PHYSIOLOGICAL CONCOMITANTS OF ELECTRONARCOSIS.*

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ELECTRONARCOSIS is the term applied to a sleep-like condition produced by the passage of an electric current through the central nervous system.

The condition only superficially resembles sleep; the term electronarcosis is a misnomer, and electrocoma therapy might be a more apt term for this form of treatment.

The condition has been known for many years. Leduc (1902) produced a narcosis-like state in animals by means of a unidirectional pulse count. He had the procedure applied to himself, and described the experience as a dream-like state in which painful sensations were diminished.

Frostig *et al.* (1944) published a relatively safe technique of producing electronarcosis in human beings. Tietz, Thompson *et al.* (1946) first used electronarcosis in the treatment of schizophrenia. Spencer Paterson (1947) introduced the treatment to Great Britain, and reported that the first impressions of the results of treating schizophrenia by this method were favourable.

There are as yet very few reports available on the efficacy of the treatment; furthermore, little is as yet known of the precise mode of action of electrotherapeutic procedures, or the exact physiological or biochemical changes, if any, which play a part in producing therapeutic results. It is, therefore, of intrinsic as well as of practical interest to determine the physiological effects of electrotherapeutic procedures, and this paper describes some observations on physiological changes noted in a group of 30 schizophrenics undergoing electronarcosis therapy.

TECHNIQUE.

The technique adopted was that described by Spencer Paterson (1947) using the Shotter-Rich apparatus.

Premedication.

Seconal (grains 3–4½) and atropine (one to two seventy-fifths of a grain) is given about 40 minutes before starting treatment. The seconal is given to allay apprehension and reduce restlessness, and the atropine to reduce parasympathetic effects such as excessive salivation, bronchial secretion and cardiac inhibition.

In the observations to be described, control experiments were carried out to determine whether any of the changes were due to the pharmacological effects of atropine or seconal.

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An alternating current is applied through two head electrodes in two stages.

First stage consists in the passage of a current of 160–200 milliamps for 30 seconds. In the group of patients on whom the observations to be described were made the current was applied in full strength immediately; the so-called "glissando" technique of gradually increasing the current strength to the required level during the course of a few seconds was not used in this series.

Second stage begins at the end of 30 seconds with the reduction in current strength to 65 milliamps. Respiration, which was arrested during the first stage, restarts in about 15 seconds. When respiratory action becomes well established the current is increased by 15 milliamps every 15 seconds until the required degree of narcosis is produced, as is conveniently indicated by a moderate degree of inspiratory stridor. The current is terminated at the end of 7 minutes.

Motor System.

Immediately the current is switched on there is a tonic contraction of the extremities. After 10–15 seconds there is an extension of the legs with opisthotonos. The arms are flexed, sometimes adducted and sometimes abducted, with the hands clenched in the tetanic position or with thumb interposed between second and third fingers.

When the current is reduced at the commencement of the second stage clonic movements occur, qualitatively similar to those occurring in electroconvulsion, but of smaller intensity. The higher the initial current the more strong the clonic movements tend to be.

Respiratory movements follow the clonic contractions usually 40–45 seconds after the commencement of the treatment. Extensor tone in legs and flexor tone in the arms persists until the current is switched off, when muscle tone returns almost immediately to normal.

Cardiovascular System.

(a) *Heart rate.*—Immediately following the application of the initial current there is a short period of cardiac arrest followed by a slow irregular heart rate. The inhibitory effect on the heart is transient, and after a few seconds the rate returns to normal, to be followed usually by a tachycardia in the second stage, sometimes reaching 160–170 beats a minute.

The cardiac inhibitory effects of electronarcosis were less noticeable after premedication with atropine gr. $\frac{2}{75}$ths, suggesting that they were attributable to vagal overactivity. Animal experiments by Frostig (1944) in fact confirm this as the inhibitory changes were abolished by vagal section.

