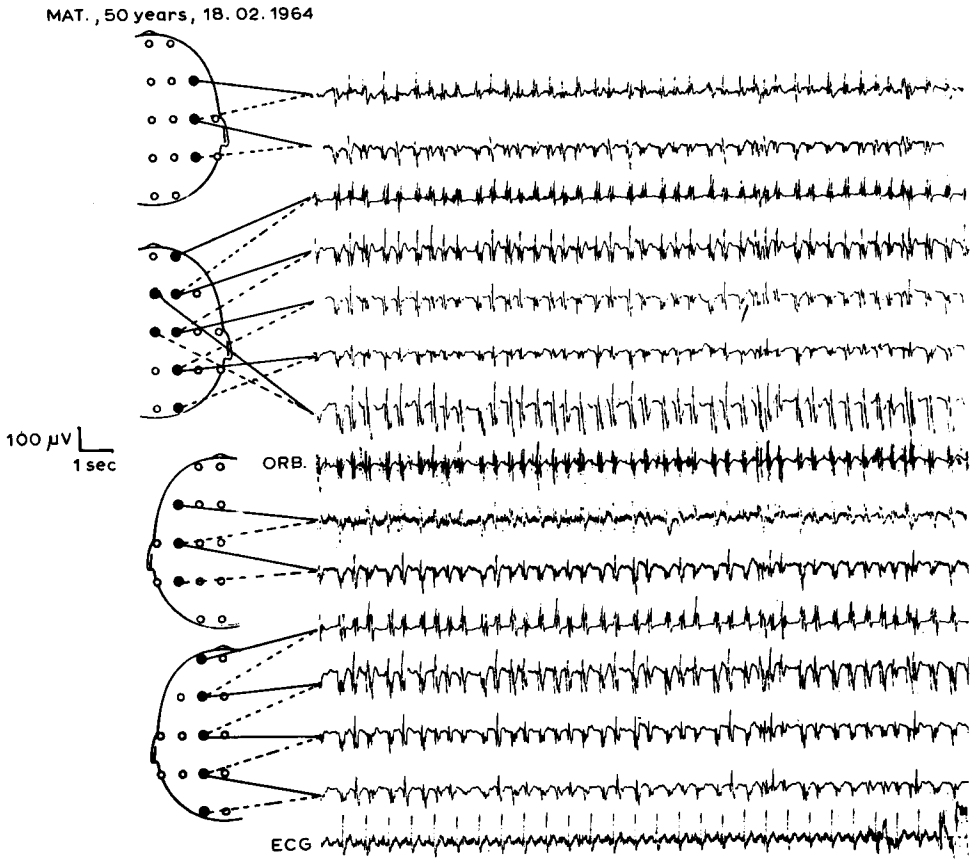


Fig. 2. MAT., 50 years old (Case 16). Bipolar EEG recording combined with electrocardiogram (ECG) and with electromyogram of the orbicular muscle of the eyelid (ORB.). Typical activity of an absence or petit mal status.

by slow intravenous injection. The patient regained consciousness and the EEG became normal before the injection was completed (Figs. 3, 4). Rarely have we witnessed such a dramatic change in such a short time.

#### Case 17

*History.* The same patient, MAT., aged 50 years, was readmitted two months later. *Observed status* identical to the previous one; responded as dramatically to 10 mg Valium i.v.

#### Case 18

*History.* THE., aged 5 years, had suffered from numerous typical absences since the age of three, together with occasional atonic and tonic seizures. Very sensitive to



