THE CONTROL OF POST-CONVULSIVE CONFUSION BY VALIUM

By YIP CHONG, WONG

Consultant Psychiatrist, 24, Ngee Ann Building (1st Floor), Orchard Road, Singapore 9.

In a private practice where modified E.C.T. has to be given out infrequently there is a need to reduce this to a reasonably safe and simple procedure.

This need is generally satisfied by the selection of one of the several techniques available which combines a hypnotic and a muscle relaxant to be followed by artificially maintaining the respiration until recovery from the drug effects sets in.

Where a quick turn-over is desirable for various reasons the choice of a hypnotic often falls on one of the shorter acting barbiturates, whereby patients are able to awake soon after the convulsion that they may leave for home without undue delay.

A quite occasional complication is the post-convulsive confusion. Besides delaying departure time it may well constitute a threat to the practice.

Extra care is required to manage the confused patient. Besides falling he may injure others or do damage to property. He may be a fearsome spectacle and when exposed to other awaiting patients may cause them untold anxiety. Where the patient is big and strong a state of emergency can arise.

My experience in controlling this complication by the use of Valium in about 250 cases is herewith reported.

METHOD AND PROCEDURE

To a syringe containing about 5cc of a 1% solution of Sodium Brietal (Methohexitone) (for an average Asian adult weighing between 50Kg to 70Kg.) is added about a third of a cc of an ampoule of gr. 1/100 Atropine. The patient if cooperative is asked to take deep breaths and the mixture is then given intranveously immediately in order not to allow time for precipitation which begins to come on after 10 mins. or so.

This syringe is then withdrawn from the needle and another syringe containing between 30mg to 50mg (depending on the size of the patient) of Suxamethonium Chloride (Scoline) is connected to the needle and injected when the patient begins to fall off to sleep.

This second syringe is further exchanged for a third containing between 10mg to 20mg Valium depending on requirements. The Valium is not injected until fasciculations have clearly reached the toes in earnest. It is given not too slowly over a period of about 5 – 10 seconds.

The patient is then immeidately given the convulsion treatment which is effected by an Ectonus machine delivering an alternating current of 150 volts over a period of about two seconds.

On the odd occasion where no definite signs of a convulsion (even modified) is evident a repeat dose of the current is given with a longer time interval, say 1 or 2 seconds more.

These signs must include at least a sustained down-going toe with some bilaterally symmetrical clinic movements of the toes or eyelids or lips.

Very rarely (in only a few cases) was there a need to go on to deliver a third dose.

RESULTS AND DISCUSSIONS

With the above procedure a rousable light sleep follows on the tail end of the short acting anaesthesia.

An attempt may then be made to lightly awaken the patient by patting his cheek, pinching his ears or calling his name. On obtaining a response he is allowed to continue with his sleep for a while more. When it is known that his confusion is a particularly bad one he is not awakened at all but is left to sleep on.

When the sleep reaches the stage when he can turn over to one side he can then be more or less left alone or attended to by his relatives where available.

Ten to 20 minutes after the treatment the majority if not all will either awaken on their own or be aroused with ease by relatives, unless the higher doses have been used.

In this way the confusion may be totally avoided. Or course where the first dose of Valium has only led to a partial effect a higher dose is given with the subsequent treatments.

I had no occasion to exceed 20 mg in this series of cases. In my experience almost invariably where the second dose of current is required it was because of one or more of the following interfering factors having been present:–

1. electrodes had become too dry;

2. insufficient Bicarbonate powder had been used (to wet electrodes);
3. electrodes not pressed hard enough against skin;
4. electrodes slipping away due to movements during the start of the convulsions.
5. greasy skin;
6. heavily powdered skin especially with foundation cream.

The need to give a second dose (about 5% of cases) and even a third, appeared to me to be no more than in the cases when no Valium had been used.

This series of about 250 Valium cases constitue a relatively small percentage of all modified ECTs given. Generally the incidence of post convulsive confusion is not very frequent. As such it would appear unnecessary to give Valium to every patient on a preventive basis. The less drugs given the better is the maxim I subscribe to.

However, where there is a reasonable anticipation that this complication may come on then the Valium is given preventively. Among the reasons for this anticipation are exceptionally strong and big-sized patients and curiously when they are relatively not very literate – as farmers, hawkers and labourers and etc.

Of some interest is that Valium is known as an effective anti-convulsant and therefore theoretically it should not be used in a treatment procedure, where the primary purpose is to obtain a grand mal convulsion even though peripherally modified. This is particularly so when we bear in mind the work done by Ottoson et al;(1) that the therapeutic value may well be closely related to the neuronal release or grand mal convulsion produced by the current.

In my experience there has been no difficulty in the large majority of cases in obtaining a modified convulsion with Valium despite its anti-convulsant property.

