# ELECTRIC CONVULSION TREATMENT OF THE PSYCHOSES

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presented by

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# Introduction

It has long been known that any severe physical illness, or shock whether traumatic or psychological, occurring during the course of a psychosis might occasionally lead to a remission of the psychotic symptoms. Indeed Hippocrates 460-357 B.C. appears to have been aware of this when he observed that "Haemorrhoids appearing in melancholic and nephritic affections are favourable." (Aphorisms). The modern conception of what is now called "shock therapy" however dates from the observation of Weigert (1871) that a local necrosis or injury usually starts reparative processes in excess of requirements or as Pfluger (1877) expressed it "Injury is the incentive to removal of injury." The subject grew up around the treatment of specific bacterial infections with vaccines and sera, but it was soon found that these could be applied non-specifically to the treatment of other diseases. Among the first to apply the method in the treatment of the psychoses was Wagner von Jauregg when he began to treat general paralysis of the insane with malaria in 1917. This was 30 years after he had postulated in 1887 his general theory concerning the influence of febrile illnesses on the course mental disorders.

To-day in the field of psychiatry the term "shock therapy" is used with somewhat narrower connotation and denotes the

induction of hypoglycaemic coma by the administration of insulin (Sakel 1933), or the induction of epileptiform convulsions by such drugs as cardiazol, triazol, picrotoxin, or ammonium chloride. Recently, however, Cerletti and Bini (1938) described a method of inducing the convulsions by means of electricity and already papers by Fleming, Golla, and Walter (1939); and Golla, Walter and Fleming (1940) and by Shepley and McGregor (1939, 1940) have appeared in this country. These papers dealt mainly with the apparatus, and the application of the method. In this thesis it will be my purpose to show that the electrical method is safe, that its therapeutic efficacy is equal to that of the pharmacological convulsants, and while possessing the advantage of the latter it has few, if any, of their disadvantages.

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# O U T L I N E

## CONVULSION THERAPY

The development of convulsion treatment appears to have been the logical outcome of earlier observations of antagonism between schizophrenia and epilepsy. Nyirö and Jabolonszky (1929) made the observation that in their institution the recovery rate among epileptics was 1.05%, but when schizophrenia was connected with the epilepsy 16.05% of the epileptics recovered. Later Müller (1930) described two cases of schizophrenia in which the symptoms disappeared after the onset of idiopathic epilepsy, and Glaus (1931) found on examination of 6,000 schizophrenia subjects 8 only in whom the combination of schizophrenia and epilepsy existed. In 4 of these the schizophrenic process did not make its appearance until the epilepsy had ceased to manifest itself. Steiner and Strauss (1932) also in an examination of the records of 6,000 subjects of schizophrenia found only 20 with a history of epileptic seizures, and these they considered such a rarity that they doubted the accuracy of the diagnosis. Nyirö in 1932 unsuccessfully attempted to make use of these observations of apparent antagonism between schizophrenia and epilepsy by transfusing schizophrenic subjects with blood from subjects of epilepsy. Meduna (1935) considered that the sixteenfold increase in the recovery rate, noted by Jabolonszky

and Nyirö among schizophrenic patients when epilepsy became superadded, could not be due to coincidence but to some form of "biological antagonism" between the two diseases. It is upon this hypothesis that he bases his treatment. Since then Shepley (1938) has reported marked improvement in a chronic schizophrenic patient, following a spontaneous epileptiform convulsion. This patient, a female, whose condition had been one of marked mental and physical deterioration improved under convulsion therapy, instituted after the occurence of the spontaneous fit, to such an extent that she became employable and cooperative. This improvement, unfortunately has not been maintained although she has not relapsed to her former condition of self-absorption and complete withdrawal from reality.

Before attempting to induce convulsions in human beings, Meduna carried out a series of experiments on animals, the results of which he published in 1933, without, however, stating their object. The substance adopted for this work was 25% camphor in oil, a drug well known to the earlier workers in this field to possess convulsant properties, and as the monobromide of camphor was much used by Muskens (1928) in his experimental work on Myoclonic epilepsy. In the course of these experiments, Meduna established the fact that there was no relationship between the dose of camphor required to induce a fit and the body weight of the animal. This observa-

tion he later found to hold true for the human subject. When he was satisfied that no damage resulted to central nervous system of the animals Meduna proceeded to his first human experiments. These he made on apparently hopeless patients who had been under institutional care for years. Convulsions, he found, could readily be induced by means of the camphor in oil used for his animals experiments, but such large doses had to be given intramuscularly that considerable pain was caused, and occasionally abscesses developed in the gluteal muscles, a complication which often necessitated temporary discontinuance of the treatment. A further disadvantage was the excretion of the camphor by the lungs which often caused considerable nausea and vomiting. These and other difficulties impelled Meduna to seek a more suitable agent for inducing the convulsions. This, he found in Cardiazol a substance which had been shown by Hildebrandt (1926) to possess convulsant properties. At first Meduna employed this in a 10% aqueous solution, later increasing the strength to 20%, but finding that this caused sclerosis of the veins, by which route it was administered, he reverted to the 10% solution. It is in this form that the method has been adopted by most psychiatrists practising the method.

In this country, perhaps because the majority of alienists were preoccupied with the Hypoglycaemic treatment

of Sakel (1934) Meduna's early publications received but little notice. At the Munsingen conference in 1937, however, the method was favourably commented upon, since when, it has been widely accepted and applied in the treatment of mental disorders, more particularly of schizophrenia.

In the early reports on the method attention was mainly directed to a discussion of the therapeutic successes and little or no mention was made of the complications and dangers that might arise as a result of, or during the course of treatment. More mature consideration, nevertheless, has revealed that the method is not without danger, and the later reports include accounts of many and varied complications. In the report of the Board of Control for 1938 (England and Wales) 10 deaths are recorded as a result of cardiazol treatment. No analysis of these figures is given.

In an effort to overcome the disadvantages of cardiazol, Mayer Gross and Walk (1938) published an account of their experiences with Triazol (Azoman), and Low, Blaurock, Sachs, Wade and Ross (1939) with Picrotoxon. The use of ammonium chloride as a convulsant has been reported on by the Italian workers Bertolani (1939) Maffa (1939) and Baraldi (1939) and also by Dax (1940) in this country. While advantages are claimed for all these substances neither picrotoxon, nor ammonium chloride have undergone extensive trial in this country. As

regards triazol the main advantages claimed for it by most workers are that it does not cause sclerosis of veins, and can be given in quite small doses to patients whose veins are inaccessible. The promise of eliminating the subjective sensations of impending annhilation or dissolution, so common after cardiazol exhibition, has not been fulfilled.

## THE THERAPEUTIC VALUE OF CONVULSION TREATMENT

It is very difficult, owing to differing standards by which remissions are judged, to form an exact estimate of the value of convulsion treatment. Kennedy (1940) in a review of the literature dealing with convulsion therapy observes that remission rates of from 7 - 100% have been claimed for the method. While the latter figure may be regarded as optimistic, the former is lower than the average remission rate among patients treated by ordinary routine hospital methods, and was in all probability obtained from the treatment of a group of mixed, or very chronic patients. Without details of the patients' heredity, prepsychotic personality, age at the onset of the illness, and type of illness it is difficult to make a comparison of results, for it is well known that spontaneous remissions are more common among patients with sound heredity, and also among patients whose illness is of the catatonic form, and more particularly is this the case in the first year of the illness. The presence of exogenous precipitating factors is also regarded as of good prognostic import. Despite this lack of uniformity of standards by which comparison can be made it is possible, by comparing the so called spontaneous remission figures , with those obtained by cardiazol treatment, to form some opinion of the value of the method. Most of the convulsion treatment results that have been published are

those resulting from the treatment of schizophrenia, and it is therefore convenient to make the comparison with the spontaneous remission figures for the same disease.

In a review of the literature for the years between 1912 - 1938, Hunt, Feildman and Fiero (1938) found that the spontaneous remission rate among 1,741 schizophrenics was 196 or 11.3%. Their figures are reproduced in Table I.

| Author | | No of Cases | No of Remissions | Percentage |
|---|---|---|---|---|
| Stearne | (1912) | 395 | 51 | 13% |
| Rosanoff | (1914) | 213 | 25 | 11 |
| Bond | (1921) | 20 | 0 | 0 |
| Bond | (1921) | 34 | 3 | 18.8 |
| Pollock | (1925) | 19,927 | - | 6.4 |
| Stricker and Willey | (1924) | 186 | 38 | 20.4 |
| Levin | (1931) | 592 | 35 | 5.9 |
| Wooton et Ae | (1935) | 95 | 18 | 20.0 |
| Whitehead | (1937) | 90 | 14 | 15.5 |
| Bond and Braceland | (1937) | 116 | 12 | 10.3 |
| TOTAL (Excluding Pollock's figures) | | 1,741 | 196 | 11.3% |

Table I showing the number of cases and spontaneous remission rate among untreated cases. Hunt, Fieldman and Fiero. (1938).

Strecker (1938) in 581 patients found that 23.6% had made a spontaneous recovery. Rees Thomas and Wilson (1938) quote remission rates of from 15 - 77% occurring spontaneously.

Their figures are shown in Table II.

| | |
|---|---|
| Fromentz | 15 % |
| Faurbye | 42% |
| Lange | 77% |
| Ederle | 26% |

Table II Perentage spontaneous remission rates among untreated cases. Quoted by Wilson and Thomas. (1938).

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No details are given of the criteria by which these remissions were judged, nor is any information given regarding the age, type, or duration of the illness, and one is forced to the inference that the very high figure of 77% was obtained from among very early cases. Ravn (1934) has reviewed the results obtained by early workers in this field. His observations covered the years 1903 to 1911 and he found that the percentage cures recorded varied from 2 - 30%. His figures are reproduced in Table III.

| Author | Year | Type of Case | Number of Cases | Percentage Cures |
|---|---|---|---|---|
| Kraeplin | - | Catatonia | - | 13 % |
| Meyer,E | - | Hebephaenia | - | 8 % |
| Meyer,E, | 1903 | Dementia Praecon | 46 | 30 % |
| Meyer,E, | 1909 | " " | 142 | 21.8 % |
| Albrecht, | 1905 | " " | 693 | 2.0 % |
| Kahlbaum | 1902 | Catatonia | 27 | 30.0 % |
| Ralcke | 1910 | " | 117 | 15.8 % |
| Schmitt | 1911 | Dementia Praecon | 455 | 16.2 % |

Table III Spontaneous remission rates obtained by earlier workers. Ravn (1934)

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Taking the average of all these figures the spontaneous remission rate among a large number of patients suffering from schizophrenia is approximately 23.5%. This figure includes all groups of the disease, and is irrespective of the duration of the illness.

Meduna (1935), Cook (1938) and Low (1939) and his co-workers have published remission figures obtained by them among schizophrenic patients treated with cardiazol. These are reproduced in Table IV which shows that the total number of

Showing some of the published remission rates among cases of schizophrenia treated by induced convulsions.

| Author | Total Number of Cases | Duration of illness | No. of Remissions | Unchanged |
|---|---|---|---|---|
| Meduna (1935) | 110 | Less than 1 year | 39 (90%) | 40 |
| | | 1-2 years | 9 (50%) | 9 |
| | | 2-5 years | 6 (35%) | 17 |
| | | Over 5 years | - | 26 |
| | | | 54 (99%) | 56 |
| Cook (1938) | 45 | Under 1 year | 7(77.7%) | 2 |
| | | 1-2 years | 6(60%) | 4 |
| | | Over 2 years | 11(42.3%) | 15 |
| | | | 24(53.3%) | 21 |
| Low & Co-workers (1938) | 43 | Under 1 year | 5(38.4%) | 8 |
| | | 1-2 years | 1(10%) | 9 |
| | | Over 1 years | 2(10%) | 18 |
| | | | 8(18.6%) | 35 |

Table IV showing the remission rates obtained by treatment with cardiazol.

patients treated was 198 and that of that number 86 or 43.5% approximately recovered. Reitman (1939) has analysed the results of cardiazol treatment in Europe and America and found, that of a total 2,011 schizophrenic subjects whose illness was of less than 18 months duration 51% recovered. A less favourable figure was however obtained by Meduna and Friedman (1939) who carried out a similar analysis of the results of treatment of 2,937 patients and obtained a remission rate of 25.1%. Closer inspection of the figures published by Meduna and Friedman, however reveals that, in America, when the duration

of the illness is taken into account 60.55% of those whose illness was of 6 months of less duration recovered, and 36.8% when the illness was of 6 - 12 months, while only 8.36% recovered when the illness had persisted for more than 1 year. In Europe, among cases of less than 18 months duration a remission rate of 49.66% was obtained, and 30.37% from among all cases. The results obtained by Meduna and Friedman are reproduced in Table V.

Showing analysis of the results of convulsion treatment as obtained by Meduna and Friedman.

Total No.of Cases 2,937
Total No.of full remissions 737 (25.1%)

| America | 1,465 cases | No.of Remissions. | No.of Improvements |
|---|---|---|---|
| Less than 6 months | 201 | 128 (60.55%) | 40 (20%) |
| 6 - 12 months | 201 | 74 (36.8%) | 47 (23.13%) |
| over 1 year | 1,054 | 88 (8.36%) | 398 (37.7%) |

Europe 1,472 cases

Acute and sub-acute cases of less than 18 months duration ... Remission rate 49.66%

All cases .... Remission rate 30.37%

Table V showing the remission rates among schizophrenic patients treated by convulsion therapy as obtained by Meduna and Friedman. (1939)

Comparison of these figures with those obtaining among non-treated patients suggests that the prognosis of schizophrenia is little if at all improved by convulsion treatment

when the disease has persisted for more than 2 years. If, however, an average is taken of the all remission rates for all stages of the disease we find that an average of 37.1% of patients recovered under convulsion treatment as compared with the average of 23.5% from among untreated cases. Table VI shows this comparatively. When the duration of the illness is taken into consideration the prognoses is considerably improved, for with the exception of the figures obtained by Low and his co-workers all are considerably better than the 23.5% obtained among untreated cases. This is further illustrated by the work of Bain (1940) who treated 16 chronic schizophrenic patients with cardiazol. In some of these there was considerable initial improvement, which however was not maintained and relapse to the former state soon occurred. In others the mental state became worse during the course of treatment.

It seems clear then, from this brief analysis, that the prognosis of schizophrenia is improved by about 14% when all types and stages of the disease are treated. On the other hand if treatment is undertaken in the early stages of the illness the results are very much better and convulsion treatment can be regarded as improving the outlook in this disease which as Blair (1940) has quoted is well termed "The sickness that destroyeth in the noon-day."

| Author | Average % remissions among untreated cases. | Author | Average % remissions among convulsion treated cases. |
|---|---|---|---|
| Hunt, Fieldman & Fiero (1938) | 11.3. | Meduna (1935) | 49.0 |
| Strecker (1938) | 23.6 | Cook (1938) | 53.3 |
| Rees Thomas and Wilson (1928) | 40.2 | Low et Al (1938) | 18.6 |
| Rav (1934) | 19. | Meduna and Friedman (1939) | 27.7 |
| Average of Total | 23.5% | Average of Total | 37.1% |

TABLE VI comparing the average percentage remission rate among treated and untreated cases of schizophrenia. All types and at all stages of the disease.