Electrocardiograms taken before and after electronarcosis showed no significant changes.

(b) *Blood pressure.*—It is not possible to measure blood pressure during the first stage owing to rigidity, or in the second stage until clonic movements pass off.

It can reasonably be assumed that a fall in blood pressure occurs during the stage of cardiac arrest and slow heart rate, as has been found in animal

experiments (Frostig, 1944). With the return of heart rate to normal the blood pressure rises to the pre-treatment level.

Blood-pressure readings in the second stage show an increase up to 180–220 mm. Hg systolic pressure and up to 100–150 mm. Hg diastolic pressure.

As a rule the pressure tends to remain high throughout the electronarcosis, but immediately falls to about 140–150 systolic mm. Hg when the current is switched off, returning to the pre-treatment level in 2–3 minutes after the current is switched off.

This marked rise of blood pressure is of practical importance, as it indicates the need of caution in applying electronarcosis to hypertensive patients.

The sudden drop in blood pressure occurring when the current is switched off suggested that the increased muscular tension might contribute to the high blood-pressure readings.

In order to determine whether the rise in blood pressure was attributable to increased muscular tension, tubocurarine was given to patients immediately prior to electronarcosis, to produce muscular relaxation. Blood-pressure readings in curarized patients showed a similar marked increase in blood pressure, and a similar sudden fall with cessation of the current in spite of the flaccidity of the muscles.

The changes in blood pressure, therefore, cannot be attributed to the changes in muscular tension, and are assumed to be due to sympathetic stimulation analogous to the vagal overactivity noted in the first stage.

Autonomic Responses.

In addition to the autonomic effects noted in connection with the cardiovascular system, there are many clinical changes indicative of autonomic stimulation both in the sympathetic and parasympathetic divisions, such as—

1. Flushing of face and neck.
2. Increased bronchial secretion.
3. Increased lachrymation.
4. Increased sweating.
5. Pilomotor stimulation.
6. Increased gastro-intestinal activity.
7. Enuresis.

The type of autonomic response varies with the patient, and is not always constant with each treatment.

Blood Sugar.

Blood-sugar readings were taken before electronarcosis and at regular intervals for 2½ hours afterwards. Control experiments were carried out under precisely similar conditions with the omission of electronarcosis. The results are shown in Fig. 1. There is invariably a rise in blood sugar with electronarcosis which is maintained for about 30 minutes and then gradually falls, reaching the resting level in 2–3 hours.

The degree of hyperglycaemia is not constant with each treatment, and may vary from 40–100 mgm. per 100 c.c. above the resting level. The control experiments showed no significant changes in blood-sugar level, indicating that

the changes were attributable to electronarcosis and not premedicative drugs or other causes.

The blood-sugar changes cannot be attributed to anoxaemia, as Frostig was able to show that a blood-sugar rise occurred during electronarcosis in tracheotomized dogs. It is assumed that the rise in blood sugar is another result of sympathetic stimulation occurring during electronarcosis.

Cerebrospinal Fluid.

Examination of the cerebrospinal fluid before and after electronarcosis failed to show any significant changes in ammonia content and other chemical constituents. This investigation is described in detail elsewhere (Richter, Dawson and Rees).