MAT., 50 years, 18. 02. 1964

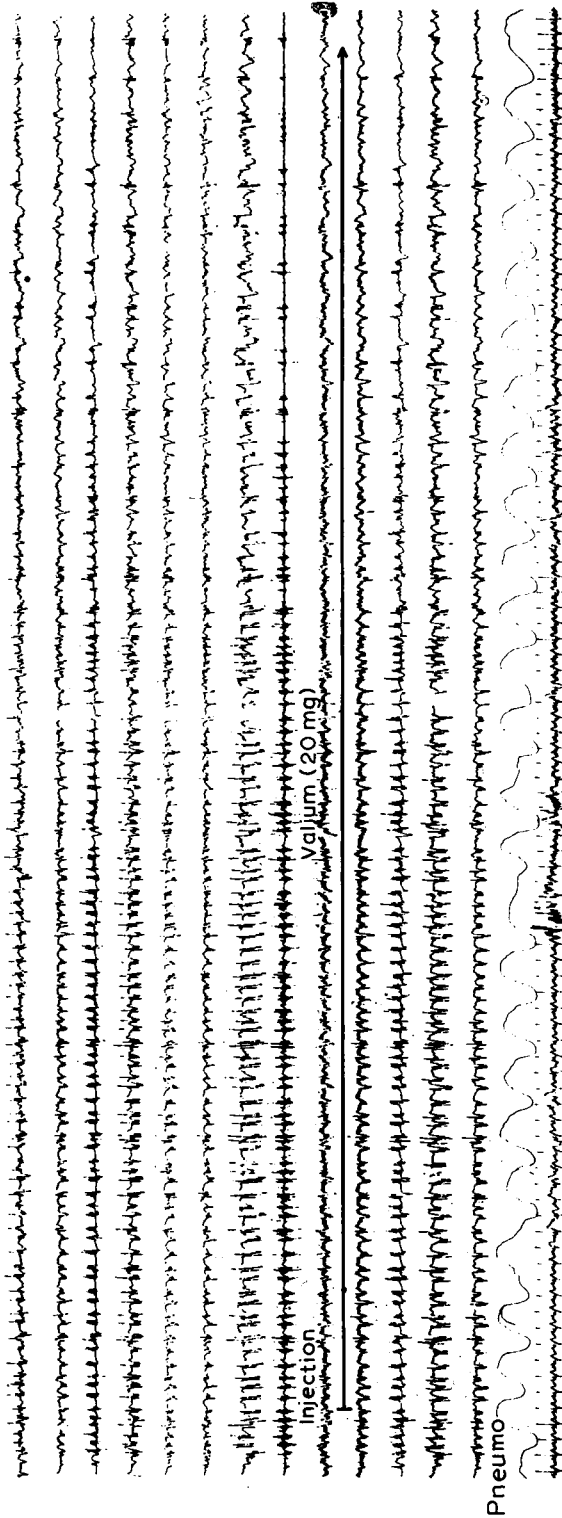


Fig. 3. MAT., 50 years old (Case 16). Intravenous injection of 20 mg of Valium, with the same derivations as before (Fig. 2), but the 14th channel is now occupied by pneumogram (Pneumo). During the injection, the myoclonus discharges of the orbicular muscle disappear progressively, and at the same time the EEG is modified, the "spike and wave" discharges disappear.