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## COMPLICATIONS OF CARDIAZOL AND TRIAZOL CONVULSIONS

The complications that are most frequently encountered during cardiazol and triazol treatment may be considered from subjective, and objective aspects. On the subjective side the chief concern is the fear and apprehension that is aroused in the majority of patients after the first few injections. This is due to sensations, usually terrifying, that occur in the interval between the injection, and the onset of the fit, during which time the patient is fully conscious. In this hospital where cardiazol was in use from January 1938 until November 1939 when it was supplanted by the electrical method, in one year, 1938, eighteen male patients undergoing treatment made attempts to escape. These were all responding favourably and were accommodated in convalescent villas at some little distance from the main hospital buildings, but such was their apprehension that the approach of each treatment day induced them to attempt escape. These attempts became such a nuisance that until the course of injections was completed, all patients undergoing treatment were thereafter housed in the hospital buildings. Neustatter and Freeman (1939) were so exercised by this apprehension as to administer a general anaesthetic before making the injection. Others among whom, Cook (1938) administered combinations of morphine and hyoscine to overcome this complication.

On the objective side complications may arise during the fit, in the post-accessual period, or at a later date. Of those arising during the fit fractures and dislocations are the most common. Of the fractures, the most frequent is a compression fracture of the thoracic vertebrae. Fractures of the femora and other bones although less common have also been reported. In this hospital two patients sustained a fractured femur. These occurred in the clonic stage of the fit. Of the dislocations the most frequent is a dislocation of the jaw, and in my own experience with cardiazol this occurred so frequently and was so readily reduced in the flaccid period at the end of the fit that I ceased to regard it as a complication. No patient was ever known to suffer discomfort or pain from its occurrence. Dislocation of the shoulder has also been reported.

The frequency of fractures as reported by various writers is shown in Tables VII, VIII., and IX.

Fear has been expressed by many writers that as a result of these vertebral fractures Kummel's post traumatic disease might develop. This fear, however, appears unlikely to materialise for Cook, Comerford and Sands (1940) found in the radiographs of 100 epileptics, 16 showing varying degrees of deformity of the vertebral bodies. Of this number, 7 showed a severe degree of compression fractures. All these injuries

| Author | | No of Cases treated | No.X-Rayed | No. of Fractures |
|---|---|---|---|---|
| Carp,L. | (1939) | 687 | Not given | 2 |
| Bennett & Fitzpatrick | (1939) | 17 | 8 | 8 |
| Polaten et al | (1939) | 58 | 51 | 22 |
| Wespi, J. | (1938) | - | - | 1 |
| Palmer, H.A. | (1939) | 20 | 20 | 6 |
| Stalker,H. | (1938) | - | - | 1 |
| Blair, D. | (1940) | 120 | 20 | 6 |
| Kraus & Viersma | (1940) | 51 | - | 5 |
| | Total | 953 | 99 | 51 |

Table VII showing the frequency of vertebral fractures following cardiazol or triazol convulsions.
Kraus & Vieroma (1940) also report the occurrence of infraction without evident compression in 3 cases.

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| Author | | No.of Cases Treated | Unilateral | Bilateral |
|---|---|---|---|---|
| Kersteus, | (1938) | - | 0 | 1 |
| Pameijer,J.H. | (1938) | 1,200 | 2 | 3 |
| Nightingale, G. | (1939) | 44 | 1 | 0 |
| Goldstein et al | (1938) | 102 | 1 | 0 |
| Walter & Mayer Cross | (1938) | 61 | 1 | 1 |
| Beckenstein | (1939) | 695 | 2 | 0 |
| Pollock,H.M. | (1939) | 1,140 | 5 | 0 |
| Bellinger, C.H. | (1939) | 538 | 1 | 0 |
| Hausen & Bennett | (1939) | - | 4 | 0 |
| Carp. L. | (1939) | 687 | 5 | 0 |
| Somers & Richardson | (1939) | 1 | 0 | 1 |
| Blair,D. | (1940) | 95 | 1 | 1 |
| W.P.H. (Unpublished) | | 250 | 2 | 0 |
| | Total | 4813 | 25 | 7 |

Table VIII showing the frequency of Femoral fractures during cardiazol or triazol convulsions.

| Author | | No.of Cases | No. of Fractures Humerus. | Maudible. | Scapula |
|---|---|---|---|---|---|
| Pameijer | (1938) | 1,200 | 3 | 1 | 3 |
| Goldstein et al | (1938) | 102 | 3 | 0 | 0 |
| Pollock H.K. | (1939) | 1,140 | 2 | 1 | 0 |
| Carp.L. | (1939) | 687 | 5 | 1 | 0 |
| Kraus,G. | (1938) | - | 1 | 0 | 0 |
| Briner, O. | (1938) | 111 | 0 | 0 | 1 |
| Good,R. | (1939) | 0 | 1 | 0 | 0 |
| | Total | 3,240 | 15 | 3 | 4 |

Table IX showing the frequency of other fractures following cardiazol or triazol convulsions.

were entirely symptomless and moreover no signs of Kummel's disease were observed. Kraus (1940) in 21 epileptic patients found 1 certain and 1 doubtful vertebral injury, again without signs of post traumatic disease being present. In connection with this question it is of interest to note the opinion of Watson Jones (1940) who states that "There is probably no such condition. Kummel's disease is simply an overlooked fracture of the spine in which failure to immobilise the injury has caused progressive wedging and absorption of bone."

Respiratory complications have been reported as occurring during the course of cardiazol treatment by several workers. Pameijer (1938) reported a tendency for latent tuberculosis to be lighted up as a result of treatment. Harris (1938), Zeifert (1939) and others have reported the occurrenee of pulmonary abscesses. Zeifert believes that these are due to pulmonary emboli, the emboli being formed in the syringe by the coagulation of the blood withdrawn into the syringe for the purpose of making certain that the needle is properly in the vein. To overcome this danger he adds 2.5% sodium citrate to the solution of cardiazol. Other workers are of the opinion, which seems the more likely explanation, that these abscesses are caused by the aspiration of infected material from the mouth and throat. Ross (1939) reported the occurrence in 3 patients of pneumonia as a result of cardiazol treatment. Arrest of respiration has also been reported but

is not regarded as serious being readily overcome by pressure on the chest wall.

The occurrene of cardiac complications was at first unsuspected. Meduna (1935) had electro-cardiographic examinations made in 10 of his patients whose psychosis had remitted as a result of treatment. Of this number one only showed a record deviating from the normal, and Meduna concluded, despite the fact that no examination of this nature had been made before treatment, that no damage resulted to the cardio-oascular system as a result of treatment. Since then however, several workers, Geraudel (1938) Dick and McAdam (1938), Pameijer (1938) Schmitt (1939) and others have recorded cardiac abnormalities arising during the course of treatment. Geraudel reported temporary inversion of the T wave in the electrocardiogram. This he regarded as a sign of myocardial distress and an indication for termination of the treatment. Dick and McAdam reported cardiac complications in 4 patients. The first of these developed reduplication of the second mitral sound, the second, complete arrhythmia of the pulse with displacement of the apex beat one inch to the left. Irregularity of the pulse also developed in the third patient, who had a previously existing left sided preponderance. This lasted for 12 hours and the electrocardiogram showed partial flutter. The fourth patient developed a partial heart block after the second injection. Pameijer recorded several cases of myocardial

weakness developing during the course of treatment. Two of his patients subsequently died. Schmitt recorded several cases of sinus tachycardia, and extra-systoles of both auricular and ventricular origin. These latter occasionally gave rise to a pulsus bigeminus. In 10 of his patients the electrocardiagraphic records before treatment were normal. He concluded therefore that cardiazol is capable of inducing minor lesions of the myocardium. Cardiac complications have also been reported as a result of triazol administration by Molony and Conlon (1939) who reported the occurrence of temporary heart block, praecordial pain and tachycardia with extra systoles enlargement of the heart to the left. These workers concluded that because of the longer time for a convulsant dose of triazol to be excreted it was more likely to damage the myocardium than cardiazol. Good (1940) has reported the occurrence of cardiac anomalies in 42 patients undergoing cardiazol treatment. These he divided into the following 4 groups.

1. Fixed disturbances of rate.
   (a) tachycardias
   (b) bradycardias
2. Irregularities
3. Mixed
4. A heterogenous group.

With Malony and Conlon (1939) Good agrees that it is the fit that leads to the production of the cardiac lesion, but adds

the proviso of an anoxaemic heart muscle unaccustomed to such strain as an epileptiform fit would place on it. In support of this Good records that 10 of his patients treated with sub-convulsive doses of cardiazol did not develop any such cardiac anomalies.

Disturbances of the Heamopoietic System

Epstein (1939) has recorded the occurrence of fatal aplastic anaemia in a female patient during a course of cardiazol treatment. This patient whose psychosis was of 3 months duration collapsed after the 24th injection and developed purpura of the skin and mucous membrane. Morphological examination of the blood revealed a progressive anaemia with failure of the bone marrow to regenerate. No other drugs were being administered at the time of this occurrence and Epstein concluded that the condition was due to cardiazol administration. Disturbance of the reticulo-endothelial system has also been recorded in three patients undergoing cardiazol therapy, by Wender and Epstein (1939). In each of these cases the disturbance was characterised by an alteration in the normal white cell relationship and enlargement of lymph glands. There was no pyrexia. In my own experience with cardiazol, one patient a female aged 20 years developed large purpuric eruptions of the skin. These persisted for

2 - 3 days and recurred after each succeeding injection. In this patient no alteration was noted in the normal blood cell relationship and I concluded that they were due to rupture of skin capillaries during the fit. No improvement occurred in her mental condition and she has subsequently undergone treatment by the electrical convulsion method without any recurrence of the purpuric spots, a fact suggesting that they may have been a direct result of the cardiazol injections.

Vomiting

Vomiting is a frequent and often distressing complication of cardiazol treatment. It almost always occurs in the post-accessual period, but when triazol has been administered it may occur before the onset, particularly when the drug has been given intramuscularly. This occurrence can, however, often be anticipated and prevented by premedication with atropine, and apart from distressing the patient, ordinarily gives rise to little difficulty. When, however, vomiting occurs as a complication of summation therapy, Georgi and Strauss (1937), it may result in the complete loss of the customary nasal feed of sucrose given to terminate the coma. In these circumstances it is usually necessary to introduce glucose by intravenous injection to prevent relapse into coma.

Nervous and Mental Complications

In the early days of cardiazol treatment it was feared

that as a result of artificially induced fits spontaneous epilepsy might develop. This, however, appears unlikely to materialise for although Hobson, quoted by Wilson and Thomas (1938), Harris (1939), and Thorpe (1939) have reported the occurrence of spontaneous epileptiform seizures after the induction of convulsions by cardiazol, these ceased after a short time and have not since recurred. In Hobson's case 5 spontaneous fits occurred after the course of cardiazol treatment had been terminated, These ceased after a month and the patient whose mental condition had improved relapsed. Harris reported two cases in which one spontaneous convulsion occurred some time after treatment had been discontinued, and Thorpe the occurrence of one spontaneous fit seven days after the induction of a cardiazol convulsion.

Changes in behaviour as a result of treatment with cardiazol have been reported by Harris (1938), Nightingale (1938) and others. Harris observed that stuporose patients failing to respond favourably to treatment tended to pass into a state of catatonic excitement. This, however, may not have been due to treatment for it is well recognised that schizophrenia may spontaneously alternate between excitement and stupor. Harris' patients had not previously shown alternation and he inferred that it was brought about by the stimulatory effect of the cardiazol injection. Nightingale records that

two of his patients became hypomanic and sustained Colles' fractures necessitating the discontinuance of the treatment. This tendency to excitability was found by Nightingale to be a troublesome feature of cardiazol treatment, and he noted that a number of previously lethargic patients become mischievous and interfering.

Impairment of memory has been noted by Dynes (1939). Tooth and Blackburn (1939) and others. In 4 of seven patients reported by Dynes as showing marked intellectual deterioration there was marked loss of memory.

Severe personality changes have also been reported by Berrington (1939) as due to convulsion treatment.

Sclerosis of veins frequently gives rise to difficulties during cardiazol treatment, and with this there is some risk of part of a thrombus becoming detached at subsequent injections with pulmonary abscesses resulting. This difficulty has been overcome with triazol, which is given in much smaller doses and shows little tendency to cause thrombosis, and furthermore it is equally efficacious as a convulsant when given intramuscularly and therefore could be used after the veins had become occluded by cardiazol.

The tendency for patients to become tolerant to cardiazol is a disadvantage which often gives rise to distressing sequelae, for when the injection fails to induce a fit extreme

agitation, restlessness and excitement may result. When this occurs it is desirable to give a further injection of increased dose at once so as to induce a fit as otherwise the patient tends to remain in an over excited and inco-operative state for the remainder of the day.

## THE PHYSICAL PROPERTIES OF ELECTRICITY

Before proceeding to examine the literature dealing with the physical and pathological results of contact with electric currents it is necessary to consider briefly some of the physical properties of electricity for it can be shown that it is, to a certain extent, upon these properties that the biological effects depend.

### Voltage.

The unit of pressure or electromotive force is the volt. It is customary to describe voltage as being of high or low tension. Voltages of over 1000 volts are arbitrarily considered as high tension. The high tension cables of the "grid" carry 132,000 volts over long distances.

There is a belief that low tension circuits such as those used for domestic and industrial purposes are not dangerous to life. This however is erroneous for in America during the years 1918 - 1926, 34 deaths occurred from contact with circuits of 110 - 250 volts, and 18 from contact with 110 - 550 volts. In the years 1912 - 1919, 27 deaths due to contact with low tension circuits were recorded. (Journal of Industrial Hygiene, 1928, Vol.10, pg 115). Yet paradoxically, people have been known to survive contact with the grid, and Fetterman and Smiley (1937) have reported the recovery of a boy after contact with a circuit of 4,000 volts.

## Current

The unit of current is the ampere. For biological purposes however this is much too large and the milli-ampere, Ma, is the unit commonly adopted. This is equal to 0.001 ampere. Quite small currents of the order of 1 milli-ampere are readily detected by sensitive people, and to most a current of 10 milli-amperes is painful. In connection with the effects of current we see the same paradox noted in the case of voltage, namely that quite small currents may seriously affect the organism while relatively large currents may have little or no effect.

## Type of Current

In this country the type of current most commonly in use is alternating current at 50 cycles per second and a pressure of 230 volts. In some districts direct current is still in use. The reason for the general tendency to adopt alternating current is the ease with which it can be carried long distances by the grid cables, and readily transformed to domestic and industrial requirements by step-down transformers. This cannot be done with direct current which must be generated in or close to the district in which it is required.