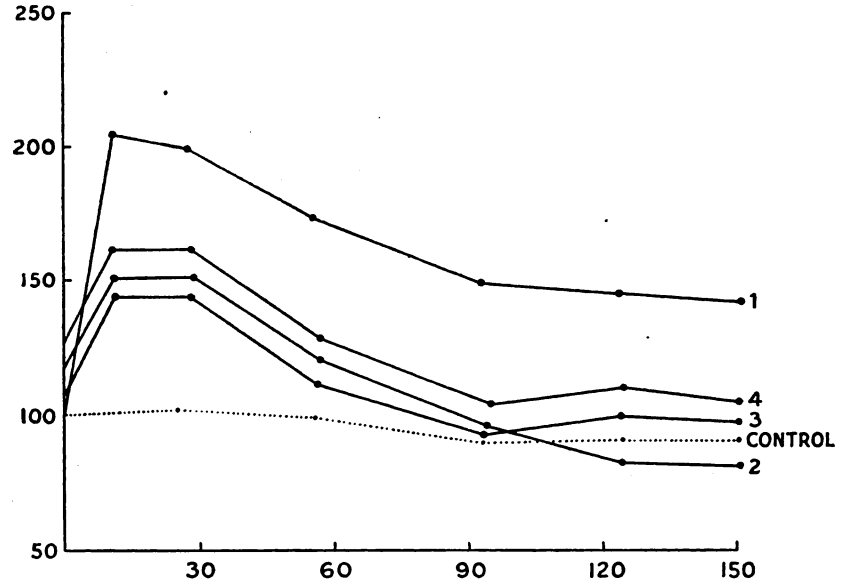

FIG. 1.—Blood-sugar curves of a patient undergoing electronarcosis.

Blood Picture.

Frostig *et al.* (1944) reported an increase in total red and white cell count accompanying electronarcosis in animals, but did not report any investigation into possible fluctuations in the various types of leucocyte.

For this study special attention was, therefore, paid to the fluctuations of the various white cells, particularly the lymphocyte, the importance of which has been established by recent work.

METHOD.

Total leucocyte and differential counts were taken before electronarcosis, immediately on termination, and at regular intervals for 2½ hours afterwards. Control experiments were carried out before starting electronarcosis treatment, in which patients received seconal and atropine premedication with blood-

counts being taken at the same time and for the same period, under identical conditions as on the treatment day. In all some 40 experiments were carried out in 10 female schizophrenics undergoing the treatment.

Results.

Total white cell count.—There is considerable fluctuation in the total white cell count after electronarcosis. Usually there is marked leucocytosis immediately following electronarcosis. There is considerable individual variation, and the changes are not constant.

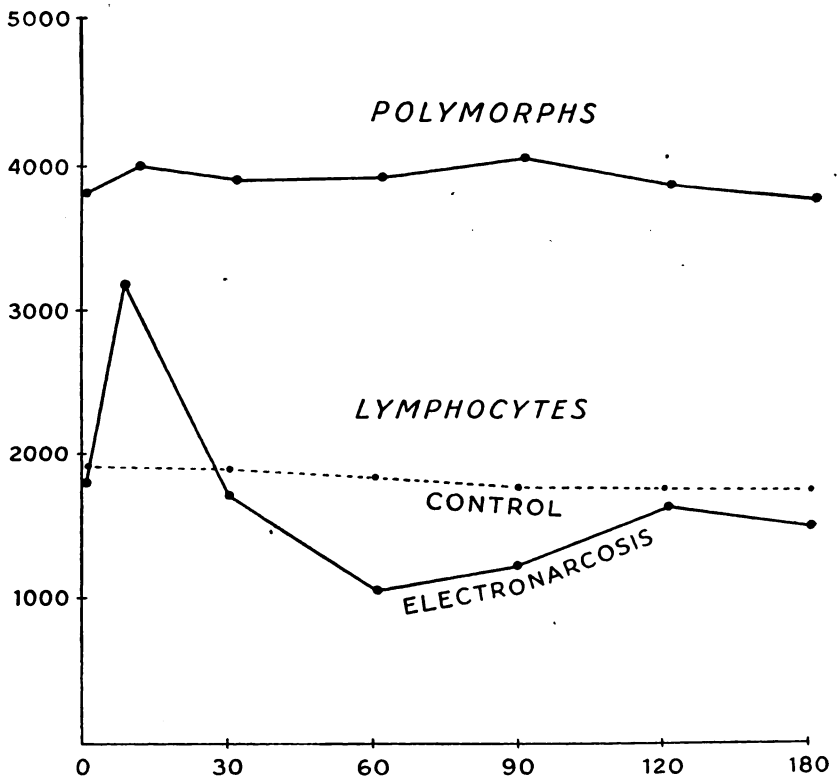

Fig. 2.—Variations in circulating lymphocytes after electronarcosis.

