Brit. J. Psychiat. (1963), 109, 399-403

Clinical Signs with Modified Electroconvulsive Therapy

By J. G. WHITWAM, T. MORETON and J. NORMAN

In many electroconvulsive therapy centres atropine is used to mitigate adverse effects resulting from stimulation of the parasympathetic nervous system, while the violence of muscular contractions is controlled with a muscle relaxant. It is customary to secure hypnosis with a short acting barbiturate prior to administration of the relaxant. The first report of the use of thiopentone for E.C.T. was by Rubinstein (1945), and methohexitone (Lilly 25398) was introduced by Friedman (1959). The use of succinylcholine (iodide) for E.C.T. was first reported by Holmberg and Thesleff (1951). The neurological signs during unmodified E.C.T. have been well documented (e.g. Klein and Early, 1948a, 1948b, 1949; Kalinowsky and Hoch, 1952). There is as yet no comparable detailed clinical description of E.C.T. when modified by atropine and a short acting muscle relaxant preceded by a barbiturate. This discussion is limited to some of those signs which may be of assistance in the management of therapy.

Methods

Observations were made on skeletal muscle movement, respiration, eyelash reflex, pupil reactions and the timing of the various events during E.C.T. For a period of four months up to December, 1961 the following technique was adopted. Premedication: atropine 0·9 mg. (1/75 gr.) subcutaneously 30 minutes prior to therapy. In addition, to study the effect of subcutaneous atropine on the pupils, on a number of occasions the dose was reduced to 0·65 mg. Induction: thiopentone 3 mg./kg. and methohexitone 1 mg./kg. were used alternately on each patient. As soon as hypnosis occurred, suxamethonium was injected in a dose of 0·5 mg./kg. The patients were inflated with 100 per cent. oxygen and the electrodes placed in position during the period of fasciculation resulting from the action of the relaxant. As soon as movements of the feet ceased, the jaw was held closed and the shock administered from a Reiter machine (MOL-AC II) with the electrodes positioned bi-frontally. Following the convulsion the patients were gently inflated with 100 per cent. oxygen until spontaneous respiration returned, and then placed in the right lateral position. Sixty observations on fifteen patients were made using atropine 0·9 mg. and fifteen observations on five patients using atropine 0·65 mg.

In December, 1961 methohexitone was adopted as a standard agent in a dose of 1·5 mg./kg. Twenty observations on six patients were made in this group.

In February, 1962 a further modification was introduced, atropine being given in a dose of 1 mg. intravenously shortly before the barbiturate. Thus the standard technique became: atropine 1 mg. intravenously—pause of 5–10 seconds—methohexitone 1·5 mg./kg.—pause for hypnosis to occur (average time 23 seconds)—scoline administered (0·5 mg./kg. diluted to 2 ml. with water)—patient inflated with oxygen until fasciculation in the muscles controlling the feet have ceased—shock administered (approximately 85 seconds after commencing the injection of barbiturate). Fifty-five observations on thirteen patients have been made in this group.

To investigate further the effect of intravenous atropine on the pupils, the dose of atropine was increased in a number of cases to either 1·3 mg. (ten observations on five patients) or 2·0 mg. (ten observations on five patients) using the same timing in all the groups.

All the patients were receiving long-term drug therapy consisting of an antidepressant agent (imipramine, phenelzine or tranylcypromine), together with a nightly hypnotic (either a medium acting barbiturate or di-

https://doi.org/10.1192/bjp.109.460.399 Published online by Cambridge University Press

chloralphenazone). In addition some patients were also receiving a tranquillizer (either trifluoperazine or chlordiazepoxide). The use of tranylcypromine was discontinued in the unit during the early part of this study.

Clinical Findings

1. *Atropine 0·65 mg. Subcutaneously 30 minutes before treatment*

The clinical picture was essentially similar, using thiopentone (3 mg./kg.) and methohexitone (1 mg./kg. and 1·5 mg./kg.).

Following the administration of barbiturate the patient loses consciousness; he may take a few deep breaths, after which respiration becomes shallower. The eyelash reflex is brisk, the pupillary light reflex is present and the size of the pupils variable; very often they are moderately dilated. Apnoea does not occur until the administration of suxamethonium, following which fasciculation appears, terminating in the feet. At this stage the eyes are central, the pupils moderately dilated with a brisk light reflex. The eyelash reflex and spontaneous respiration are abolished.