Where there has been difficulty on the odd occasion almost invariably one or more of the six interfering factors were found to have been present. With their removal the second (and the rarely required third) dose of current would then unfailingly bring on the modified convulsion.

The summative effect of the subsequent doses of current no doubt would have contributed to bringing on the modified convulsion to some extent.

Reducing the amount of Scoline has also helped. This is particularly so where the patient has been under heavy sedation just prior to the treatment. It is possible that the excessive Scoline could have modified the convulsion to such a degree that any minimal muscular flickering that could have been present may have gone undetected.

It is also possible that the short time interval consequent upon my procedure between injecting the Valium and delivering the current may be a contributory cause as to why the anti-convulsant effect of the Valium did not set in.

On an average my injection time for a 10mg dose is about five seconds and a 20mg dose about 10 seconds.

As the Valium is not injected until fasciculations from the Scoline have reached a point of time that the current is ready for delivery the time interval between the end of the injection and the delivery of the current is normally not more than 5 – 10 seconds (the time taken to put aside the syringe, remove the pillow, place the electrodes on to the temporal region and press the button.)

It is possible that the anti-convulsant property of the Valium may not have sufficient time to act fully to prevent the convulsion.

Thus while it has been shown that 10mg of Valium given intravenously will often stop a grand-mal attack both clinically and electro-encephalographically within 10–30 seconds of injection(2) nevertheless it is possible that there could have been an interval of a few seconds between the delivering of the current and the full effect of the Valium coming on.

Further it may be that the stopping of an already started grand-mal seizure of spontaneous origin and the preventing of an electrically initiated grand-mal may not be one and the same thing.

Of course one could give the Valium after the convulsion, but I find this unnecessary as it involves further steps including another vein puncture or if the needle has been left in during the convulsion, the risk of trauma from unexpected jerks or the possibility of clotting within the needle if the convulsion, were to be of some considerable duration.

In my experience there appears to be no difference in the clinical response to the modified convulsion in patients with and in patients without the use of Valium.

The lack of any significantly undesirable side-effects observed by other workers was also experienced in the study.

Consequently, it may be reasonably concluded that Valium may be used quite freely and with

good effect in the control and prevention of the post convulsive confusion despite its known anti-convulsant property in the procedure aforementioned.

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PSYCHOTROPIC DRUG IN THE TREATMENT OF PSYCHIATRIC COMPLICATIONS AND SEQUELAE OF HEAD INJURY

By PRASOP RATANAKRON and PRAMOTE CHAOWASILP

Prasop Ratnakron, Professor and Director, Prasat Neurological Hospital, Puyathai, Bangkok, Thailand.
Pramote Chaowasilp, Psychiatrist, Prasat Neurological Hospital, Puyathai, Bangkok, Thailand.

The incidence of head injury has been increasing every day and the most common cause is traffic accident. Therefore the need for careful review of the medical status of brain disorders associated with trauma has become increasingly evident. On the one hand, the diagnosis, evaluation, and treatment of head injuries have become more complicated. Concurrently, psychiatric syndromes following injury to the head are of growing medical interest because of their frequency, severity, and medico-legal importance. Prior to 1940, psychiatrists and neurologists were concerned primarily with the demonstrable organic damage which resulted from head injury, and with the treatment of gross sequelae, such as epileptiform attacks and major deviations in behaviour. However, over the past 40 years, physicians have gradually come to realise that injury to the head does not necessarily involve injury to the brain. Nor does brain injury inevitably result in demonstrable disabling symptoms or mental disorders.

In brief, current medical concern is no longer restricted to the nature and residual effects of permanent brain damage. It has been extended to apply to all syndromes following head injury, which may or may not involve brain damage. And, concomitantly, clinicians have become aware of the innumerable complex and subtle variables which must be considered in connection with such phenomena. These include the patient's pretraumatic personality, including his capacity for object relationships, and the conscious and unconscious factors which governed his relations with other people, his occupational and domestic history; and, in some cases, the circumstances of the accident itself. Investigation of the patient's post traumatic behaviour is equally crucial, of course. And, in fact, although evaluation of the complex interaction of psychosocial variables is an inherent part of psychiatric practice, perhaps nowhere is a more intricate combination of organic, social, and psychological components encountered than in the stages of adaptation following head injury.

The classification of brain disorders associated with head injury is very confusing. Different authorities and text books have their own ways which are not similar. In our opinion, the classification in the Henderson and Gillespie's Text book of Psychiatry, tenth edition, 1969 is simple and more practical. According to the text book, the classification is as follows.

1. Post-concussional and post-contusional syndromes; including deliria and transient dysamnesic states.
2. Post-traumatic dementia; including personality changes and Korsakoff's psychosis.
3. Other neurological complications and sequelae; including epilepsy, aphasia and subdural hematoma which we shall not mention today.

After a minor head injury with concussion, the mental symptoms are transient, disappearing usual-