Polymorphonuclear leucocytes showed wide fluctuation, sometimes increasing and at other times decreasing. A composite curve for the total series shows an irregular horizontal line (Fig. 2).

Lymphocytes.—Lymphocytes in contrast to the polymorphs tended to show consistency in variation.

Fig. 2 shows a composite graph for the total series. It will be seen from the graph that there is marked lymphocytosis immediately following electronarcosis, persisting for 5–15 minutes and tending to return to the resting level by 30 minutes.

From 30–90 minutes there is a tendency for a fall in the number of circulating lymphocytes, with a gradual return to the resting level by 2 hours.

There are thus two distinct parts of the curve :

1. Lymphocytosis during the first 15 minutes after electronarcosis.
2. Lymphopenia at 1½–2 hours afterwards.

Lymphocytosis.—A similar lymphocytosis has been noticed after E.C.T. by Felici (1940), who found a lymphocytosis starting 5 minutes after electroconvulsion and lasting 30–45 minutes afterwards. Carse and Slater (1945) similarly reported a systematic rise in lymphocytes within the first 15 minutes of E.C.T. The authors do not discuss the possible explanation of their findings.

It seems probable that the mechanisms producing the lymphocytosis will be similar in both electronarcosis and E.C.T.

The following processes are possible explanations for the lymphocytosis :

1. Increase of total circulating blood elements due to depletion of blood reservoirs.
2. Abstraction of fluid from the blood.
3. Specific increase of circulating lymphocytes.

Frostig and co-workers (1944) found an increase in both red and white cells in dogs during electronarcosis, but they found that the difference in the dried weight of corpuscles before and after electronarcosis was too insignificant to account for these changes in terms of fluid loss. The findings could in fact be explained by depletion of blood reservoirs, particularly the spleen, as a result of sympathetic stimulation during electronarcosis.

This release of leucocytes from blood reservoirs, together with those from the unused capillaries, particularly of the lungs and liver, would probably explain the increased polymorphonuclear leucocyte level and possibly account for the lymphocytosis.

If the increase of lymphocytes and polymorphs were due to the same process, viz., depletion of blood reservoirs and from unused capillaries, one would expect to find a parallel fluctuation. This, however, is not so, as polymorphs showed wide fluctuation and variability, sometimes increasing and other times decreasing. The lymphocytes, on the other hand, showed a consistent and relatively more marked increase during the first 15 minutes. It seems probable, therefore, that the early lymphocytosis is mainly due to a specific increase in the number of circulating lymphocytes.

Drinker and Yoffey (1941) point out that an increase in the level of lymphocytes in the blood can follow—

1. Entry of lymphocytes into blood stream in increased numbers while the number leaving is unchanged (active lymphocytosis).
2. Entry of lymphocytes into blood in normal numbers, but with an exit numerically less than normal so that there is a retention of lymphocytes (relative lymphocytosis).

Process (2) *ex hypothesi* should be a gradual process, and therefore not applicable to our present problem of explaining the sudden lymphocytosis.

Drinker and Yoffey (1941) maintain that lymphocytosis of sudden onset is probably mechanically determined. In the case of electronarcosis a possible

mechanism would be an increased flow of lymph conveying lymphocytes formed in the lymph nodes to the blood stream mainly via the thoracic duct and right lymph duct.

It is well established that increased lymph flow can be caused by—

1. Muscular activity.
2. Increased venous pressure.

Both these conditions occur in electronarcosis, the more important factor probably being muscular activity according to Rous (1908).