In general, alternating current is more dangerous than direct. Prevost and Battelli (1899) showed that the minimum voltage of direct current that would induce ventricular

fibrillation in dogs was 60 - 70 volts, while the same result could be obtained by an alternating current of 10 - 20 volts. With high voltages, however, direct current may be more dangerous than alternating. Thus, in rabbits Prevost and Battelli found that an alternating current of 50 volts could be passed from head to foot for 3 seconds without causing a fatal result, while in similar circumstances a direct current of the same voltage might be fatal in 1 second. Langworthy and Kouvenhoven (1932) in their experiments on rats found that a direct current voltage of 1000 volts when applied to the animals caused the death of all rats experimented on, when, however, an alternating current of the same voltage was applied some of the animals survived. These workers also found that when low voltages were employed, alternating current was more dangerous than direct, while at intermediate pressures of 500 volts the results were approximately the same in both cases. In this connection Langworthy and Kowenhoven quote the observation of Boruttau (1917) that of 212 fatal accidents due to electricity 8 only were due to direct current, and these workers themselves have learned of only one case of death occurring from 120 volts direct current.

Frequency

The frequency or number of cycles per second is also of considerable importance in determining the biological effects

of alternating currents. The low frequency of 50 cycles per second used for domestic purposes is much more dangerous than frequencies of 1200 upwards. Urquhart (1927) as a result of his animal experiments announced that there was no difference in the effects produced by frequencies between 12.5 - 75 cycles per second. The most dangerous frequency for dogs was found, by Prevost and Battelli (1899), to be 150 cycles. In 1891 Telsa found that large currents could be passed through the body with impunity and D'Arsonval (1892) demonstrated that he could pass 1 ampere through his body when the frequency was 4,000 - 1,000,000 cycles per second. In the following year 1893 he demonstrated that at these frequencies as much as 3 amperes could be passed through the body with no more than slight heating effect. To account for these differences in effects between high and low frequencies a variety of theories have been advanced. Jones (1893), stated his view that these currents should be regarded as milliamperes not amperes for, he asserted, at these high frequencies the effective resistance of the body is immeasurably increased so reducing the amount of the current. Others suggested that at high frequency the current did not penetrate the tissues but was dissipated over the skin surface. Yet another explanation offered was that the nerve response to one stimulation did not have time to register before it was annulled by the next succeeding alternation. D'Arsonval's (1893) theory that human muscles and nerves

are so constituted as to be unable to respond to stimuli of such short duration is the one that is generally accepted and as has been shown by Lapique's(1926) work on chronaxie the obvious one. Kennelly and Alexanderson, quoted by Jex-Blake (1913) have shown that at 60 cycles per second only 8 milliamperes can be borne from one hand to the other, and at 11,000 cycles 30 milliamperes, while at 100,000 cycles 800 milliamperes could be readily endured.

Resistance

The unit of resistance is the ohm. In entering and leaving the body the current encounters the high resistance of the skin which has been variously computed at 300 to 1,000,000 ohms. Jellinek (1913) has stated the dry horny palm of a workman may have a resistance as high as 2,000,000 ohms. This extremely high resistance is apparently due to the amount of keratinisation in the horny layers of the skin, keratin being practically a non-conductor. The resistance varies with the closeness of contact, being high with poor contact, and low with good contact. The presence or absence of moisture is also a factor, the resistance being lower when the skin is moist either from water or perspiration. The distance between the points of contact is also a factor in determining the resistance, the greater the distance the higher will be the resistance. In certain diseases such as hysteria and exopthalmic goitre the

resistance of the skin is said to be diminished and this observation has been made use of by Brazier (1933) and by Sargent, Frazer and Brazier (1938) who have calculated the impedance angle in exopthalmic goitre as a substitute for the estimation of the basal metabolic rate. The method is said to be particularly suitable for patients who are for various reasons unable to co-operate in carrying out the exacting conditions required in determinations of basal metabolism.

The position and size of the points of contact. (Electrodes)

Prevost and Battelli (1899) found that 4 amperes alternating current at a pressure of 1,200 could be passed through the body of a dog without fatal result when the electrodes were placed on the hind limb. This they considered was due to the fact that with the electrodes in that position very little current reached the heart. With the electrodes on the fore limbs only 80 milliamperes could be tolerated at a pressure of 80 volts. When the electrodes were placed on either side of the thorax so as to concentrate the current on the heart death resulted with 50 milliamperes at 15 volts pressure. In each of these positions the resistance was about 300 ohms. Jex-Blake (1913) in a review of the effects of electrical accidents states his opinion that this is the most convincing proof of the dangerous effects of electric currents on the heart. Crile and MacLeod (1905) also found the position of the electrodes to be

of the greatest importance in determining the biological effects of electric currents. Using an alternating current at a frequency of 60 cycles per second they found, when the electrodes were placed, one in the mouth, and the other in the rectum of a dog, that the momentary application of 1,000 volts was fatal. When, however, the electrodes were placed on the hind limbs 2,300 volts could be repeatedly applied for periods of from 1 - 10 seconds without necessarily causing the death of the animal. This they also concluded was due to the larger amount of current reaching the heart when the electrodes were in the mouth rectum positions.

With regard to the size of the electrodes, the current density per square centimetere is proportional to the size of the electrodes. When these are small therefore the current will be concentrated at the site of application.

Duration of Contact

The time during which the current is allowed to flow appears to be of the greatest importance. Prevost and Battelli (1899) found that currents which were not always fatal when applied for less than 1 second, were invariably fatal when applied for 1 to 3 seconds, and Crile and MacLeod (1905) found that an alternating current of 2,300 volts was fatal to anaesthetised dogs when applied for 20 seconds, but

not necessarily fatal when applied for 1, 5, or 10 seconds. Similar results have been recorded by Langworthy and Kouvenhoven (1932).

Despite the existence of certain anomalies it appears clear from this brief review of the properties of electricity that the most likely combination to cause damage to organic tissues is a low voltage, low frequency current such as is employed for domestic and industrial purposes in this country. It is a tribute to the skill and excellence of electrical craftsmanship that such accidents are not, with the increasing use of electricity, much more common.

## THE BIOLOGICAL CHANGES RESULTING FROM CONTACT WITH ELECTRICAL CIRCUITS

For many years, indeed, almost since the first death caused by electricity was reported, which is believed by Jex-Blake (1913) to have occurred in 1879 when a stage carpenter at Lyons was killed by an alternating current of 250 volts, controversy has existed as to the exact mode by which death occurred. Opinion nowadays tends to be divided between two theories namely that, death is the result of primary heart failure, or lesions of the central nervous system involving the respiratory centre. There is, however, a good deal of evidence to show that the mode of death varies with the voltage, being by primary heart failure with low tension voltage, and by lesions of the nervous system with high tension voltage.

Brouordel quoted by Grange (1884) reported on the post-mortem appearances of a man killed by an alternating current of 250 volts, and expressed the opinion that death was the result of primary heart failure. Bourrot, also quoted by Grange performed an autopsy on a similar case - and gave it as his view that death was the result of cardiac failure resulting from vagal inhibition.

This theory was not generally accepted, and Shield and Delepine (1885) who performed an autopsy on the body of a man

who had been dead for 40 hours as a result of an electrical accident, found that the heart was large, muscular and uncontracted. From this they concluded that death was due to lesions at the base of the brain, although they did not report on the appearance of that organ. D'Arsonval (1887) suggested that death was due to simultaneous action on both the heart and the respiratory centre. In 1889 Donlin, quoted by Jex-Blake (1913), put forward the theory that death from electricity was the result of spasm of the heart muscle, brought about by conduction of the current to the ganglia of that organ. This theory was based on the post-mortem appearances of 10 electrocuted criminals.

Prevost and Battelli (1889) to whose work reference has already been made were able to cause ventricular fibrillation in dogs by applying a current of 10 volts to the thorax. During the passage of the current respiration ceased as a result of fixation of the chest wall by muscular spasm. When the current was cut off respiration was spontaneously resumed, but later ceased from failure of the heart to keep the respiratory centres in the brain adequately supplied with blood. The life of the animal could, however, be prolonged by massage of the heart and artificial respiration, but death ensued when these manipulations were discontinued. These workers found that guinea pigs recovered completely from electrically

induced ventricular fibrillation after cardiac massage and artificial respiration. Rabbits recovered spontaneously, while in rats they found that the heart could not be made to fibrillate. With high tension voltages of from 1,200 - 1,400 volts the results obtained by Prevost and Battelli were quite different. Ventricular fibrillation did not occur, but the nervous system showed marked cellular damage, and respiration was arrested. With intermediate voltages of 240 to 600 volts damage to both the central nervous system and ventricular fibrillation occurred. These results were obtained with alternating current. With direct current the results were similar. Voltages of 50 - 70 volts caused ventricular fibrillation and arrest of respiration only after several minutes from failure of the vascular supply to the brain, while pressures of 550 volts damaged the central nervous system.

Tatum (1890) in his electrical experiments on animals was unable to arrest respiration without causing an immediate arrest of the heart. He also found that weak currents might suspend respiration without affecting the heart, while strong currents arrested the heart without arresting respiration. From these results he concluded that the cause of death in electrical accidents was primary heart failure.

Buchanan (1892) performed an autopsy on a man who had been dead for 31 hours as a result of an electrical accident. The victim in this case had made contact with an alternating

current at 2,400 volts pressure. He was first seen by Buchanan within 10 minutes of the accident and artificial respiration was adopted without success. At the post-mortem the blood was found to be "tarry" in appearance and Buchanan concluded that this was consistent with death from asphyxia. Oliver and Bolam (1898) as a result of their animal experiments stated their view that the heart is first arrested and that respiratory arrest is secondary to this. A similar conclusion was reached by Cunningham (1899) who found no evidence to suggest that the current had a direct effect on the medulla.

Stanton and Kridda (1910) were the first to advance the theory that the cause of death varied with the voltage, namely that low tension currents caused death by primary heart failure, and high tension currents brought about death by respiratory paralysis. Jellinek (1913) however disputed this theory and pointed out that it was frequently possible to resuscitate apparently electrocuted individuals by artificial respiration. MacWilliam (1922) has supported the theory of Stanton and Kridda.

MacMahon (1929) carried out a series of experiments on guinea pigs with a voltage of 110 volts. Under aether anaesthesia he exposed the heart and observed that during the passage of the current, except for two or three irregular beats due to vagal escape, the heart remained contracted. When the experiment was made after premedication with atropine the heart

continued to beat normally during the passage of the current, which suggests that arrest of the heart when it occurs is due, as first suggested by Bourrot to vagal inhibition. MacMahon also reported that, during the passage of the current, respiration was arrested in the inspiratory phase. This lasted for some seconds after the current had been interrupted, but was then resumed spontaneously. The peripheral nerves and spinal cord were also studied by MacMahon after these experiments. These he found, on microscopic examination, showed swelling of the axis cylinders and myelin sheaths. The white substance of the spinal cord was the more seriously affected in this way, and more particularly in the region of the posterior columns.

Ivy and Barry (1931) have reported on the use of electricity as a means of stunning dogs. During their experiments they found that the effect on the respiratory system depends on the strength of current used. With strong currents respiration was arrested in deep inspiration while weak currents caused the respirations to become rapid and forcible.

Auricular fibrillation in man as a result of electrical accidents has been reported by Hay and Jones (1922) and by Laslett (1927).

Lesions of the Nervous System

The majority of the lesions found in the nervous system

after contact with electrical currents have been the result of either judicial electrocution or, experimental work on animals.

Spitzka and Radsch (1912) performed post-mortem examinations on the brains of 5 electrocuted criminals. The voltage used for the purpose was between 1.750 and 1,800 volts and the applications were made for 1.12 to 1.22 seconds. The current passing through the body was 8 to 11 amperes. Lesions were found in the mid-brain, the pons, medulla and in other parts of the nervous system. These consisted for the most part of peculiar areas varying in size 25 - 300 $\mu$ in diameter. These, in their most characteristic form presented two zones, a central rarefied zone, surrounded by a zone of condensed tissue. Many of these contained a small blood vessel.

Langworthy (1930) examined, post-mortem, the central nervous system of two men following electrocution, one judicial, and the other from accidental contact with a circuit of 2,200 volts alternating current. In the latter case the duration of contact was about 20 minutes. In each case he found changes in the nerve cells. These were most marked in the medulla and more particularly in those centres concerned with respiration.

Hassin (1933) also examined the brains of 5 electrocuted criminals. In each of these, four applications of the current were made. The first and third applications were made with

current at a pressure of 2,300 volts and the duration of each was 7 seconds. The second and fourth were at a pressure of 550 volts and each lasted 52 seconds. At post-mortem examination no microscopic evidence of damage to the nervous system was observed. Microscopic examination, however, revealed swelling of the ganglionic cells, many of which in the deeper layers of the cortex presented satellitosis, some were dislocated and others appeared eaten away, while many presented vacuolation of their protoplasm, or appeared liquefied. In the last named the cell body and processes were pale, granular, devoid of chromaphil material, and the nucleus possessed an ill defined membrane, and was often misshapen. These changes were found by Hassin throughout the central nervous system but were most marked in the cerebral cortex especially along the line of the fissures. Many of the small blood vessels were congested and exhibited the shrinkage spaces of His. The larger vessels often showed rupture of their walls, while in others the walls were merely thin, and the adventitia which was the only coat unaffected protuded in the form of an aneurysm. These changes were not present in the smaller blood vessels.

Langworthy and Kouvenhoven (1932) whose work on rats has already been mentioned, found quite well marked changes in the nervous systen. These changes were chiefly shrinkage of the nucleus, and loss of staining reactions while the nucleolus

had become indistinguishable. These changes were chiefly seen when voltages of 500 - 1,000 volts had been applied, and the Purkinje cells were the ones most affected, indeed, these showed degenerative changes after the application of pressures as low as 100 to 120 volts. Langworthy and Kouvenhoven also observed injured nerve cells in the dorsal nucleus of the vagus.

With the exception of damage to the cells of Purkinje reported by Langworthy and Kowvenhoven damage to the nervous system has been the result of exposure to high voltages, and many workers among whom Kalinowsky (1939), have expressed the opinion that low tension currents do not damage the nervous tissues. Bini (1937) however, has shown that widespread damage may be caused to the nervous system of the dog by voltages as low as 120 volts, even when the duration of contact is as short as 1/20 - 1/15th of a second, and the current is passed through the entire body of the animal. The changes reported by Bini included cloudy swelling, retraction and hyperchromatosis of the cytoplasm, and distortion of the cell processes. The glial tissues were also involved in pathological change.

Besides these more important effects of electric currents, lesions have been described in most, if not all, of the bodily systems as a result of electrical accidents. Jellinek (1913) has discussed the characteristic effects of electric currents on the skin. These are chiefly current

markings and burns. Jaffe (1928) observed the occurrence of small lesions of the bones of the skull when the current had entered the head. Albuminuria has been said to occur after electrical accidents, and Langworthy and Kouvenhoven (1933) found blood and hyaline casts in the kidney tubules of many of their rats after exposure to the electric current. These writers also noted the presence of a lencocytosis about 1 hour after the application of the current.