MAT., 50 years, 18. 02. 1964 10' later

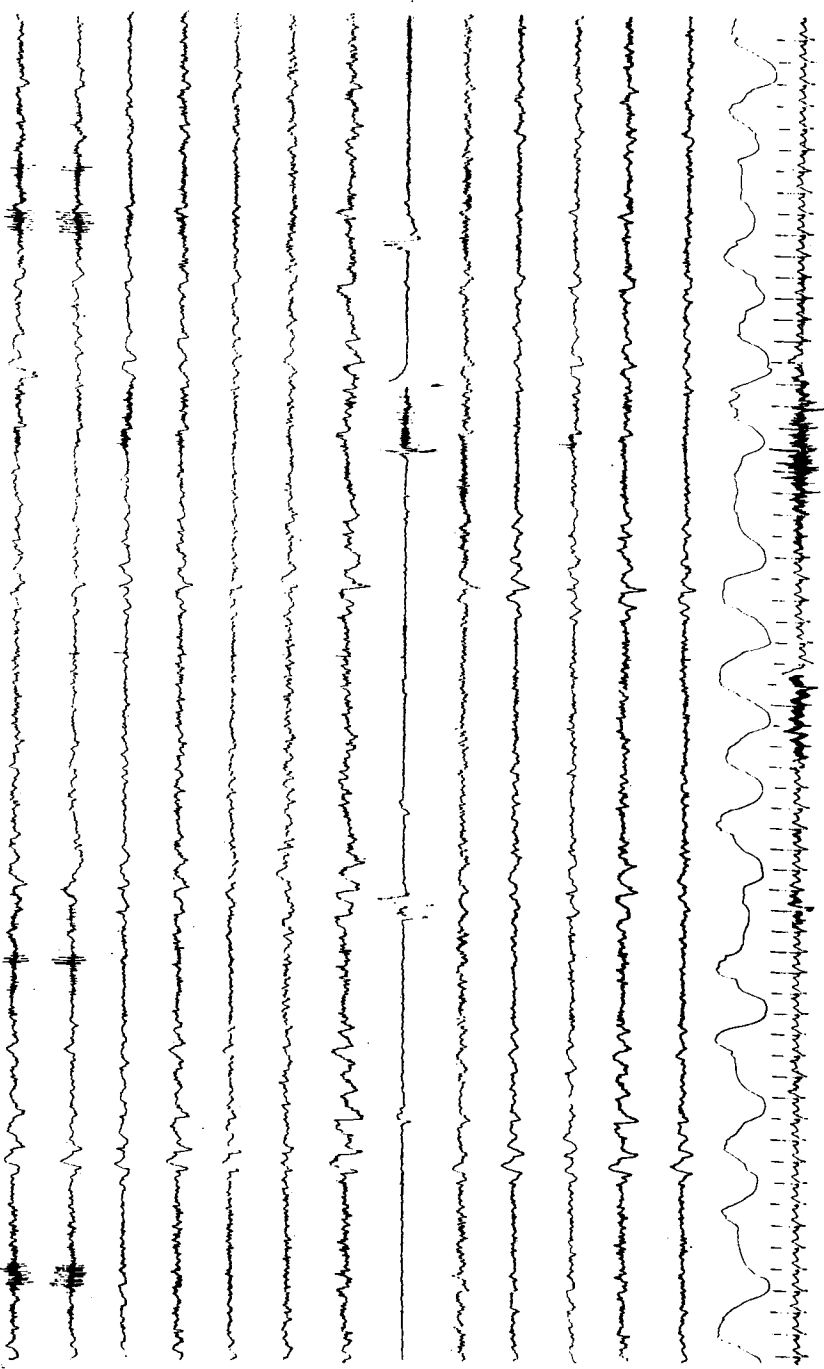


Fig. 4. MAT., 50 years old (Case 16). Ten minutes later, the background activity has come back to the limits of normal. The "spike and wave" discharges have disappeared completely. Only some bursts of generalized delta-waves persist.

photic stimulation. The EEG showed interseizure spike and wave episodes, easily provoked by photic stimulation.

*Observed status.* Absence status with slight clouding of consciousness and characteristic EEG pattern. The status epilepticus terminated within 20 sec of 10 mg Valium i.v.

#### *Case 19*

*History.* COL., aged 40 years, underwent a bilateral prefrontal leucotomy in 1954. Residual irritative focus located in right frontal region by EEG.

*Observed status.* Megimide 21 ml i.v. during an EEG study induced an absence status with marked impairment of consciousness, together with specific EEG changes and marked photosensitivity. The status epilepticus ceased abruptly when only 7 mg Valium had been injected intravenously.

#### *Case 20*

*History.* GUI., aged 27 years, suffered from convulsive and non-convulsive seizures of an ill-defined nature since childhood, with non-specific EEG.

*Observed status.* Megimide 25 ml given intravenously for a diagnostic EEG caused a classical absence status with typical EEG appearances. All signs of the status epilepticus stopped immediately after 10 mg Valium i.v.

#### *Case 21*

*History.* C., aged 16 years, oligophrenia.

*Observed status.* Cardiazol 3 ml i.v. during an EEG study caused a generalized tonic-clonic seizure, followed by a typical absence status confirmed on the EEG. Valium 5 mg i.v. stopped the status epilepticus.

### *II. Valium in Unilateral Convulsive Status Epilepticus (3 cases)*

#### *Case 22*

*History.* DI NAP., aged 9 months, suffered from hemiclonic attacks, alternately right- and left-sided, since the first week of life; obviously retarded development.

*Observed status.* Predominantly right-sided clonic seizures with EEG signs typical of a clonic status in left cerebral hemisphere. Valium 6 mg i.v. stopped the status epilepticus in 20 sec with flattening of the EEG waves. Respiration became shallow and apnoea with collapse was feared. However, the patient slowly returned to normal. The next day the status epilepticus returned. Eunocetal was given, because of the respiratory complications following Valium given previously. The status epilepticus disappeared without respiratory complications.

#### *Case 23*

*History.* FIN., aged 6 years, got in 1960 a left H.H.E. syndrome following left-sided otitis. First hemiclonic status epilepticus in 1962.