Increased venous pressure together with anoxaemia and increased blood CO_2, increase lymph flow by facilitating permeability of capillary wall. Increased venous pressure would be present during the first stage, but its action and that of increased blood CO_2 is unlikely to be marked not only because of their transience, slight degree and short duration in electronarcosis, but because, as Maurer (1940) has shown, increase of lymph only occurs when oxygen saturation of the blood falls to 75 per cent. of the normal and when CO_2 is increased to 10 volumes.

In view of these considerations it was clearly desirable to carry out a control experiment under identical conditions except that the patient, instead of undergoing electronarcosis, was made to do what was estimated to be at least a comparable amount of exercise (stepping on and off chair 10 times). It was found in fact that a 30 per cent. lymphocytosis was produced during the first 15 minutes, with return to resting level in 30 minutes.

In view of these findings it is considered that the lymphocytosis immediately following electronarcosis and electroconvulsive therapy is of mechanical origin, and resulting from increased lymph flow caused mainly by muscular activity.

Lymphopenic Part of Circulating Lymphocyte Curve.

Lymphopenia may be due to—

1. Diminished entry of lymphocytes into blood stream with normal rate of exit.
2. Normal rate of entry with increased exit rate.

There is no evidence to suggest that there is a diminished rate of entry of lymphocytes into the blood stream during this ½–2-hour period after electronarcosis due to mechanical or other causes. The fact that a fall in lymphocyte level does not occur during the corresponding interval after muscular exercises is against mechanical explanation, as is the fact that the lymphopenia was unaffected when the muscular factor was diminished by tubocurarine. The reason for the fall must therefore be looked for in factors which accelerate the exit of lymphocytes from the blood stream.

The fate of the lymphocyte has been a subject of controversy among histologists and physiologists for many years. Rich (1936) emphasized 13 years ago that the complete ignorance of the function of the lymphocyte was one of the most humiliating gaps in all medical knowledge. It has now been established that the blood lymphocytes are replaced at least twice a day. The following theories have been put forward to explain this high rate of replacement:

1: *Progenitor of other blood cells.*—Maximow (1931) thought the lymphocyte

gave rise to all other types of blood cells. Yoffey believes that the excess lymphocytes are transformed erythrocytes.

2. *Excretion into lumen of alimentary canal* was a theory advanced by Bunting and Huston (1921), and has not gained general support.

3. *Return to lymph glands.*—Sjoevall (1936) maintained that lymphocytes passed out of blood stream into tissue spaces and returned to lymph nodes by lymphatics. It is difficult to account for disposal of all the lymphocytes in this way, since the number of lymphocytes in the lymph entering a gland is one-tenth that of the lymph leaving it. Herberg (1922) maintained that lymphocytes returned to the lymph gland in the afferent capillaries, and were eventually destroyed by the macrophages of the germinal centre.

Ehrlich (1946) in a review of the subject thinks Herberg's theory the most satisfactory, and could account for the exit from the blood stream of the large numbers involved. The lymph glands would therefore be the birth-place and graveyard of the lymphocyte.

Recently White and Dougherty (1946) have shown from animal experiments that the disposal of lymphocytes is controlled by the adrenal cortex. Injections of both pituitary adrenotrophin and cortical extract produced lymphopenia in animals. They have identified the responsible corticosteroid as 11-oxycorticosteroid. Six hours after the injection the germinal centres of the lymph nodes became oedematous and filled with degenerating lymphocytes and nuclear debris.

The interesting question now arises whether the fall in circulating lymphocytes noted in the ½–2-hour period after electronarcosis is due to increased adreno-cortical activity after electronarcosis. There is evidence that the diencephalic pituitary mechanism is stimulated by electrotherapy. Thus Hemphill and Reiss (1942) found an increased excretion of 17-ketosteroids after electrical convulsion. As estimation of 11-oxycorticosteroids is very difficult, it would be of interest to carry out estimation of 17-ketosteroids as an index of adrenal cortical activity after electronarcosis therapy and, if practicable, to carry out parallel lymphocyte counts in order to clarify the question. If it were found that increased excretion of 17-ketosteroids was accompanied by a fall in the number of circulating lymphocytes, it would support the view that the fall in lymphocyte level, after electronarcosis, was due to increased activity of the adrenal cortex.