In the eyes late lesions have been described by Bruner (1924) Franklin and Cordes (1925) and others. In Bruner's case bilateral cataract occurred in a man aged 27 years some 15 months after contact with a high tension of 22,500 volts. The visual defect was first noted by the patient about 8 to 10 weeks after the accident when he reported that his vision was not so good as formerly. Bilateral cataract extraction was successfully performed. In the case reported by Franklin and Cordes, lenticular opacities developed some time after contact with 220 volts. In each of these cases there had been a severe degree of burning of the face, and it is possible that the lenticular changes were the result of a slowly developing coagulative necrosis. Optic atrophy occurring in a man aged 48 years was reported by Rollet and Arnaud (1927) as a result of contact with a high tension circuit of 12,000 volts. In this case the current entered the forehead. Bainbridge (1930)

has reported a similar case in which macular changes accompanied the optic atrophy. Contact was with 460 volts. The pathological process in this case was reported to resemble a slowly progressive non-infective inflammation.

Psychotic changes have been also ascribed to electrical accidents. Schiff, Picard and Ponffray (1928) reported the occurrence of a hallucinatory psychosis in a man aged 48 years after an electrical accident. Fetterman and Smiley (1937) reported on a boy aged 12 years who was injured by contact with a 4,000 volt circuit in 1934. Two weeks after the accident the boy was visually hallucinated, and in 1937 was showing a behaviour disorder marked by hyperkinetic tendencies. Conversely, MacDonald Critchley (1936) notes that psychotic disorders may improve following electrical accidents.

Organic nervous disorders the result of electrical accidents have been reported by continental workers especially Panse (1935) and although both British and American workers discuss this problem, I have been unable, in a review of British and American literature for the past 30 years, to find a well authenticated case of this nature. Among the conditions described by Panse as resulting from electrical accidents, are hemipareses, aphasia, Parkinsonism and many others.

From this brief resume of the more important literature dealing with the biological effects attending the passage of

electricity through the body it will be seen that the damage resulting may be widespread involving, the skin, bones, eyes, kidneys, the heart, the central nervous system and peripheral nerves, and even the mind the last resulting in psychotic changes and behaviour disorders. It also appears to be fairly well established that when death results it may be due either to primary heart failure from ventricular fibrillation, or asphyxia from paralysis of the respiratory centres. The former mode being the more probable from contact with low voltage and the latter from contact with high voltage.

With regard to the means by which the lesions of the nervous system are produced Spitzka and Radsch (1912) whose post-mortem findings in electrocuted criminals have already been noticed, suggested that the lesions were the result of liberation of gas by the electrolytic action of the current. Hassin (1933) however stated his view that they were brought about by purely mechanical factors, the action of the current being equivalent to a direct mechanical injury. Critising these theories Pritchard (1934) observed that the electrolytic theory of Spitzka and Radsch is untenable for two main reasons, firstly that it is only direct and not alternating current that causes electrolysis, and secondly that the amount of gas that could possibly be liberated during the passage of the current used in judicial electrocution, at most only a few cubic centimetres

could not bring about the lesions. With regard to Hassin's theory Pritchard states that "The only mechanical effects which attend the presence of neighbouring electric currents are due to the electro-magnetic forces; these are entirely negligible in the case of currents passing through the body and, such as they are, they will tend to produce contraction and approximation of the conductors along which they pass and not dilatation." He then attempts to explain the production of the lesions in terms of electrostatic effects. This, even he finds unconvincing for he concludes by observing that "The proportion in which these lesions of the central nervous system is to be ascribed to current and to electrostatic effects can only be decided when we have a more accurate idea of the electrical state of affairs within the body than we have at present."

## EARLY USES OF ELECTRICITY IN PSYCHIATRY

The induction of convulsions by electricity is by no means the first application of this modality to the treatment of mental disease, for ever since Stephen Grey in 1731 showed that the human body could be electrified by induction physicians and others have been attracted by the possibility of using electricity as a therapeutic agent. It was not until sometime later, however, that these ambitions were realised. This was after the invention of the Leyden jar in 1746 and from then until the end of the 19th century electricity was hailed as a panacea for all the ills of the flesh, and it would have been unnatural if the disorders of the "spirit" had been neglected in this respect.

Among the first to apply electricity to the treatment of mental disorder was one Richard Lovett, a lay clerk at Worcester Cathedral. This is not so strange as it at first appears for this period was but shortly removed from that when the evil spirits, with which it was thought the insane were possessed, were exorcised by the clergy. Lovett, in 1756 published a book entitled "Subtil Medium" in which he claimed many successes and expressed surprise that many of his patients discontinued treatment on account of the pain and discomfort experienced. Later in 1780 John Wesley, another

member of the clergy expressed the view that for nervous disorders of all kinds there was nothing in nature to equal the proper and continued use of the electrical machine.

As it was not until 1880 that Volta invented the simple electric cell, these early electro-therapists, if one might so name them, must have made use of static electricity derived from electrical machines. This method, which later came to be known as "Franklinisation" after the inventor of the modern form of condenser, was unreliable and never became popular in this country where the uncertainty of the weather made the behaviour of the machines, which depend for efficient working on a dry climate, erratic.

Although Huff, in 1853, reiterated Wesley's belief in the efficacy of electricity as a means of treating the mentally afflicted no attempt was made to study the subject systematically until about 1870. In that year, Arndt gave an account of his experiences with both galvanic and faradic currents. In his experience only those psychical disorders depending on functional disturbance, or anomalous nutritive processes, or vasomotor disturbances could be influenced by the electric current.

Allbutt (1892) has given an account of the electric treatment in use at that time in the West Riding Mental Hospital. The current was obtained from 100 modifications of the Daniell cell. The current of from 5 to 20 of these cells was applied

to the head of the patient for 10 minutes. The Daniell cell has a voltage of 1.1 volts, therefore it is likely in Allbutts method that the voltage applied to the head would be of the order of 5 to 20 volts. Assuming that the head resistance of the patient was 1000 ohms the initial current applied to the head would, by Ohm's law, be of the order of 5 to 20 milliamperes. Organic tissue, however, does not obey the ordinary electrical laws and one of the first effects of the current is to reduce the resistance of the tissues, which falls progressively with the passage of the current until a minimum in the region of 200 to 300 ohms is reached. The ultimate current reaching the head would therefore be between 17 to 70 milliamperes, assuming that the resistance of the head reached the level of 200 ohms. Nothing was noted by Allbutt regarding the feelings of his patients during these applications, although he observed that the immediate result of application of the current was to "tranquilize." This effect he considered could not be due to the mere "astonishment" evoked by the procedure for it did not lessen on repetition. Since so small a current as 1 milliampere can readily be detected by sensitive people, and 10 milliamperes cause considerable pain, one can only imagine that the effect of 17 to 70 milliamperes would be, to say the least of it astonishing!

Erb (1883) wrote at considerable length on the use of

electricity in treating mental disorders. He held the view that many of these illnesses were the result of vasomotor disturbance and could readily be influenced by the electric current when applied to the head and the cervical sympathetic nervous system. While admitting that his experience was too limited to permit him to furnish definite laws for the electrical treatment of the psychoses, he suggested that the method should only be used in mild and recent cases. Erb was aware that little could be expected from the application of electricity to the treatment of general paralysis.

Haynes (1884) recorded favourable results from electricity in recent functional cases of mental disorder. He did not observe any change in old standing cases, or cases of an organic nature. In this respect it is interesting to note that these results are closely similar to those being obtained by present day shock methods.

Towards the end of the 19th century perhaps, because little or no discrimination was made between the organic and the inorganic psychoses, and all received the same treatment interest in electricity began to wane and by 1900 was no longer practised by psychiatrists.

## ELECTRICAL CONVULSION TREATMENT

Impetus to re-examine the possibility of using electricity in the treatment of psychotic disorders was afforded when Meduna (1935) introduced his method of treating schizophrenia by pharmacologically induced convulsions. Cerletti, who had made much use of electricity in studying convulsive phenomena in animals, was immediately attracted by the idea that a similar use might be made of it in the human subject. This was strengthened when it became apparent that cardiazol was by no means the ideal method of inducing convulsions. Together with Bini, Cerletti made many experiments on animals in order to determine the maximum and minimum current strengths that could be applied with safety. It was Bini who overcame the difficult problem of application of the electrodes, and designed the simple and efficient apparatus used to apply the current for a pre-determined time. Despite these experiments and observations, it must have been with some degree of anxiety that they proceeded to their first human experiments for as Urquhart (1927), Jaffe (1928) and others have emphasised, care must be exercised in applying the results of electrical experiments on animals to interpret the results of electrical currents when applied to man. The success of these experiments is now fairly well known and the method is likely to receive an

extended trial in this country.

The Apparatus

The apparatus employed throughout this work is a slightly modified version of that used by Cerletti and Bini (1938). It has already been described in some detail by Shepley and McGregor (1939) and (1940), so that it is only neessary to describe the more essential features here.

Alternating current at 50 cycles per second is used, that is, the ordinary industrial and domestic supply. By means of a tapped transformer voltages of between 50 - 150 volts are obtained in sixteen steps. For measurement of the resistance of the head 1 milliampere of direct current is employed. This is obtained by rectifying 5 volts alternating current. The current is applied to the head by means of electrodes which are applied over the parieto-temporal region. Since the original paper by Shepley and McGregor the design of the electrodes has been modified slightly, fine copper wire gauze being substituted for the silver plated copper strips originally in use. This has proved much more reliable and durable. The duration of the shock is pre-determined by a condenser type of time switch which gives readings of 0.1 - 0.5 second. The shock is applied by means of a press button switch. The apparatus is housed in a light portable switch table. Figures

1 - 4 illustrate the apparatus and electrodes.

The head resistance

The head resistance varies within fairly wide limits, even in the same individual and appears to depend to some extent on the degree of activity of the sweat glands, for when the method is applied to patients undergoing the insulin treatment, introduced by Sakel (1934) those patients in a wet coma, show a lower resistance than those in a dry coma. When, however, the head resistance of both groups is measured on an insulin rest day, it has been found that the resistance of the dry group is about the same as that obtaining when in coma, while the resistance of the wet group is now considerably higher than when in coma. This is shown in Table X.

Head Resistance

| Dry group | | Wet group | |
|---|---|---|---|
| Coma | Rest Day | Coma | Rest Day |
| 650 ohms | 800 ohms | 150 ohms | 500 ohms |
| 1200 " | 1200 " | 200 " | 600 " |
| 700 " | 700 " | 250 " | 700 " |

Table X showing the influence of moisture on the head resistance.

The upper limit of head resistance appears to be about 2,000 ohms, resistances higher than this indicating faulty

Figure 1. General appearance of apparatus.

apposition of the electrodes, or an increase in the resistance of the electrodes themselves due to formation of copper oxide by the action of the current on the 20% salt solution with which they are coated before application to the head and also with the paste used to moisten the patients' hair and scalp. The lower limit of head resistance that has been recorded is 150 ohms in a patient during insulin coma, and 200 ohms in several patients undergoing convulsion treatment only.

Neither direct nor alternating current give the true value of the resistance of organic tissues, for with the former the effect is partially one of "charging," as when an accumulator is charged, and with the latter it is "impedance" that is measured. This value includes other factors such as "Capacitance." In the present instance however the amount of direct current used, 1 milliampere, is so small that the charging effect can be ignored, and the value accepted as the true resistance for that amount of current.

As has been pointed out by Cerletti and Bini (1938), Kalinowsky (1939) Fleming, Golla and Walter (1939) and Shepley and McGregor (1939), the head resistance as measured in this way bears little relationship to the amount of current that passes through the head when the shock current is applied, for in every case the current registered is much greater than anticipated by calculation from Ohm's law. This is illustrated in Table XI

TABLE XI

| Duration of shock | Measured resistance | V | Current anticipated by Ohm's Law | Current Registered | True Resistance by Ohmn's Law |
|---|---|---|---|---|---|
| 0.2 sec. | 600 ohms | 110 | 180 MA | 750 MA | 147 ohms |
| 0.2 " | 400 " | 120 | 300 " | 850 " | 141 " |
| 0.2 " | 450 " | 125 | 268 " | 830 " | 151 " |

TABLE XI showing the disparity between the anticipated current, and registered current, and also the true resistance for the shock circuit.


Figure 2. Control panel of apparatus.

which shows that in each instance the current which passed through the head was approximately three times as much as expected. The reason for this is that organic substance does not obey the ordinary electrical laws.

To obtain the true value for the head resistance on the shock circuit Ohm's law is applied, using the voltage employed and the current registered as passing through the head. In this way it is found that the resistance offered to the passage of the current on the shock circuit, is very much less than that measured on the potentiometer. Table XI. Theoretically, in making this calculation the capacity of the head between the electrodes should be taken into consideration but as this is of such a small value probably 0.001 microfarad it can be neglected.

In 64 patients the average head resistance was 947.3 ohms, and the standard deviation 76 ohms.

It has also been observed that, after the application of the shock current, whether of sufficient intensity to induce a major fit or not, the head resistance has dropped considerably. This was thought by Shepley and McGregor (1940) to be the result of electrolysis in the tissues and was found by them to be progressive, after each shock, until a minimum in the region of about 100-200 ohms was reached. This point is illustrated in tabular form in Table XII for one patient who was given six shocks before a major fit was elicited. It will be seen that

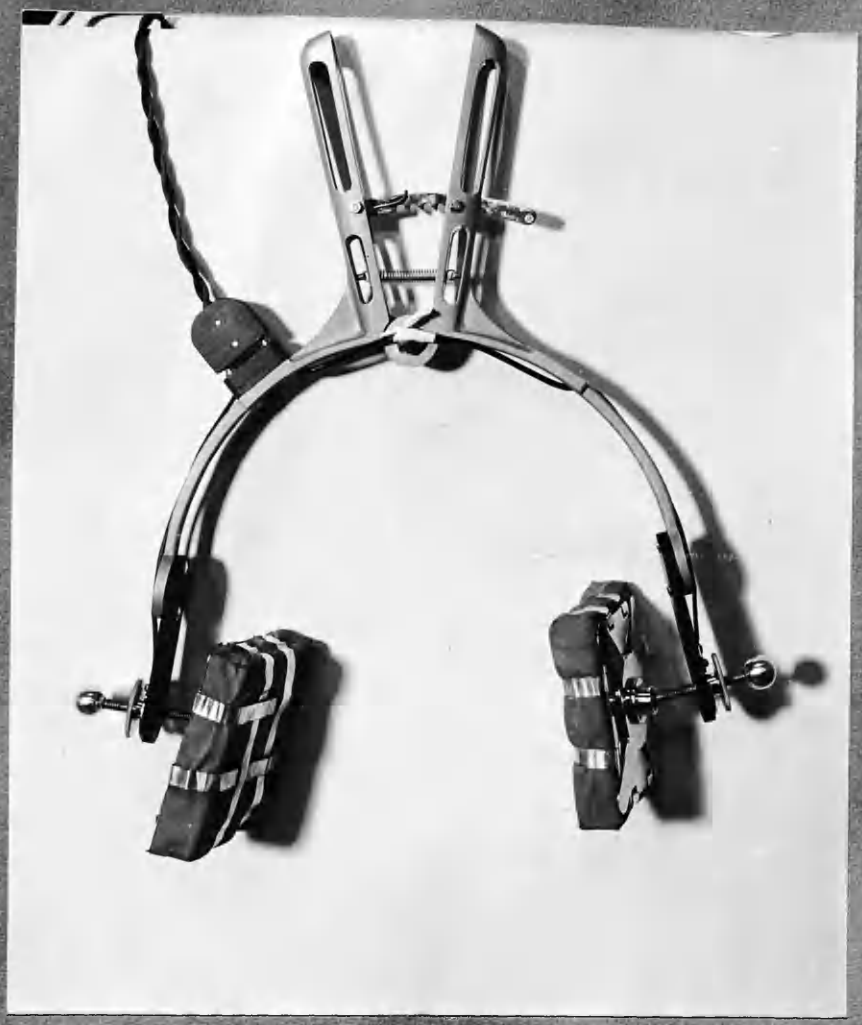


Figure 3. The electrodes.

the resistance drop becomes progressively smaller.