*Observed status.* Typical left-sided clonic status both clinically and on the EEG followed a recurrence of otitis. Valium 10 mg i.v. stopped the attack in a few seconds.

*Case 24*

*History.* SAU., aged 40 years, got his first attack during cardiac surgery, when an accidental air embolism caused a hemiclonic status epilepticus with periodic spike discharges on the contralateral side of the scalp. Autonomic disturbances, life endangered. Valium 10 mg i.v. stopped the epileptic attack immediately. Treatment continued with 100 mg Valium daily, given by intravenous drip. The status epilepticus remained under control and the disordered autonomic system improved considerably.

*III. Valium in Partial Epileptic Status (3 cases)**Case 25*

*History.* RO., aged 29 years, underwent leucotomy in 1957. In 1960 a partial seizure with secondary generalization. In 1961 first status epilepticus.

*Observed status.* Seizures of Jacksonian type, 84 in six hours, affecting the right arm and face with corresponding seizure discharges in the left fronto-central area. Marked clouding of consciousness.

Valium 10 mg i.v. stopped the clinical signs but had no effect on the EEG appearances. To prevent recurrence and stop the electrical discharges, 60 mg Valium was given over the next three hours. The abnormal EEG signs grew less and disappeared after the last injection. The patient slowly returned to consciousness.

*Case 26*

*History.* CH., aged 56 years, showed progressive psychic deterioration for one year. Ideomotor apraxia.

*Observed status.* Left tonic-clonic focal seizures with ictal discharges in the right Rolandic area. Paroxysms repeated every 15–20 sec. After 10 mg Valium i.v., clinical signs disappeared but the EEG abnormalities remained. Ten minutes later, clinical signs returned. A further 10 mg Valium was given intravenously; clinical signs disappeared quickly and the EEG changes more slowly.

*Case 27*

*History.* RIC., aged 16 years, suffered from frontal sinusitis and had no previous history of epilepsy.

*Observed status.* Sudden left-sided convulsive status epilepticus shown on the EEG to be connected with a localized discharge from the right cerebral hemisphere (Fig. 5), probably due to a thrombo-phlebitis of the same hemisphere. Coma. Pyrexia, with a temperature of 40.5°C (104.9°F); and pulse 150/min. Valium 10 mg was given by slow intravenous injection. When about three-quarters had been given, the clinical signs and EEG abnormalities grew less and ceased as the injection was completed. No further seizures occurred.