These and other findings indicate the need for further research into the physiological, biochemical and endocrinological concomitants of electrotherapeutic procedures, in order to determine whether the changes have any prognostic value, or can help in the elucidation of the precise mode of action of these empirical methods of treatment.

Summary.

1. Physiological changes accompanying electronarcosis used in the treatment of a group of 30 schizophrenics are described.

2. Cardiovascular changes consist of cardiac arrest and irregular bradycardia in the first stage and a considerable rise in blood pressure in the second stage.

The cardiac inhibition is regarded as being due to vagal overactivity, and the rise in blood pressure a sympathetic effect. The rise in blood pressure is of such a degree that it might constitute a danger in hypertensive patients.

3. Rise in blood-sugar level was found to occur after electronarcosis. The degree of rise shows considerable variation with the same individual at different treatments. Available evidence suggests that the rise in blood sugar is due to the stimulation of the sympathetic-adrenal mechanism.

4. A variety of autonomic changes were noted clinically, indicating intense stimulation of sympathetic and parasympathetic parts of the autonomic nervous system during electronarcosis.

5. No significant changes were found in cerebrospinal fluid.

6. Electronarcosis was found to have marked effects in the blood picture. A polymorphonuclear leucocytosis sometimes occurred, but was not constant, and sometimes a decrease in number was found. Lymphocytes showed a consistency in fluctuation with electronarcosis. A marked lymphocytosis was found within the first 5 minutes after electronarcosis, which was maintained for about 15 minutes with a return to a normal level in 30 minutes. During the ½–2-hour period after electronarcosis a steady fall below the resting level was found.

7. The available evidence suggests that the early lymphocytosis is due to mechanical causes, probably muscular activity causing an increased entry of lymphocytes into the blood by increasing lymph flow to the blood stream. The causation of the lymphopenia is less clear. Recent work on the regulatory action of 11-oxycorticosteroids on lymphocyte blood level is discussed, and the possibility of the lymphopenia being attributable to increased pituitary-adrenocortical activity is considered.

References.

Bunting, C. H., and Huston, J. (1921), *J. Exp. Med.*, **33**, 593.
Carse, J., and Slater, E. (1946), *J. Neurol., Neurosurgery and Psychiat.*, **9**, 1.
Drinker, C. K. and Yoffey, J. M. (1941), *Lymphatics, Lymph and Lymphoid Tissue*, Harvard.
Ehrlich, W. E. (1946), *Ann. N.Y. Acad. Sc.*, **46**, 823.
Felici, M. (1940), *Riv. Sper. Frenial.*, **64**, 513.
Frostig, J. P., Van Harreveld, A., *et al.* (1944), *Arch. Neurol. Psychiat.*, **51**, 232.
Hemphill R. E., and Reiss, M. (1942), *J. Ment. Sci.*, **88**, 559.
Kalinowsky, L. B., and Hoch, P. H. (1947), *Shock Treatments.*
Leduc, S. (1902), *Comp. Rend. Acad. Sci.*, Paris, **135**, 199.
Maurer, F. W. (1940), *Am. J. Physiol.*, **13**, 331.
Maximow (1931), *Text-book of Histology*, Philadelphia.
Paterson, A. S., and Milligan, W. L. (1947), *Lancet*, **2**, 198.
Rich, A. R. (1936), *Arch. Path.*, **22**, 228.
Rous, F. P. (1908), *J. Exp. Med.*, **10**, 236.
Sjoevall, H. (1935), cited by Ehrlich (1946).
Tietz, E. B., Thompson, G. N., *et al.* (1946), *J. Nerv. Ment. Dis.*, **103**, 144.
White, A., and Dougherty, T. F. (1945), *Endocrinology*, **36**, 16.