TABLE XII

| Date | Shock | Resistance |
|---|---|---|
| 24.12.39 | 1 | 1250 |
| | 2 | 450 |
| | 3 | 350 |
| | 4 | 330 |
| | 5 | 250 |
| | 6 | 250 |

Voltage and Duration of Shock

The voltage ordinarily required to induce a major fit is 120 volts applied for 0.2 second. In some few patients, however, it has been necessary to increase the voltages, and duration of application to 150 volts, and 0.5 of a second respectively before a major fit could be successfully induced. These resistant cases are to be found among both sexes, and there does not appear to be any relationship between the degree of resistance and the body weight, or physique or temperament, neither do they show unduly high head resistance. Shepley and McGregor (1940) expressed the view that a major fit might be induced by a high voltage applied for a short time or a low voltage applied for a long time. Within the limits set by the apparatus, this has since been shown to be true.

Figure 4. The electrodes in position.

TABLE XIII

| | Date | Time | Resistance | Voltage | M.A. | Result |
|---|---|---|---|---|---|---|
| | 21.3.40 | 0.1 sec. | 400 | 115 | 700 | Loss of consciousness |
| a) | 25.3.40 | 0.1 " | 400 | 125 | 800 | Major fit |
| b) | 28.3.40 | 0.5 " | 650 | 72.5 | 320 | Major fit - slight delay |
| | 2.4.40 | 0.5 | 640 | 68.0 | 310 | Loss of consciousness |
| | 6.4.40 | 0.5 | 600 | 72.5 | 320 | Major fit |

Table XIII showing the induction of a major fit by a high voltage for a short time, and also by a low voltage for a long time.

Table XIII illustrates this point quite clearly. A major fit was induced when 125 volts were applied for 0.1 second, and also when 72.5 volts were applied for 0.5 second. These were the lowest voltages that would successfully induce a major fit in this particular patient. As to which of these methods is the more desirable is a matter for some speculation. The exact mechanism by which the convulsion is produced is by no means clear, nor is it clear which of several electrical properties are involved. Fleming, Golla, and Walter (1939), observe that an application of 0.2 second allows of 10 diphasic stimuli reaching the brain when an alternating current of 50 cycles per second is used, and assume that it is the negative half of each cycle that stimulates, so that by their reckoning each hemisphere of the brain reserves 10 supramaximal stimuli in 0.2 second. By reducing the time to 0.1 second only half of

that number of stimuli would reach the brain, which is of importance should it emerge that this form of treatment gives rise to cerebral damage. If it is supposed, however, that the fit is induced by virtue of the electrical energy expended then in the examples in Table XIII the amount of energy expended in Joules, obtained from the formula E.C.T., where E = voltage, C = current and T = time, is 10 and 11.6 Joules respectively. In each case

a) 125 x 0.8 x 0.1 = 10.00 Joules
b) 72.5 x 0.32 x 0.5 = 11.6 Joules

therefore, the electrical energy expended is approximately equal and little or nothing is gained by selecting one method in preference to the other. But, if it is supposed, that it is the power factor, or wattage that is responsible for the fit, then the position is entirely different. Watts are obtained from the formula E x C, no account being taken of the time during which the current is allowed to flow. In the examples cited above we find that the power factor or wattage is 100, and 23.2 watts respectively. Clearly then it is better to employ.

a) E x C = 125 x 0.8 = 100 watts
b) E x C = 72 x 0.32 = 23.2 "

low voltages and long times. It is, however, inconceivable that the duration of the shock can be disregarded for as shown by Prevost and Battelli (1899), Crile and MacLeod (1905), and

Langworthy and Konvenhoven (1932), to whose work reference has already been made, animals may survive current applications of short duration while succumbing to applications of longer duration.

## The position of the Electrodes

The site of application of the electrodes can be varied considerably, without conspicuously altering the result of the shock. Fleming, Golla and Walter (1939) apply the electrodes to the frontal bone about the level of the outer canthus. In this position stimulation of the supraorbital nerves when the head resistance is being measured gives rise to sensations described by the patient as "burning" or "tingling". In Japan the position of the electrodes has been the subject of considerable variation. Yasukoti and Mukasa (1939) have placed one electrode over one motor area and the other on the cheek, hand, or arm of the opposite side. Best results, however, were obtained when the electrodes were over both motor areas, for in this position there is, they consider, little likelihood of the current reaching the heart directly.

## Tolerance

When cardiazol or triazol is used as the convulsant agent, it soon becomes apparent that tolerance for the drug

develops and the amount given has gradually to be increased in order to induce a fit successfully, sometimes to as much as 3 to 4 times the initial dose. In one of my cases under treatment with cardiazol 25 ccs. given at once failed to induce a fit, although in the beginning of treatment 7½ ccs. were sufficient to induce a strong convulsion. With the electrical method however the question is not so simple as this owing to the number of variables, but all things being equal it is doubtful whether any real degree of tolerance does develop. One of the chief difficulties that one is confronted with in determining this problem is the variation in the head resistance, which as already noted, occurs in individual patients. When this variation is in an upward direction a difference of 200 ohms, may cause a voltage which had hitherto proved sufficient, to fail to induce a fit. When this happens it is usually necessary to increase either the voltage or the time of application by 5 to 10 volts or 0.1 second respectively in order to induce a convulsion. This point will be taken up again when abortive fits are under consideration.

Preparation of the patient.

No special preparation of the patient is necessary. Each receives a light breakfast in the morning. This usually consists of tea and toast or biscuits, and is taken at the

usual breakfast hour. It is doubtful if even this is necessary for when the full breakfast has been taken in error no vomiting or other untoward occurrence has been observed. It has not been necessary to resort to premedication of any kind. Patients, whose habits are faulty, are given an enema on the night before treatment. This usually eliminates any tendency for evacuation of the bowels to occur during the fit.

Treatment is carried out in a single bedded room, a couch however being substituted for the bed, as the head end of the bed would foul the electrodes when in position. This could be overcome by substituting a foot part for the head end, for on most hospital beds the foot end is low enough to allow the electrodes to be readily applied. Meduna (1938) considers that convulsion treatment carried out in a single room is harmful to the patient and advises that ten to fifteen patients should be treated together in a small dormitory, screens being used at the time of the injection. This method was adopted when cardiazol was in use at this hospital and was in my own opinion no better than the single room method.

Modification of the method adopted in America.

In America, although early notice was taken of the work of Cerletti and Bini, perhaps because of a not unnatural prejudice in a country where judicial execution is mainly carried out by electricity, the method has so far been adopted only in a much modified form.

This is not surprising, for even in this country the method has been, rather ineptly, compared with those adopted in Sing Sing. (Lancet 1939 vol. 2 page 1373).

Berkwitz (1939) who considers that the high milliamperages, and low frequency adopted by Cerletti and Bini are liable to cause damage to the central nervous system has adopted the following modification. Faradic current obtained from a six-volt battery is employed. This is boosted to high potential by means of a motor car ignition coil. To keep the milliamperage low, a resistance of 500,000 ohms is connected in series with the coil. With the patient prepared as for a general anaesthetic, one electrode is placed on the forehead and the other on the nape of the neck. From 1 - 10 shocks are given at intervals of one second. The duration of each shock is one second. These applications arouse considerable fear and pain in the patient, but Berkwitz does not think there is any real "mental anguish". Immediately the treatment is over from 4 - 7 ccs of 5% pentothal sodium are given intravenously. This relieves the patient of his fear and his anxiety. Berkwitz who does not believe that either cardiazol or insulin treatments "cure" schizophrenia, but only pave the way for psycho-therapy, apparently has belief in the efficacy of fear. As has already been shown, in both, the method and its effects, analogies may be found in the literature of the 19th century.

## RESULTS OF APPLICATION OF THE CURRENT

Shepley and McGregor (1939) in their preliminary report on the application of Cerletti and Bini's method described the results of the passage of the current as the major fit, and the abortive fit. Although, in the light of more experience, it is probable that a more suitable term would be abortive shock, it is convenient to adhere to the original description.

### The Major Fit

This closely resembles the convulsion of idiopathic epilepsy, or that induced by cardiazol or triazol injections. Its duration is fairly constantly 45 to 50 seconds from the onset of the tonic stage to the end of clonic stage. Its severity is however less than that induced by cardiazol and I have not observed the marked opisthotonus, so common in the cardiazol convulsion. Usually the fit begins immediately the shock button is pressed, no appreciable lag being noted, while much less frequently there is a delay of perhaps 30 seconds before the onset of the tonic stage. During this time the patient is quite unconscious. In the application of the method to patients in insulin coma a delay as long as 1½ minutes may occur. This greatly increased delay was thought by Shepley and McGregor (1940) to be due to the depression of cortical activity by insulin. Golla, Walter and Fleming (1940) describe an initial "start" or "convulsive movement of all four

extremities, facial and spinal musculature," before the onset of the tonic stage. Since I had rarely or never observed anything of this nature as a prelude to the fit I designed a pair of electrodes suitable for application to the frontal bone. These were mounted on a strap in such a way that they could readily be adjusted over the site adopted by Fleming, Golla and Walter, figure 5. When this was done application of the current was in nearly every case followed by the initial "start" reaction described by those workers. It is difficult to explain this difference and I can only conclude that in the frontal bone position the current stimulates some area of the brain producing the start reaction, before reaching the frontal adversive area, 6aβ, which is claimed by Cerletti and Bini to be the area from which the convulsion arises.

The stages of the fit are usually well marked. Commonly it is ushered in with a cry and the mouth opens permitting the insertion of a soft gag to prevent biting of the tongue. In my experience, gags made of such material as rubber induce excess salivation, and although economical are to be avoided for this reason. It is interesting to note that a cough preceding the onset of the fit has not yet occurred, although it is a common enough feature of the cardiazol convulsion. Flexion of the limbs and trunk is moderate. The face flushes and the pupils dilate widely.

Figure 5. Electrodes mounted on strap for application to the frontal bone.

At this stage the pupillary and conjuctival reflexes are absent. When there is a latent period before the onset of the fit the pupils usually dilate to their fullest extent, a sign that can be taken as heralding the approach of the fit, occasionally, however, they contract almost immediately and no fit follows. The eyes usually show conjugate deviation to the left or right and in an upward direction, less commonly they are directed downward. In some patients the onset of the fit is marked by flexion of the thighs, but only on rare occasions has opisthotonus been observed. The great toes are as a rule dorsi-flexed in the tonic stage of the fit. During the clonic stage of the fit, or at the onset of the stage of flaccidity urine and faeces may, rarely, be evacuated, and in male patients seminal emissions are not uncommon. The deep and superficial reflexes are usually absent and there is an extensor plantar response in the stage of flaccidity. The plantar responses usually remain extensor until consciousness begins to return. Strong stimulation of the plantar aspect of the feet in this period usually causes defensive withdrawal movements of the whole limb. With the onset of flaccidity the pupils contract but remain inactive for as long as 1 to 3 minutes in individual patients, during this time they may show coarse movements of contraction and relaxation resembling the movements of Hippus. These movements are unrelated to the respiratory cycle and seldom last for more than 10 to 20 seconds.

TABLE XIV

| | | |
|---|---|---|
| 1. | Pupils contract, light response returns | 1-3 minutes |
| 2. | Biceps jerks returns in | 1st minute |
| 3. | Knee jerks returns in | 1st minute |
| 4. | Pronators of arms returns in | 2nd minute |
| 5. | Triceps of arms returns in | 2nd minute |
| 6. | Supinators of arms returns in | 2nd minute |
| 7. | Hamstrings of arms returns in | 3rd minute |
| 8. | Angle of arms returns in | 3rd minute |
| 9. | Epigastric reflexes ) | |
| 10. | Abdominal reflexes ) | 6 - 10 minutes |
| 11. | Plantar reflexes ) | |

Table XIV showing the order and time of return of deep and superficial reflexes.

Table XIV shows the order and time relationships of the returning reflexes from the end of the clonic stage of the convulsion. Normally, all have returned within 5 minutes, with the exception of the epigastric, abdominal and plantar responses. These appear to depend for their return on recovery of consciousness, which fact suggests that the pyramidal tracts have been involved in some physiological process of inhibition. Frequently knee and ankle clonus can be elicited in the flaccid stage. A moderate degree of cyanosis is present at the end of the fit, but this is seldom so marked as after the cardiazol fit. Arrest of respiration has occurred in four patients only. In three of these it was spontaneously resumed without causing any anxiety, in the fourth however it was accompanied by consider-

able cyanosis of the face in a patient known to have a left ventricular preponderance, so pressure was exerted on the chest wall without waiting to see if respiration would resume spontaneously, this resulted in almst immediate resumption of the respiratory cycle.

Variations in the fit

In applying the method to patients undergoing insulin treatment Shepley and McGregor (1940) noted the occurrence of atypical fits of a unilateral nature. It is now clear that atypical forms of the fit may also occur in non-insulin patients. This is illustrated in the following; W.H. a male patient aged 46 years received on January 23rd 1940 two shocks. The first of these was at a pressure of 110 volts and was applied for 0.3 second. This resulted in loss of consciousness, pallor of the face and a slow small but regular pulse, which rapidly returned to its previous rate, rhythm and force. The second shock 120 volts applied for 0.3 second, after a latent period of 30 seconds, gave rise to the following. The eyes became fixed and staring in a forward direction, then deviated to the right, but almost immediately the head rotated to the left, and clonus of the circum-oral muscles with firmly clenched teeth began and continued for some seconds. This was then followed by opening of the left side of the mouth, an action which gave the patient an appearance resembling the efforts of a victim of Bell's

palsy to open his mouth. Clonic movements of the arms and legs now began and lasted for abut 15 seconds. To this point the fit had lasted 25 seconds. The head was now rotated to the right and this time the right side of the mouth was opened, and clonic movements of the facial muscles and limbs followed. Cyanosis of the face was marked and there was a fair amount of salivation. The fit lasted in all 60 seconds. Throughout the pupils were small fixed, and slightly irregular. Two and a half minutes after cessation of the fit the pupils had dilated slightly and showed a sluggish reaction to light. At 8 minutes from the end of the fit the patient had recovered sufficiently to answer simple questions, and at 20 minutes he had almost fully recovered, showing only slight confusion of cognitive functions and retrograde amnesia for the events immediately preceding the convulsion. This was the only occasion on which this patient had an atypical fit. Similar fits have been observed in other patients, but seldom occur more than once.

