MODIFIED LEPTAZOL CONVULSIVE THERAPY

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THE use of chemically induced convulsive therapy introduced by Von Meduna in 1934 was followed by the introduction of electric convulsive therapy by Cerletti and Bini in 1937. The latter method is now of almost universal use. The main reasons for the change over were the elimination of the unpleasant aura experienced in connection with the leptazol fit and the fact that body accidents were said to be less frequent with E.C.T. There does not appear to be any evidence of greater therapeutic efficiency with E.C.T. Bini and Bazzi (1949) in summarizing statistics covering twelve thousand cases, found no difference in the remission rates in the two therapies. Dedichen (1946) indicated that the value of the two therapies in depressions was similar and Ross (1946) found this also for cases of acute excitements.

At this hospital leptazol has been retained in the treatment of psychotic excitements of all types, the clinical impression being that it was quicker and more effective than E.C.T. We were, moreover, loath to forgo the rapid quietening effect in cases of mania and catatonic excitements, and the clinical ease of induction of a fit in ward or sideroom by means of a single injection, as compared with the difficulties attending the use of the apparatus associated with E.C.T. Since the introduction of relaxants in conjunction with E.C.T. in this hospital in 1950, we have continued to use leptazol, but with the growing feeling that we were adding an unnecessary physical risk to the treatment.

Before the introduction of succinylcholine and other brief muscle relaxants, it was clearly impossible to devise a satisfactory technique in which convulsants could be used in conjunction with relaxants, because the waiting period of several minutes between the injection of decamethonium iodide or of the curare derivatives necessitates the use of evipan or pentothal, which are themselves antagonistic to the convulsant action of leptazol. In 1941 Bennett did introduce curare modified leptazol fits without intravenous barbiturates, but the method was abandoned after a few years because of the unpleasant sensations experienced by the patient during the three to four minutes' interval between the injections of relaxant and convulsant, and because, with E.C.T., a barbiturate could be given in conjunction with the relaxant.

With the introduction of suxamethonium and suxethonium compounds, it was thought that it might be possible to devise a technique for modifying the leptazol fit without the need of inducing sleep with barbiturates owing to the extremely rapid effect of these relaxants. Our first attempt was to give an

intravenous injection of suxamethonium bromide, wait 20 to 30 seconds, and then inject the leptazol through the same needle which had been left in situ. We obtained well modified fits but the patient experienced, and afterwards bitterly complained of, the feeling of suffocation normally experienced with suxamethonium when unaccompanied by barbiturates, as well as the unpleasant aura associated with leptazol. Following this we tried suxethonium followed after a shorter interval by leptazol, with a consequent shortening of the unpleasant prodromata.

The modification that we have now used for over two hundred treatments and have found effective is to dissolve 50 to 100 mgm. of the active cation of suxethonium bromide in 7 to 12 c.cm. of 10 per cent. leptazol (that is, using leptazol solution as the solvent and not water). This mixture appears to be stable for at least ten minutes, but it is our practice to use the solution immediately. It is injected rapidly, preferably through a number 18 needle, and the resultant fit appears to be as well modified as that seen in modified E.C.T. This appears to be due to the fact that the leptazol convulsion, unlike the electroconvulsion, builds up from a series of clonic jerks to a tonic spasm, giving just that amount of time necessary for the relaxant to become effective. There is none of the "black" cyanosis seen in the straight leptazol fit, although it is just as necessary to oxygenate the patient in the post-convulsive apnoeic period as it is with modified E.C.T. A positive pressure insufflator must be used for this purpose.

Using this technique we have noted that less oxygen is necessary than in the modified E.C.T. and that the patient remains a much better colour throughout, probably due to the fact that leptazol is itself a respiratory stimulant. The patient usually wakes from his fit without showing any undue excitement and often without realizing that he has already had treatment. In a few cases there has been a wild post-convulsive confusional period as sometimes occurs following E.C.T. and for this an intravenous injection of sodium amytal 0·25–0·5 gm. is necessary.

We have timed the length of the fit and of the post-paroxysmal apnoea in 70 leptazol treatments and made recordings of the B.P. before and immediately after the fit in a few cases, using leptazol and E.C.T. alternately. We have also timed the fit and the post-convulsant apnoeic period during 30 E.C.T.s given to some of these patients. There was no difference in the length of the fits induced by E.C.T. and leptazol. The average length of the apnoeic period following a leptazol-induced fit was 52 seconds. The average length of the apnoeic period following an E.C.T.-induced fit was 48 seconds.

There was a greater increase in the B.P. following a leptazol-induced fit than following modified E.C.T. The range of increase in diastolic pressure following leptazol was 0 to 46 mm.; for E.C.T. it was from 0 to 18 mm. mercury. The range of increase in systolic pressure following leptazol was from 36 to 160 mm. of mercury and for E.C.T. from 10 to 70 mm. mercury.

So far we have given treatment to patients with an age distribution of 16 to 75 years and there have been no mishaps.

Atropine gr. 1/50 to gr. 1/75 is injected hypodermically less than one hour prior to the treatment which is usually given in the morning, the patient having been without food for at least three hours. We have found it advisable to deny the patient food or drink for an hour or two following the treatment as otherwise some become nauseated and may vomit. Severe nausea can be effectively prevented by antihistaminics or by 50 mgm. of largactil given with the atropine before the next treatment.

We find that the majority of patients, male and female, are satisfactorily relaxed after 75 mgm. a.c. of the active cation of suxethonium bromide. Elderly patients and small thin women are well relaxed after 50 mgm. a.c. and muscular men may need 100 to 150 mgm. a.c. of suxethonium, although this dose does tend to prolong the apnoeic period by about 30 to 60 seconds.

The dose of leptazol for the average male patient is 10 c.cm. Again, women and elderly patients may need only 7 or 8 c.cm. Occasionally it has happened that a patient has failed to convulse, either because insufficient leptazol has been given or it has not been injected quickly enough, or because sedatives have been given to the patient within the previous twenty-four hours.

To us the most surprising finding has been that, in comparison with the straight leptazol-induced fit and the fit induced by separate injections of suxethonium and leptazol, the combined suxethonium and leptazol injection is associated with much less apprehension and far fewer reports of unpleasant aura. In fact many patients are unable, as after E.C.T., to recall anything after the injection. It has been the practice in this hospital to treat a certain number of schizophrenics with E.C.T. rather than with insulin, the former method of convulsive therapy being used more because of the lessened risk of physical injury than because of any therapeutic advantage over leptazol. In fact from our early experience of leptazol prior to the introduction of E.C.T. we are inclined to believe that it is the more effective treatment in schizophrenia. Since we can now modify the convulsions to a similar extent, we are continuing to use it in place of E.C.T.

Because of the simplicity of this method we feel that we have now such a simple technique for inducing therapeutic convulsions that it may well be used in place of E.C.T. in the occasional emergency and in situations where the more complicated apparatus of E.C.T. is not readily available. It seems to us also that the anxiety often associated in the lay mind with anything connected with electricity often deters our patients from accepting E.C.T. We refer particularly to psychoneurotic depressions.

Summary

A new technique is offered for the employment of relaxants in leptazol-induced convulsion therapy.

A comparison of this method with relaxant-modified E.C.T. is described.

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References

Bennett, A. E., *Amer. J. Psych.*, 1941, **5**, 97, 1040.
Bini, L., and Bazzi, T., "La Schizofrenia", *Nosografie e Teorie Generali*, 1949.
Dedichen, H. H., *Acta Psychiat. et Neurol.*, 1946, Supp. XXXVII.
Ross, D., *M. J. Australia*, 1946, i, 830–833.