When the shock is administered, there is first, during the period of electrical stimulation, a modified general muscular contraction and the pupils constrict to pin-point size. Secondly, on withdrawal of the electrical stimulation, there follows a phase during which the pupils dilate and the patient usually lies still, with little or no muscular activity. This appears to correspond to the tonic phase during an unmodified convulsion. If the shock has initiated a convulsion the light reflex is found to be absent, otherwise this reflex will still be present. Finally, the modified clonic phase ensues, and the pupils are observed to exhibit spasmodic dilatation in conjunction with the skeletal muscular activity. The pupillary movements are greatest towards the end of the convulsion. (This pupillary activity may be mistaken for the light reflex during a casual inspection).

As soon as convulsive activity ceases, the pupillary light reflex returns and the pupils become smaller in size. Next, respiration returns and in the majority of instances recovery of the eyelash reflex is further delayed (usually 15–30 seconds). Within 2 to 5 minutes of the beginning of spontaneous respiration (5–8 minutes after commencing injection of barbiturate), the eyes are opened in response to command, and then full recovery of consciousness occurs within 10–15 minutes. Patients are completely recovered, "Romberg steady", and with their normal visual acuity within 30–60 minutes. Occasionally involuntary muscle movements (such as tremor, twitching or hiccough) occur after the methohexitone and before the administration of the suxamethonium (Barron and Dundee, 1962). It is of interest that on careful questioning at no time have any of the patients complained of muscle pains following therapy.

When the suxamethonium (0·5 mg./kg.) is diluted to at least 2 ml., more than adequate control of skeletal muscle activity is obtained.

2. *Atropine 0·9 mg. Subcutaneously 30 minutes before treatment*

The clinical picture was similar to that observed following atropine 0·65 mg. subcutaneously, except that the pupillary movements during the clonic phase of the convulsion were slower and less frequent.

3. *Atropine 1 mg. Intravenously*

Again the syndrome observed was essentially similar, except that, in all cases observed, the spasmodic pupillary dilatation, associated with skeletal movements during the clonic phase, was further modified with even slower, and less frequent changes in size. Following the administration of atropine, barbiturate and suxamethonium intravenously, hypnosis and apnoea occur. The pupils are slightly dilated with a brisk light reflex. As the shock is administered the pupils contract and then rapidly enlarge to a moderately dilated position. If the shock has been effective in producing a convulsion the light reflex is found to be absent. During the clonic phase, using this dose of atropine, there is some variation in the pupillary responses. Firstly, in a proportion of cases the pupil remains immobile throughout the convulsion. As soon as this is completed the light reflex reappears and the pupils return approximately to normal size. Secondly, in the majority of

https://doi.org/10.1192/bjp.109.460.399 Published online by Cambridge University Press

cases there are pupillary movements during the clonic phase. However, unlike the convulsive activity witnessed in the groups of patients who had received atropine subcutaneously, the movements in those receiving intravenous atropine consisted of more gradual variations in pupillary size and could not be mistaken for a light reflex. Again on completion of the convulsion, the light reflex returns and the pupils become smaller in size. One factor which may contribute to this variation in response, is the use of a fixed dose of atropine in patients of different diatheses.

4. *Atropine 1·3 mg. and 2·0 mg. Intravenously*

The overall clinical picture was similar to that seen in the previous group. However, further modification of the pupillary movements during the clonic phase of the convulsion was observed. With increase in the dose of atropine, pupillary movements are either further reduced or abolished. However, in one patient where no pupillary movements had been observed using 1 mg. of atropine intravenously, with an increase in dose to 2·0 mg., movements reappeared.

It has been noted that in the older patients there is less pupillary dilatation and the pupil changes generally are less consistent, the pupils tending to be sluggish in their reactions. In such patients they will sometimes remain relatively small throughout the procedure.