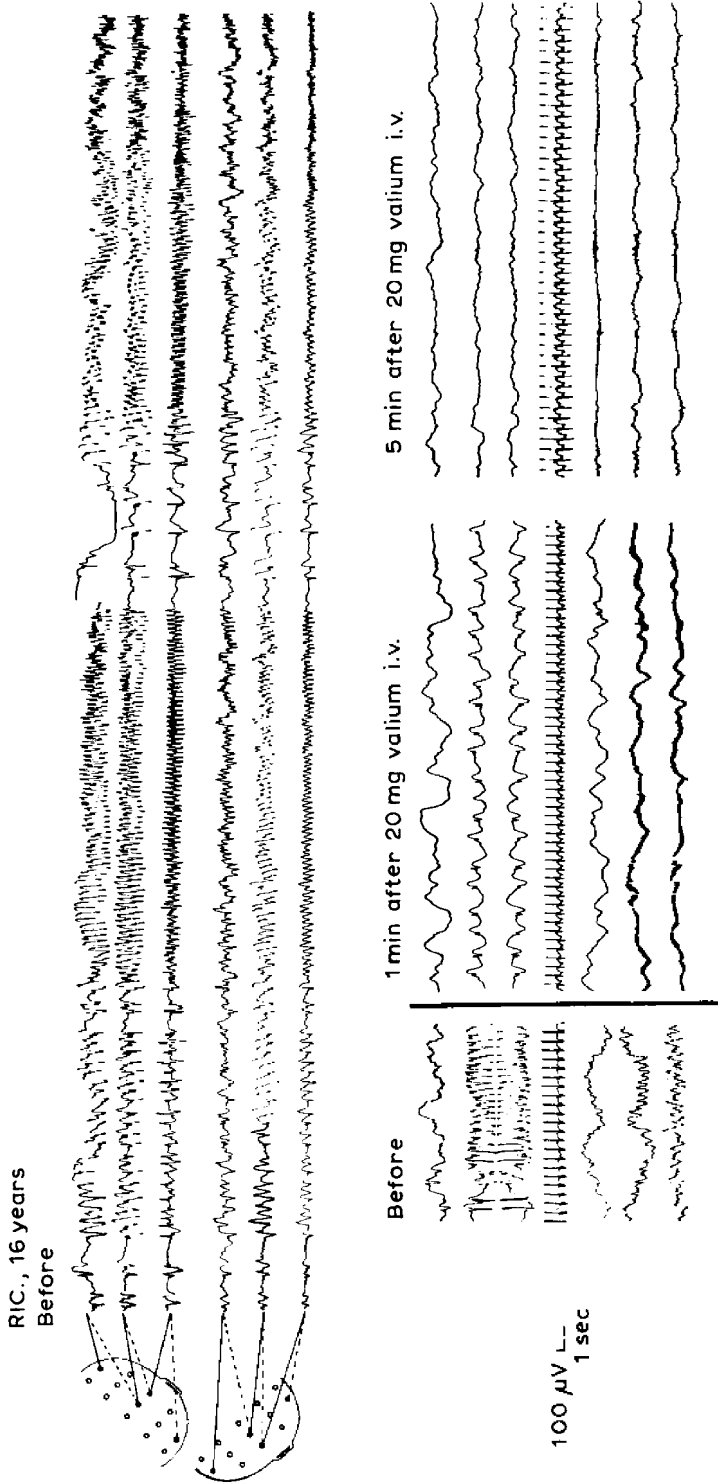


Fig. 5. RIC., 16 years old (Case 27). Recording of a succession of seizures, generalized but more predominant on the right hemisphere marked by Before. After an injection of 20 mg of Valium, the seizures disappear progressively. One minute after the injection the electric discharge persists only in the posterior part of the right hemisphere, but its frequency is slower and its morphology has changed. Five minutes after, it has disappeared completely.

*IV. Valium in Continuous EEG Discharges without Clinical Signs and in Incessant Abnormal Movements without EEG Changes*

Twenty cases were treated with Valium with spectacular results. No details are given here as such cases do not come within our definition of status epilepticus.

DISCUSSION

In the 15 cases of *generalized convulsive status epilepticus* reported here, we consider that Valium was outstanding for the reliability and rapidity of its action which together make it a more effective drug than others we have used in the past, among which have been: injectable phenobarbital, Somnifene, chloralhydrate, Eunocotal, sodium bromide, Rectanol, Novocain, and Hemineurin.

In 12 of these 15 cases, we obtained complete relief, the status epilepticus being stopped by one, or occasionally more, intravenous or intramuscular injections of 10 mg Valium. The speed of action of Valium was remarkable. Control of seizures was achieved generally during the slow intravenous injection, or within 20 seconds of completing the injection, and, in the case of intramuscular injection, a few minutes later.

We regard most of our failures as being relative and largely due to lack of experience with the drug at the time they were treated. One case (No. 7) received one intramuscular injection of 10 mg Valium only. Another case (No. 9) also received 10 mg Valium intramuscularly with partial success; but when readmitted later (as Case Nos. 10 and 11), he was successfully treated with Valium.

On the other hand we regard the relief given in Case No. 14 as only relative. The continuous muscle clonus, which constituted the status epilepticus, was controlled, but the residual clonus, due to the underlying degenerative encephalopathy, remained as a permanent feature. Furthermore, since finishing this report, a complete failure with Valium has been observed: it concerned a 14-year-old child who suffered for several days from a tonic status which entirely resembled the observations made with the cases 6 and 7. This status resisted i.m. and i.v. injection and perfusion of Valium as well as injections of Phenobarbitone sodium and Eunocotal, enemas with chloral hydrate and Rectanol, novocain perfusion and Fluctane inhalation. After a development of ten days, during which attacks of a purely electroencephalographic character were observed every 20 seconds and vegetative disturbances necessitated a tracheotomy, a fatal outcome being considered imminent, the attacks ceased spontaneously and the child returned to a normal state within three days.

Among the six cases of *generalized non-convulsive status epilepticus* we obtained relief in all cases, which, we think, is remarkable. In this group, the most spectacular and dramatic results were obtained, signs disappearing often after only 5-7 mg Valium had been injected intravenously. We have never obtained such results in the past, although we have used a variety of drugs, including: Tridione, Pentothal, Eunocotal, Hemineurin, ACTH, and aldosterone.