The post-accessual period

Following the fit the majority of patients, if undisturbed, sleep for half an hour or more. Mild motor restlessness is occasionally met with until consciousness returns, this is peculiar to the patients concerned and it occurs after each fit and can therefore be anticipated, and dealt with by the

nursing staff. Extreme excitement, such as is so common after convulsions induced by large doses of analeptic drugs does not occur, and it has not been necessary to administer sedatives in the post-accessual period. Vomiting in this period is also uncommon having occurred in only 6 non-insulin patients, and two insulin patients during the past year. In one of the non-insulin cases the vomiting was apparently due to delayed emptying of the stomach in a profoundly depressed patient, for when the evening meal was withheld on the day before treatment, this gave rise to no further trouble. In another the vomiting followed a copious draught of water injudiciously given by a nurse before the patient had completely recovered consciousness. In the others no definite cause could be ascertained, one patient showed a left ventricular preponderance which may have been a factor in its production. No cause could be found for it in the two insulin cases, and it has occurred even with the technique described by McGregor and Sandison (1940) in which potatoes are substituted for the usual nasal sucrose feeds, the coma having been terminated by intra-venous injection of 20 ccs. of 33% glucose. Vomiting, despite these 8 occurrences, is much less frequent than after cardiazol or triazol administration.

The abortive fit

In its typical form the abortive fit bears a marked

resemblance to the petit mal of idiopathic epilepsy. Much more than petit mal attacks may however occur all degrees short of the major fit with well marked stages having frequently been observed. Thus, there may be simple loss of consciousness of some seconds duration, and the patient starts up suddenly, like one roused from a dream, on being addressed. There is little clouding of consciousness, and the patient is usually fairly well oriented in time, place, and person, although presenting an amnesia for the events immediately preceding the application of the current. No complaint of pain, or discomfort has ever been made by a patient following these applications. On excited, restless, negativistic patients these abortive fits have an instantaneous salutary effect for on regaining consciousness the most aggressive patients are quiet, docile and co-operative. The only physical accompaniments of this type of fit are lachrymation and injection of the conjunctivae. On one occasion a patient remarked, almost immediately after one of these shocks "I almost went off there." This statement he could only with difficulty enlarge upon and I gained the impression that he thought he had almost lost consciousness.

While the foregoing describes the abortive fit in its most typical form there may be loss of consciousness, with slight dilation of the pupils, loss of conjunctival and light

reflexes, and spasmodic twitching of the limbs without the occurrence of the stages of the major fit. After these, consciousness is again quickly recovered and within about 20 seconds the patients are able to respond to simple questions, again with retrograde amnesia for the events preceding the shock, this is usually accompanied with slight disorientation for time and place, but not as a rule for person. The physical concomitants of this form of fit are commonly; flushing of the face, with shallow almost imperceptible respirations, so that cyanosis of the face and especially the lips develop. This cyanosis may not make its appearance until the patient has regained consciousness, or if developed often remains when consciousness has been regained, and then only disappears when the patient has been induced to breathe deeply by talking. Sometimes the pulse is quite perceptibly slowed, and the initial flushing of the face gives place to a marked pallor, which is reflected in a fall of blood pressure.

Anti-convulsant drugs such as paraldehyde, and sulphonal appear to influence the response to the shock current. One patient A.A. a female aged 50 years whose psychosis took the form of periods of pseudo-maniacal excitement marked by hypermotility, alternating with periods of normality had been receiving paraldehyde and sulphonal in 3 drachm and 30 grain doses respectively for some days during which time she was

also having electrical convulsion treatment without undue effect. On January 26th 1940 however these drugs appeared to have a cumulative effect in depressing the irritability of the cerebral cortex, for on this date, although hitherto the fits had been readily induced, three shocks were applied without inducing a major fit. The first of these caused loss of consciousness with flushing of the face, and then slight cyanosis. The second had the same effect. The third however caused flushing of the face and then rapidly developing cyanosis, especially of the lips. This was accompanied with slight dilation of the pupils. Later, 50 seconds after the application of the current, mild generalised myotonus occurred, this passed off in a few seconds. The pupils three minutes from the shock became small and fixed, and at 7 minutes she was still unconscious and breathing was sterterous,the pupils were pin-point in size and failed to react to light. At this time the deep reflexes were all present, but the superficial reflexes were absent. At 11 minutes from the application of the shock the jaw became tremulous. The blood pressure was 135/75. The eyes now began to show oscillatory movements, like those of a patient under light general anaesthesia. At 15 minutes from the shock the B.P. was 120/80 and the pulse rate 72. Stimulation of the plantar aspect of the foot caused partial dilation of the pupils, only to contract again when the stimulus to the foot was withdrawn. This condition con-

tinued until 21 minutes after the application of the shock, when she was returned to bed in the ward. Recovery was uneventful. This is the only occasion when a patient has remained unconscious for more than some seconds after an abortive shock. After this occurrence all sedative drugs were withdrawn and there has been no repetition of this event.

After the ordinary abortive fit it is customary, once the patient has regained consciousness to re-apply the electrodes and make a further attempt to induce the major fit. When this is done, as already observed, it is invariably found that the patient's head resistance is reduced. The reduction is related to the initial head resistance, for when this is large the reduction is also large, and when initially low the reduction is also low. It might therefore be expected in view of this lower resistance, that a repetition of the same voltage, and time would successfully bring about a fit. Kalinowsky (1939) however in an account of the work done on electric convulsion therapy in Rome states that it was found that if a certain voltage was applied for 0.1 second with failure to induce a major fit it was useless to re-apply the same voltage for even a longer time. Golla, Walter and Fleming (1940) on the other hand state that the effect of the fall in head resistance is to allow re-application of the same voltage to induce a major fit. In my experience both statements can be shown to be true. If the second shock of

the same voltage and time is applied as soon as consciousness is recovered after the abortive fit, it fails to induce a major fit, and indeed most often has much less effect than the first shock, and it is necessary to increase the voltage by 5 to 10 volts, or the time by 0.1 second in order to elicit the major fit successfully. If however a period of time which varies between 6 to 8 minutes is allowed to elapse before the electrodes are re-applied, repetition of the same voltage, and time will, if near the convulsion threshold, induce a major fit satisfactorily. From this observation it appears that the brain is inexcitable to the same stimulus for a period of 6 to 8 minutes. Experimental work on animals supports this view. Urquhart (1927) observed that after an electric stimulus had been applied to the brain a profound "block" occurred during which the brain was inexcitable, and providing no irreversible damage had been done to the brain cells, and artificial respiration was kept up, this was recovered from. Dusser de Barrene and McCulloch (1934) experimenting on monkeys with electric currents found that the time interval between successive stimuli was of the utmost importance. These workers employing a constant, frequency, duration and intensity of stimulus found that when the time interval, between the application of successive stimuli was 27 seconds or more, they consistently obtained the maximum possible response. When,

however, the time interval between successivve stimuli was reduced to 3 seconds the second stimulus evoked little if any response. As soon as the time interval was increased to 27 seconds or more the original response promptly reappeared. From this they concluded that some "extinguishing" factor must be operating in the motor cortex, or in the motor mechanisms involved. This phenomenon was observed in all monkeys that came under their observation. They also found that "extinction" was most marked when a slightly super-liminal stimulus was applied. This they took to indicate that the occurrence was not due to fatigue or exhaustion of the cortex, but that it was a physiological mechanism. They do not enlarge on the nature of the mechanism. In this connection, it is of interest to note that Bechterew, quoted by Samajo (1904) observed that when the brain of a newly born dog was stimulated by electricity, it remained unresponsive to further stimuli for half an hour. Similar observations have been made by Loucks (1934), Chaffe and Light (1934) Fender (1937) and others in experiments on the production of focal epilepsy. These workers showed that after the production of a seizure, continuation or repetition of the stimulus elicited no further response for a period of 5 to 6 minutes. Dusser de Barrenè, McCulloch and Nims (1937) have produced evidence to show that the phenomenon of extinction is dependent on the pH of the cortex.

## THE PHYSIOLOGICAL CONCOMITANTS OF THE FIT

Meduna (1935) recorded the effects on the blood pressure of the cardiazol convulsion and found that there was an increase, in the systolic and diastolic pressures in the post-paroxysmal stage, of 20 to 30 and 5 to 15 m.m. of Hg respectively. Harris (1938) found that the changes in the blood pressure following the cardiazol convulsion were irregular, although in the majority of his patients there was a post-paroxysmal rise. The blood pressure changes occurring in the post-accessual period of the electrically induced fit have accordingly been recorded in 15 patients. The procedure adopted was as follows. The blood pressure was recorded immediately before the application of the current and again in the flaccid state immediately after the fit, and at 5, 15, 30 and 60 minutes after application of the current. The changes recorded as shown in Table XV. In the majority of cases a fall of pressure was recorded in the flaccid stage, while in the record taken at 5 minutes an increase was observed which frequently exceeded the pressure obtaining before the fit. In the records taken at 15, 30 and 60 minutes the blood pressure is returning to its pre-paroxysmal level. In some cases the latter was high owing, most probably, to a mild degree of apprehension. In an earlier series of investigations, more frequent records appeared to cause emotional upset and were discontinued.

| Patient | Before fit | Flaccid stage | 5 mins. after | 15 mins. after | 30 mins. after | 60 mins. after |
|---|---|---|---|---|---|---|
| 1. | 130/80 | 120/70 | 110/75 | 110/80 | 100/70 | 110/70 |
| 2. | 145/90 | 105/55 | 165/95 | 105/60 | 140/80 | 120/60 |
| 3. | 125/80 | 125/80 | 170/80 | 120/70 | 105/70 | 105/70 |
| 4. | 125/75 | 100/65 | 130/60 | 100/70 | 135/80 | 130/80 |
| 5. | 140/100 | 100/70 | 130/100 | 150/105 | 140/100 | 140/100 |
| 6. | 135/85 | 125/85 | 130/85 | 125/75 | 125/75 | 120/80 |
| 7. | 120/65 | 130/60 | 125/75 | 120/80 | 120/80 | 115/65 |
| 8. | 130/80 | 120/70 | 90/50 | 150/80 | 125/85 | 105/80 |
| 9. | 150/80 | 150/80 | 155/80 | 135/75 | 130/70 | 130/80 |
| 10. | 160/75 | 200/95 | 145/70 | 145/75 | 150/80 | 140/80 |
| 11. | 150/75 | 180/85 | 130/65 | 125/65 | 120/85 | 145/70 |
| 12. | 125/75 | 130/80 | 125/75 | 120/80 | 135/80 | 125/80 |
| 13. | 130/80 | 120/70 | 140/85 | 125/70 | 135/75 | 130/75 |
| 14. | 140/90 | 135/80 | 150/90 | 120/65 | 130/80 | 135/75 |
| 15. | 100/65 | 150/80 | 135/60 | 125/70 | 120/70 | 110/70 |

TABLE XV showing the blood pressure changes after the major fit.

It is difficult to find a satisfactory explanation of this difference in the behaviour of the blood pressure in the flaccid stage of the cardiazol and the electrically induced convulsion, for the only objective difference between them is one of degree, the former being more severe than the latter. No doubt, in both, physiological and mechanical factors are involved, and consideration of these affords a possible explanation of the difference. During the tonic stage of the convulsion, owing to interference with venous return by the tonic contraction of the skeletal muscles, and fixation of the chest, blood accumulates

on the venous side of the heart. At the end of the convulsion this accumulated blood is suddenly released into the arterial circulation resulting in a rise of blood pressure. This is probably what occurs in the cardiazol convulsion and readily accounts for the findings of Meduna and Harris. On the other hand, if for some reason, there was a delay in the entry of the accumulated venous blood into the arterial circulation, and it was supposed that the same muscular tonus and fixation of the chest that had hindered venous return also impeded the arterial circulation; then at the onset of the flaccid stage a fall in blood pressure such as that noted after the electrically induced fit would result. Such a delay, even if it did occur in this way, would be of very short duration and it is doubtful if there would be sufficient time in which to take a blood pressure record before the inevitable rise in blood pressure. An explanation for the fall in blood pressure is, however, afforded by the observation of Shepley and McGregor (1940) that the heart's activity appears to cease during the passage of the current. This is in all probability the result of stimulation of the vagi and readily accounts for the fall in blood pressure recorded immediately after the fit, for there is little doubt that for some seconds after the fit the heart is not acting with its full functional powers. Support for this view is gained from the work of Urquhart (1927) who reported that an electric current applied to the brain of an animal resulted in arrest of the heart from vagal

inhibition. When the vagi were sectioned or paralysed by atropine this effect was markedly lessened.

| Patient. | Before Fit | Immed. after | 5 mins after | 15 mins. after | 30 mins. after | 60 mins. after |
|---|---|---|---|---|---|---|
| 1. | 92 | 100 | 128 | 100 | 96 | 88 |
| 2. | 72 | 96 | 76 | 96 | 96 | 76 |
| 3. | 84 | 120 | 108 | 96 | 88 | 84 |
| 4. | 80 | 120 | 95 | 90 | 88 | 88 |
| 5. | 98 | 120 | 100 | 98 | 96 | 85 |
| 6. | 76 | 95 | 80 | 82 | 80 | 70 |
| 7. | 80 | 88 | 88 | 82 | 78 | 78 |
| 8. | 120 | 130 | 120 | 115 | 105 | 92 |
| 9. | 88 | 120 | 86 | 92 | 96 | 88 |
| 10. | 70 | 90 | 80 | 78 | 80 | 76 |
| 11. | 80 | 105 | 100 | 95 | 85 | 70 |
| 12. | 75 | 120 | 100 | 95 | 85 | 70 |
| 13. | 90 | 125 | 95 | 80 | 85 | 90 |
| 14. | 84 | 120 | 90 | 90 | 85 | 88 |
| 15. | 65 | 100 | 80 | 60 | 65 | 70 |

TABLE XVI showing the pulse changes accompanying the blood pressure changes in the same patient as Table XV.

Table XVI shows the changes in pulse rate that accompanied the blood pressure changes. In most cases there is a sharp rise in rate immediately after the fit, then a gradual settling to the pre-paroxysmal level. In some cases there is a tendency to oscillate up and down before finally reaching the pre-paroxysmal level in about one hour.

The blood pressure changes have also been recorded in 15 patients after abortive fits. In these, the records were

made before and immediately after the shock only, for in each case the electrodes were re-applied and a major fit induced.

| Patient | Before abortive fit | After abortive fit |
|---|---|---|
| 1. | 140/80 | 105/80 |
| 2. | 110/70 | 130/80 |
| 3. | 205/130 | 210/130 |
| 4. | 130/70 | 160/80 |
| 5. | 140/80 | 120/70 |
| 6. | 150/90 | 135/70 |
| 7. | 130/90 | 140/100 |
| 8. | 175/115 | 130/90 |
| 9. | 140/80 | 160/70 |
| 10. | 125/70 | 135/80 |
| 11. | 100/70 | 110/75 |
| 12. | 130/80 | 145/85 |
| 13. | 130/70 | 140/80 |
| 14. | 100/60 | 120/80 |
| 15. | 125/80 | 130/80 |

TABLE XVII showing the blood pressure changes after abortive fits.