Discussion

The value of atropine in protecting against the adverse cardiac effects of electric shock was demonstrated in animals in the late twenties (Urquhart, 1927; MacMahon, 1929). More recently Nowill *et al.* (1954), Lewis *et al.* (1955) and Richardson *et al.* (1957) demonstrated the importance of vagal effects and their prevention by atropine during E.C.T. Apart from stimulation of the vagus nerves which occurs at the time of the shock, Nowill *et al.* (1954) have suggested that the rise of blood pressure associated with the initial tonic phase may produce further reflex vagal effects via the carotid and aortic inhibitory mechanisms. Gold *et al.* (1939), working with patients with cardiovascular disease, noted that, using intravenous atropine, the optimum effect on the pulse rate was achieved with a dose of 2·16 mg. (1/30 gr.). The maximal increase in rate occurred within a few minutes of administration, but partial recovery occurred rapidly, half the effect disappearing in 15 minutes. Morton and Thomas (1958) have shown that, following rapid intravenous injection of atropine ($\geqslant$1·28 mg. during a period of 30 seconds), tachycardia appears after 30 seconds, reaching a maximum in 2–3 minutes, almost the whole of the increase occurring in the first 90 seconds.

Lewis *et al.* (1955) recommended intravenous atropine 1·2 mg. to 2·4 mg. (1/50 gr.–1/25 gr.), immediately before or together with the barbiturate, prior to E.C.T. Clement (1962) found that atropine 1 mg. given intravenously immediately before the barbiturate, gives greater protection against slowing of the heart following shock than the same dose given subcutaneously 30 minutes earlier. Although the antisialogogue action of atropine probably takes longer to develop than the cardiovascular effects, salivation during therapy following intravenous atropine has never proved troublesome. Secretions already in the mouth and pharynx can be swallowed immediately prior to the administration of atropine.

The use of methohexitone has been adopted routinely to obtain experience with this agent. The dose was increased to 1·5 mg./kg. for two reasons. Firstly, the impression was obtained that, in some patients who had been under long-term therapy, hypnosis was barely achieved after the smaller dose of 1 mg./kg. Secondly, in our experience, using methohexitone, if for any reason the shock is unduly delayed, the recovery time is more prolonged. The clinical impression has been obtained that this barbiturate may provide protection against some of the less desirable features of E.C.T. (e.g. post-shock confusion). Further work is in progress to confirm or refute this statement; certainly the larger dose of methohexitone does not lengthen the period of recovery. Some patients with severe depression have a slow circulation time which becomes shorter as treatment progresses. Thus it would seem unreasonable to adhere to a strict time schedule during therapy. Further,

https://doi.org/10.1192/bjp.109.460.399 Published online by Cambridge University Press

a marked change in weight often occurs as a result of treatment, and it would also seem unreasonable to use a fixed dose of drugs for any given patient.

In our experience cessation of movement in the feet, resulting from suxamethonium, is the most useful sign in assessing the time of optimum relaxation. Using the technique outlined above, in which hypnosis is seen to occur before the administration of the relaxant, the average time is approximately 85 seconds from commencing the injection of barbiturate to the shock. In view of the work of Morton and Thomas (1958), it would appear desirable to administer the atropine intravenously 5–10 seconds prior to the barbiturate, to ensure that approximately 90 seconds will have elapsed in the average case before a convulsion is induced.

Adderley and Hamilton (1953) described a technique to assess the effectiveness of the shock in producing a convulsion, when completely controlled by suxamethonium. The opposite arm from the injection was occluded with a sphygmomanometer cuff, prior to the injection of the drugs, exhibiting slightly modified clonic activity during therapy. They also commented on the dilatation of the pupils and congestion of the conjunctivae as further signs, which they regarded as inaccurate in the presence of ganglion blocking agents which were administered to some of the patients in their study. Using the technique outlined in the present paper, the light reflex may be used to assess the effectiveness of a shock in producing a convulsion. The value of this sign has been previously mentioned by Thomas and Honan (1953). Alexander (1953) stated that the immediate effects of non-convulsive stimulation, shows slight dilatation of the pupils with bitempero-parietal treatment, and slight constriction with bifrontal treatment, the pupils, however, remaining reactive to light throughout the period of treatment.

It is widely accepted that during E.C.T. rapid intermittent movements of the pupils occur in the clonic phase. Klein and Early (1948b) described this spasmodic pupillary dilatation which occurs during electrically-produced epileptic fits and noted an inverse functional relation between voluntary muscles and the parasympathetic eye muscles. By instilling atropine and cocaine into the eyes of patients before E.C.T., they reached the conclusion that the intermittent pupillary dilatation which occurs during the clonic phase is due to inhibition of parasympathetic activity and not due to sympathetic excitation. The authors have observed that with an increase in the dose of atropine (either subcutaneously or intravenously), there is a modification of the pupillary movements as compared with the traditional descriptions. This is greatest following large doses of intravenous atropine when, in the majority of cases, only infrequent gradual variations in pupillary size occur, and in the rest the pupils remain immobile during the clonic phase of the convulsion.