In the three cases of *unilateral convulsive status epilepticus*, we also obtained ex-

cellent results. One case (No. 24) being particularly gratifying to treat as the patient suffered an air embolism during open heart surgery.

The 2 or 3 cases of *partial status epilepticus* were completely controlled, the clinical signs disappearing before the EEG abnormalities. In one case (No. 25) a much greater dose of Valium was needed to abolish the latter than the former signs.

In the cases of *induced seizures* unfortunately developing into status epilepticus, whether caused by convulsant drugs (Megimide and Metrazol) or photic stimulation, Valium was as effective as in cases of spontaneous status epilepticus.

Obviously Valium affects only the actual status epilepticus and not the basic causes, nor accompanying phenomena. For example, in the case of air embolism (No. 24), signs of cerebral impairment were present clinically and in the EEG after the status epilepticus was controlled. In several other cases of status during severe cerebral lesions, with a traumatic or particularly a vascular etiology, Valium has shown itself inefficient to stop definitely the convulsions and above all the discharges on the EEG. That is why we consider that Valium enables, in some ways, to differentiate: (1) properly so-called status, in which a functional factor (probable decrease of the convulsivant threshold) plays an essential part: most of the time Valium stops these within a few seconds or minutes, and (2) convulsive manifestations of come cerebral "catastrophes" (mostly vascular) in which the responsible factor is exclusively organic and insensible to the action of the product.

In case No. 22, an infant, 9 months old, the injection of 6 mg Valium i.v. led to respiratory insufficiency which alarmed us, but from which the patient recovered. In no other case did we experience any untoward effect following the injection. In another case (which will furthermore be the object of a special study) in which almost continuous epileptic discharges were accompanied by serious respiratory disorders of Cheyne-Stokes type, the injection of Valium has not only stopped the status, but has also made respiration regular. Therefore we concluded that this product has no dangerous depressing effect upon respiration and circulation.

In all cases, EEG's and ECG's were recorded during injections of Valium and often we were also able to monitor respiration. We arranged for the EEG records to be done simultaneously, not only because of the value of such records, but also to enable us to keep the dose to the minimum. Thus in cases Nos. 16 and 27, we were able to stop the status epilepticus successfully with less than 10 mg Valium.

Our results show Valium to have an effective, one might say spectacularly effective, action on spontaneous epileptic attacks which develop into status epilepticus, and correspond closely with those observed by Poiré *et al.* (9, 10), Lanoir *et al.* (6) and Naquet *et al.* (8), who reported the prophylactic effect of Valium on generalized epileptic seizures in man induced by intermittent photic stimulation, and on the more or less localized afterdischarges induced in cats by electrical stimulation in the neocortical or rhinencephalic areas.

#### SUMMARY

Valium has proved to be one of the most effective drugs we have used in the treatment

of status epilepticus. We have given one or more injections intravenously or intramuscularly, and also used up to 100 mg daily in a slow intravenous drip. Most status are stopped in a few seconds or minutes, whether generalized or partial, convulsive or non-convulsive. In the doses we used we met no respiratory nor cardiac side effects. Valium is, in our opinion, the drug of choice for the emergency treatment of all cases of status epilepticus. Moreover it enables to distinguish properly so-called status which stops in most cases, from convulsive manifestations of cerebral "catastrophes" upon which it has no lasting effect.

#### RÉSUMÉ

Le Valium s'est révélé être une des drogues les plus efficaces que nous ayions utilisées dans le traitement des états de mal épileptiques. Nous avons pratiqué une ou plusieurs injections par voie intraveineuse ou intramusculaire et nous avons également utilisé jusqu'à 100 mg par jour en perfusion lente intraveineuse. La plupart des états de mal épileptiques sont arrêtés en quelques secondes ou minutes, qu'ils soient généralisés ou partiels, convulsifs ou non convulsifs. Avec les doses utilisées, nous n'avons rencontré aucun effet respiratoire ou cardiaque secondaire. Le Valium est, à notre avis, la drogue de choix pour le traitement d'urgence de tous les cas d'état de mal épileptiques. De plus, il permet de distinguer les états de mal proprement dits, qui s'arrêtent dans la plupart des cas, des manifestations convulsives de "catastrophes" cérébrales sur lesquelles il n'a aucun effet durable.

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