These changes are shown in Table XVII. The most frequent, although not constant change recorded, is an increase of both systolic and diastolic pressures. This is contrary to what one might expect for the abortive fit is in most cases accompanied by flushing of the face which suggests that peripheral dilation, and therefore a fall in blood pressure has occurred. The factors concerned in bringing about this change may be respiratory depression, with accumulation of $CO_2$ in the blood causing stimulation of the carotid sinus and a reflex rise in blood pressure.

| | Blood Pressure | Pulse Rates |
|---|---|---|
| Before abortive fits | 110/60 | 72 |
| After 1st " | 110/70 | 68 |
| " 2nd " | 115/70 | 68 |
| " 3rd " | 115/70 | 60 |
| " 4th " | 115/70 | 80 |
| " 5th " | 110/65 | 72 |
| " 6th " | 110/60 | 70 |

TABLE XVIII showing the change in blood pressure and pulse rate after each of 6 abortive fits.

Records of the blood pressure have also been made in some patients after each of several abortive fits. Table XVIII shows these changes after each of 6 abortive fits in a female patient aged 54. These fits were induced at short intervals on the same day. There is but little change in the blood pressure or pulse rate.

Effect on the heart.

Reference has already been made to the observation of Shepley and McGregor (1940) that during the passage of the shock current the heart appears to be inhibited, for during this period no sounds can be heard over the praecordium, and pulsation at the wrist and in the neck ceases. Reference has also been made to the work of Urquhart (1927) which suggests that this is due to vagal stimulation. It has, unfortunately, for technical and clinical reasons, not been possible to determine the duration of this apparent inhibition. Shepley and McGregor also noted that

the appearance of the electrocardiogram was altered after the fit, the height of the R wave being increased in records taken some minutes after the fit. This feature is not however a constant one In 20 patients electrocardiographic records have been made before the induction of the fit, and compared with records taken immediately after the fit, and also 5 minutes and at one hour afterwards. The most frequent and constantly occurring change noted in the record taken immediately, or as soon as is possible after the fit is a diminution in the amplitude of the R wave. This, as a rule, does not involve all three leads but is mainly confined to leads I and II. This is shown in figure 6. This record also illustrates the increase in heart rate after the fit. An unusual feature, seen in this record, is a well marked U wave which made its appearance immediately after the fit. This is best seen in lead II. Another feature of this record is a slight degree of alternation seen in leads I and II taken after the fit. Figure 7 is an electrocardiogram of the same patient taken at about 5 minutes after the fit. This shows that the heart is returning to its pre-paroxysmal rate, and although a slight degree of alternation is still present the record is closely similar to that taken before the fit, with the exception that the skin currents present in the former, and due to rigidity in a catatonic subject, are absent, in the latter, which was taken while the patient was still unconscious. The U

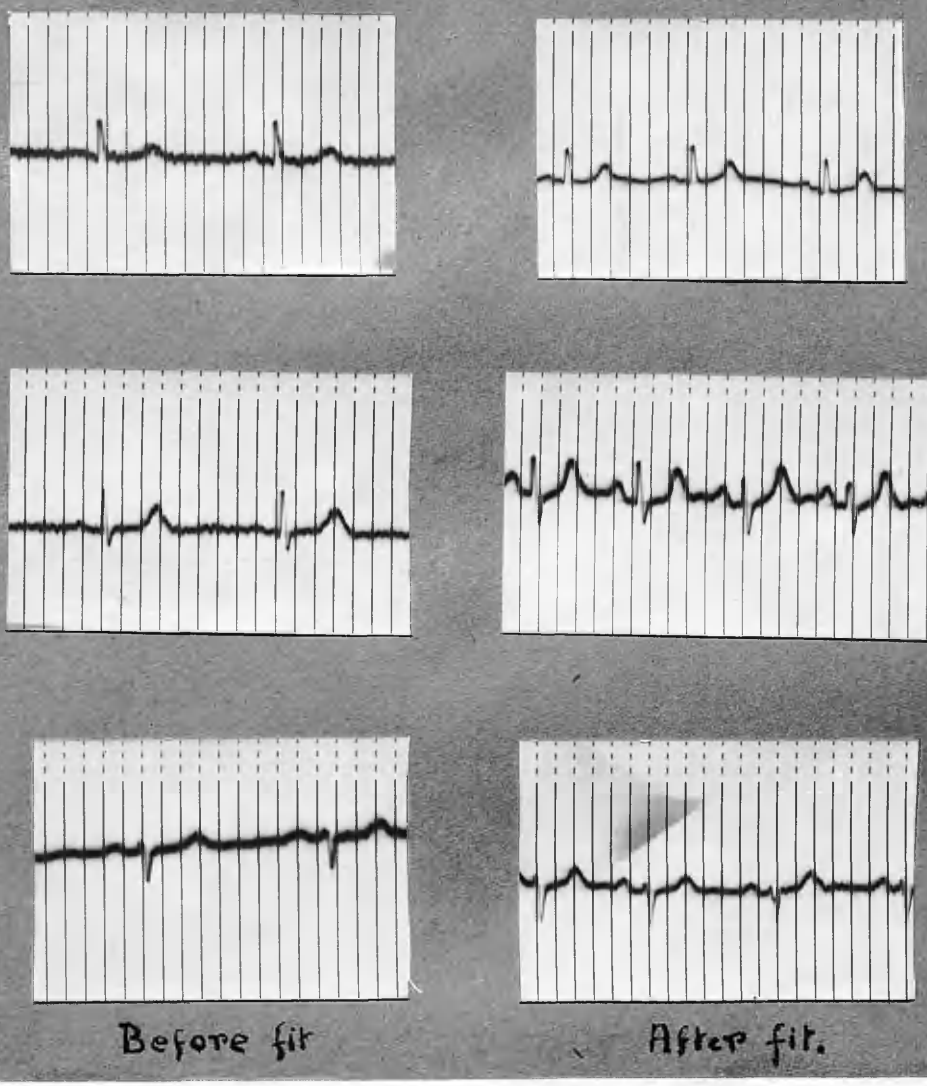



Figure 6. Showing a comparison of E.C.G!s. before and immediately after the fit. The record after the fit shows diminished amplitude of R, and a slight degree of alternation. This record also shows a well marked U wave in lead II.

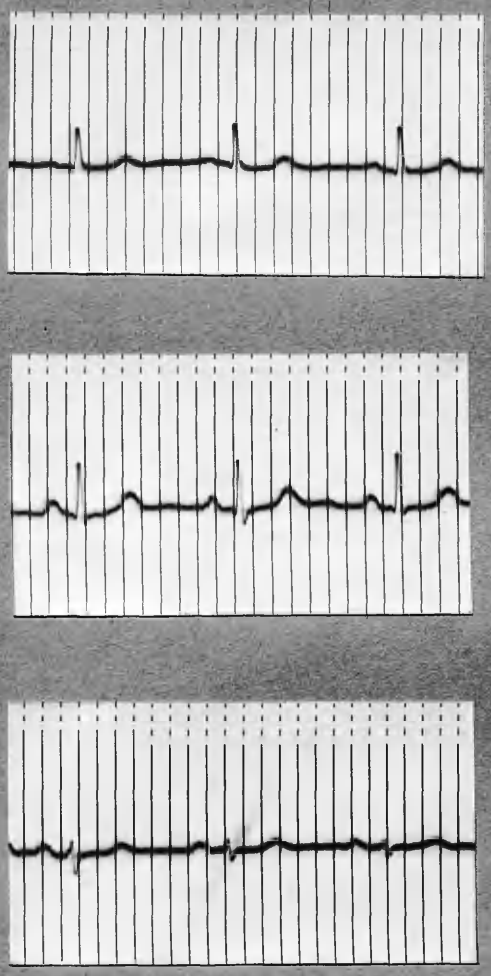


Figure 7. E.C.G. of same patient as Figure 6 taken 5 minutes after the fit. This record closely resembles that taken before the fit.

wave also is no longer distinct.

While these are the more common changes in the electrocardiogram taken after the fit, other records show that the R wave although diminished immediately after the fit, it is increased in records taken about 5 minutes later. This is illustrated in figure 8. R is diminished in height in leads I and II; lead III owing to movements of the patient is unreliable, while in the record taken some minutes after the fit the amplitude of R is increased in leads I and II.

Alterations may also be present in the T wave after the fit. These, chiefly, take the form of increased amplitude, but in one case the direction of T, which in lead III taken before the fit was inverted, had become upright in lead III taken immediately after the fit. While in the record taken some 5 minutes later T was again inverted or absent. This is shown in figure 9. Another feature of these records is that the P wave which cannot be identified in Lead III before the fit owing perhaps to its being small and obscured by skin currents, is well marked in the record taken after the fit.

The mechanisms concerned in bringing about these changes in the electrocardiogram are by no means clear. It is possible that the marked diminution in the amplitude of R so constantly present in records taken immediately after the fit is the result of diminished cardiac activity due to vagal inhibition, and the increased amplitude of R which is seen in later records may be

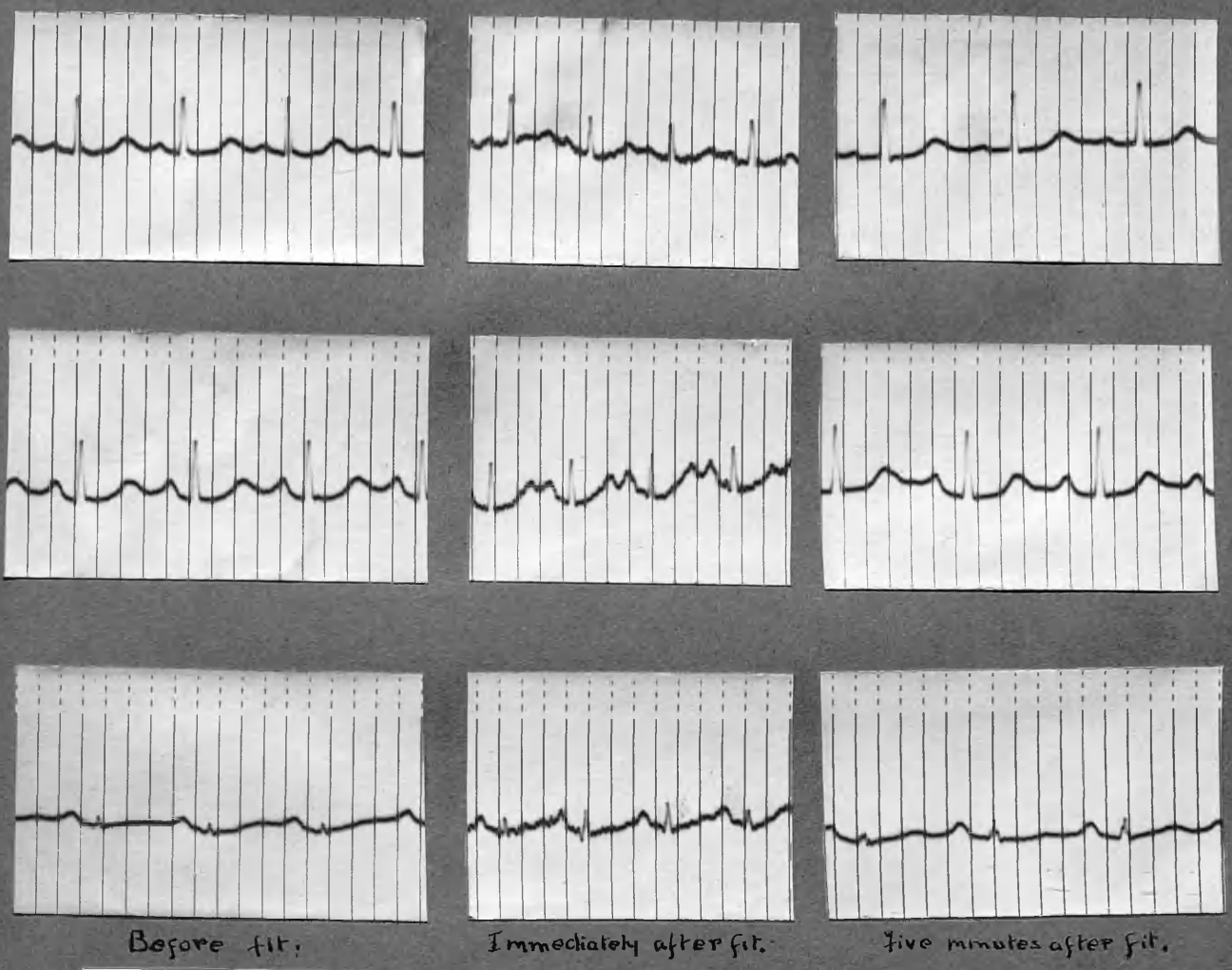



Figure 8. These records illustrate diminution in the amplitude of R in records taken immediately after the fit, while in the record taken 5 minutes after the amplitude of R is increased.

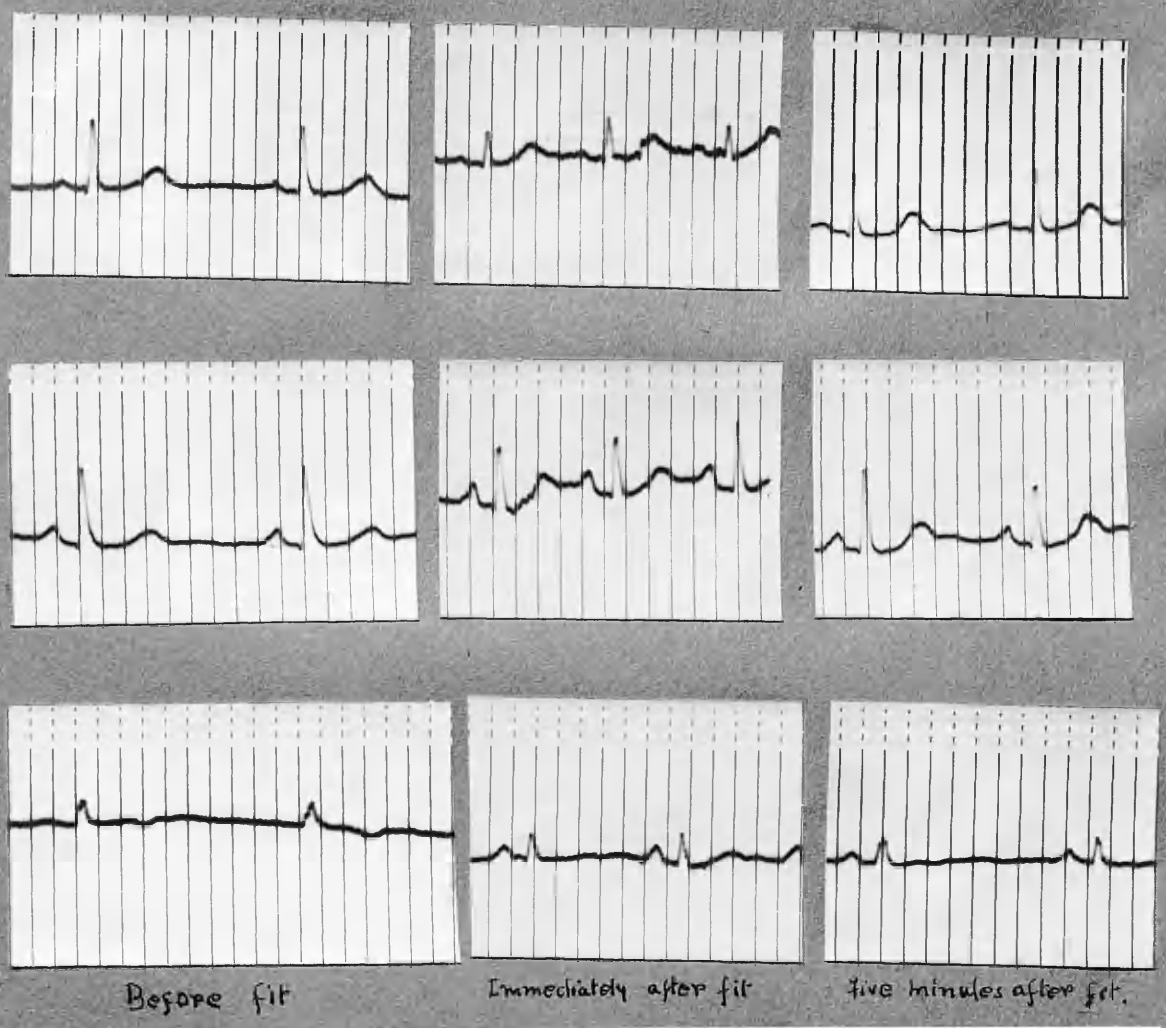



Figure 9. These records show the change in the direction of the T wave, inverted before, and upright after the fit. In the record taken 5 minutes after T is again inverted.

due to a compensatory increase in cardiac activity following the period of inhibition. The alterations in the T wave are probably of little significance for most cardiologists are agreed that both in its direction and form the T wave is much less stable than the initial deflections, and it has been shown that vagal or sympathetic stimulation may profoundly affect the end deflection while Q.R.S. are but little altered.