A practical application of this apparently minor point is that, in an apnoeic patient (the result of suxamethonium and the shock), with an unreactive dilated pupil (the result of atropine and the shock), evidence of cardiac arrest can be obtained only by direct observation on the cardiovascular system.

Summary

A technique for modified E.C.T. is described, together with some clinical observations on skeletal muscle movements, pupil changes, respiration, eyelash reflex and the timing of the various events.

The authors describe two clinical signs which are of value in the management of therapy. Firstly, the cessation of fasciculation in the muscles controlling the feet can be used as a means of assessing the time of optimum relaxation following suxamethonium. Secondly, abolition of the pupillary light reflex can be used to assess the effectiveness of the shock in producing a convulsion.

Using atropine 0·65 mg. subcutaneously 30 minutes prior to therapy, convulsive activity of the pupils was observed during the clonic phase. This activity was reduced with an increase in dose of atropine to 0·9 mg. subcutaneously 30 minutes prior to therapy, and was either further reduced or abolished when a change was made to intravenous atropine $\geq$(1 mg.) immediately prior to therapy.

https://doi.org/10.1192/bjp.109.460.399 Published online by Cambridge University Press

ACKNOWLEDGMENTS

The authors are indebted to Professor G. R. Hargreaves, on whose patients these observations were made, for advice and encouragement with this work. Also to Dr. L. A. Davies for assistance with observations during the latter part of the investigation.

REFERENCES

ADDERLEY, D. J., and HAMILTON, M., *Brit. Med. J.*, 1953, *i*, 195.
ALEXANDER, L., *Treatment of Mental Disorders*, 1953, 96. Philadelphia and London.
BARRON, D. W., and DUNDEE, J. W., *Brit. J. Anaesth.*, 1962, **34**, 90.
CERLETTI, U., and BINI, L., *Boll. Acad. Med. Roma.*, 1938, **64**, 36.
CLEMENT, A. J., *Brit. Med. J.*, 1962, *i*, 228.
FRIEDMAN, E., *Dis. nerv. Syst. Monograph Supplement*, 1959.
GOLD, H., KWIT, N. T., OTTO, H., and FOX, T. J., *J. Clin. Invest.*, 1939, **18**, 429.
HOLMBERG, G., and THESLEFF, S., *Nord. Med.*, 1951, **46**, 1567.
KALINOWSKI, L. B., and HOCH, P. H., *Shock Treatments, Psychosurgery and other Somatic Treatments in Psychiatry*, 1952. New York.
KLEIN, R., and EARLY, D. F., *J. Ment Sci.*, 1948a, **94**, 581.
—— —— *ibid.*, 1948b, **94**, 805.
—— —— *ibid.*, 1949, **95**, 638.
LEWIS, W. H., RICHARDSON, D. J., and LAWRENCE, H. G., *New Engl. J. Med.*, 1955, **252**, 1016.
MACMAHON, H. E., *Amer. J. Pathol.*, 1929, **5**, 333.
MORTON, H. J. V., and THOMAS, E. T., *Lancet*, 1958, *ii*, 1313.
NOWILL, W. K., WILSON, W. P., and BORDERS, R., *Arch. Neurol. Psychiat. (Chicago)*, 1954, **71**, 189.
RICHARDSON, D. J., LEWIS, W. H., GAHAGAN, L. H., and SHEENAN, D., *N.Y. St. J. Med.*, 1957, **57**, 881.
RUBINSTEIN, H. S., *Science*, 1945, **101**, 430.
THOMAS, E., and HONAN, B. F., *Brit. Med. J.*, 1953, *ii*, 97.
URQUHART, R. W. I., *J. Indust. Hyg.*, 1927, **9**, 140.

J. G. Whitwam, M.B., F.F.A.R.C.S., *Senior Registrar, Department of Anaesthetics, General Infirmary at Leeds*
T. Moreton, M.A., M.B., B.Chir.(Cantab.), *Registrar, Department of Psychiatry, University of Leeds*
J. Norman, M.B., *Registrar, Department of Anaesthetics, The General Infirmary at Leeds*

https://doi.org/10.1192/bjp.109.460.399 Published online by Cambridge University Press