It has been suggested by Sigler and Schneider (1936) that alterations in the electrical potentials of the heart might result from an electrical accident. These writers reported on the electrocardiographic changes occurring in a man aged 36 years who had sustained an electrical accident which had rendered him unconscious for 2 minutes. On admission to hospital this patient did not appear ill and the only abnormality noted was complete irregularity of the pulse. An electrocardiogram taken soon after admission showed auricular fibrillation with a ventricular rate of 110 and a tendency to left axis deviation. A record taken 3 hours later was normal with an auricular and ventricular rate of 75 beats per minute. The P.R. interval was 0.16 of a second and in lead I, R was of small amplitude while in lead II the amplitude of R was greater than the record taken on admission. The following day the P.R. interval was 0.2 second and there was left axis deviation. After varying from day to day the condition settled down, and the records showed only a slight tendency to left axis

deviation No clinical evidence of heart disease was found and Sigler and Schneider suggested that the changes might be due to alteration in the electrical conductivity of the heart. Since the changes described are in some respects similar to those seen after the application of the current to the head in inducing the epileptiform convulsion I decided to determine whether or not any alteration had occurred in the conductivity of the blood, for it is by this route most probably, that the current would reach the heart if indeed it does so at all. In its electrical conductivity the blood was shown by Strohl (1931) to be second only to the cerebro-spinal fluid which Eckel (1926) showed was the best electrical conductor of all the body fluids or tissues.

For the purpose of this investigation a conductivity cell was constructed. This consisted of platinum electrodes attached to fine glass tubes. Contact with the electrodes was established by means of mercury with which the tubes were filled. The remainder of the cell was a small test tube. The conductivity of the blood serum was obtained from the formula

$$\text{Conductivity} = R \times K$$

where R = the resistance of the serum as determined by balancing a conductivity bridge, consisting of wheatstone bridge, and vibration galvonometer. K = the cell constant. This was determined by finding the resistance of a known electrolyte, N/50$^{th}$ KCl, the conductivity of which is 0.00229 at 16$^{o}$C. K was

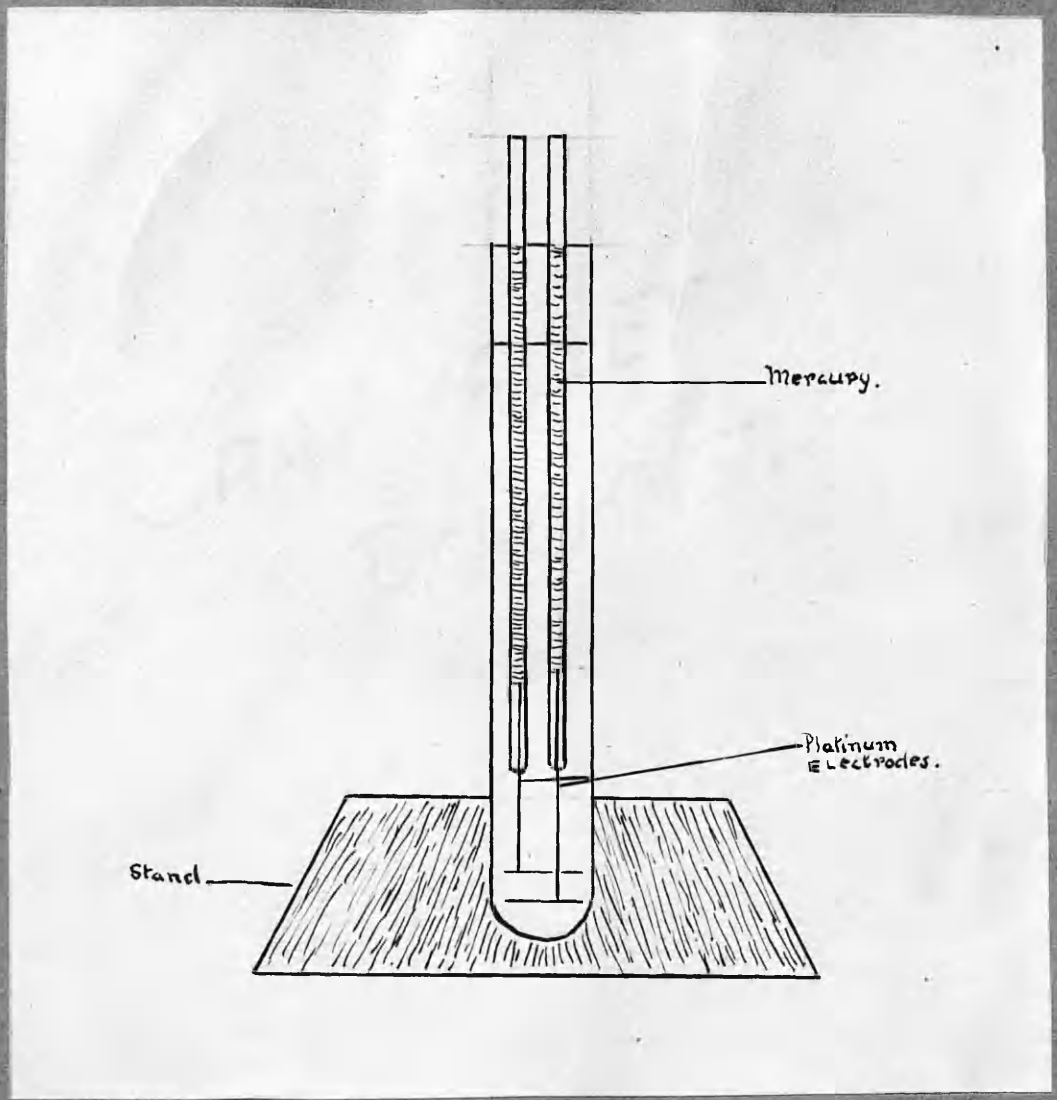



Figure 10. Diagram of conductivity cell.

then obtained from the formula

$$K = \frac{0.00229}{R}$$

where R = the resistance of the KCl solution determined on the bridge.

The conductivity of the blood serum of 10 patients before and after the application of the current as determined in this way is shown in Table XIX.

| | Before fit | | After fit | |
|---|---|---|---|---|
| 1. | 0.008790 | mhos | 0.008790 | mhos |
| 2. | 0.002156 | " | 0.001675 | " |
| 3. | 0.008790 | " | 0.008790 | " |
| 4. | 0.007780 | " | 0.006580 | " |
| 5. | 0.008350 | " | 0.008355 | " |
| 6. | 0.007960 | " | 0.007690 | " |
| 7. | 0.008970 | " | 0.008670 | " |
| 8. | 0.008650 | " | 0.008650 | " |
| 9. | 0.007963 | " | 0.007850 | " |
| 10. | 0.007860 | " | 0.007860 | " |

TABLE XIX showing a comparison of the conductivities of the blood serum before and after the induction of a fit in reciprocal ohms (mhos).

Six of these sera show no alteration in conductivity, while in four there is a variation of 0.003 - 0.005 reciprocal ohms. These latter must be regarded as too slight to be considered seriously, being well within the margin of error for the

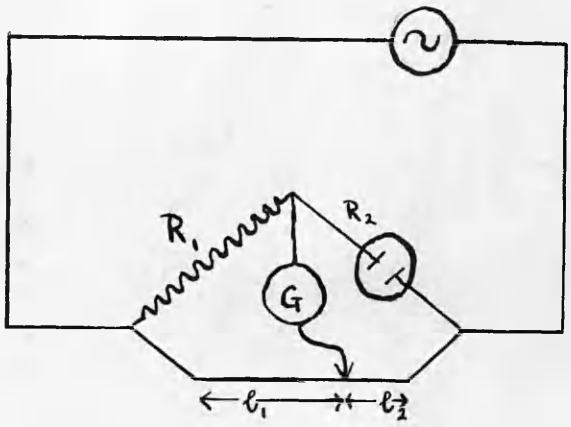



Figure 11. Diagram of bridge used for measurement of conductivity.

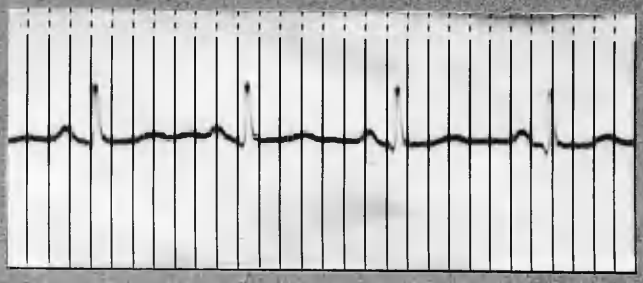
instruments used in this investigation. This is borne out by quantitative analysis of the blood serum electrolytes of which the chief is the chlorides.

| Patient | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| Before | 0.93. | 0.85. | 0.50. | 0.48. | 0.36. | 0.35. | 0.39. | 0.73. | 0.81. | 0.80 |
| After | 0.90. | 0.93. | 0.50. | 0.48. | 0.36. | 0.35. | 0.39. | 0.73. | 0.81. | 0.73 |

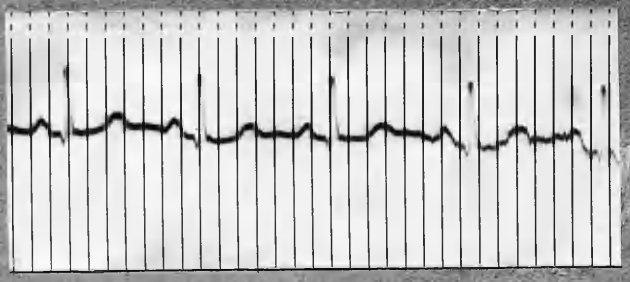
TABLE XX showing the blood chloride expressed as gms % Na.Cl before and after the fit.

Table XX shows the total chlorides in ten patients before and immediately after the major fit. In these also the variations are within the margin of experimental error, and cannot be regarded as due to alterations in the chloride content of the blood. Similar results were obtained with specimens of cerebro-spinal fluid examined both before and after the application of the current. Figures 10 and 11 show diagrammatically the conductivity cell and the wiring of the conductivity bridge respectively.

With regard to the effect of the abortive fit on the heart, the electrocardiographic changes recorded are much less marked, increased amplitude of R only having been observed. Figure 12. This suggests the explanation that those changes recorded after the major fit are the result of the fit and not of the agent used to induce the fit. While this explanation is a possibility, similar changes should emerge after artificial



Before fit.



After fit.

Figure 12. Showing increases amplitude of the R wave after an abortive shock.

fits, no matter what agent has been used, and these have not been reported to my knowledge. One is therefore forced to the alternative explanation that they result only when the current is of sufficient intensity to induce a major fit.

Effect on the blood sugar.

An increase occurs in the blood sugar following the application of the shock current whether of sufficient intensity to induce a major fit, or not. This is present in blood specimens taken immediately after the shock, and in specimens taken up to half an hour after the shock, when it gradually returns to the fasting level. This isshown in Tables XXI and XXII after major and abortive fits respectively. Both Tables show fasting blood sugar levels much less than the normal 80 - 120 mgms %. This is due most probably to the long fast of our patients who have their evening meal at 5 p.m., and were kept fasting, for this series of investigations, until after treatment. To determine whether a spontaneous rise in blood sugar might not be occurring, blood specimens were collected at 15 minute intervals before the application of the current in 5 patients.

| Patient | Before | Immediately after | 15 mins. after | 30 mins. after | 45 mins. after | 60 mins. after |
|---|---|---|---|---|---|---|
| 1. | 69 | 80 | 118 | 115 | 111 | 90 |
| 2. | 64 | 71 | 77 | 118 | 74 | 57 |
| 3. | 56 | 60 | 67 | 71 | 62 | 58 |
| 4. | 69 | 74 | 77 | 80 | 70 | 50 |
| 5. | 59 | 67 | 87 | 80 | 65 | 57 |
| 6. | 43 | 53 | 71 | 83 | 70 | 56 |
| 7. | 45 | 53 | 67 | 62 | 50 | 48 |
| 8. | 60 | 69 | 77 | 71 | 68 | 60 |
| 9. | 79 | 89 | 90 | 77 | 70 | 76 |
| 10. | 66 | 71 | 71 | 60 | 50 | 50 |

TABLE XXI showing the effect on the blood sugar of a shock which induced a major fit. Blood sugar in mgms %.

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| Before | 69 | 60 | 70 | 70 | 85 | 65 | 69 | 56 | 80 | 110 |
| After | 74 | 65 | 74 | 80 | 95 | 70 | 74 | 50 | 85 | 120 |

TABLE XXII showing the effect on the blood sugar of a shock which induced an abortive fit only. Blood sugar in mgms %.

The results are showing in Table XXIII.

| Patient | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| ½ hour before | 61 | 73 | 65 | 70 | 55 |
| 15 mins. " | 61 | 70 | 63 | 72 | 56 |
| Immed. " | 61 | 70 | 63 | 73 | 56 |
| " after | 67 | 80 | 70 | 83 | 77 |
| 15 mins. " | 80 | 85 | 80 | 90 | 71 |
| 30 " " | 87 | 98 | 85 | 100 | 71 |
| 45 " " | 83 | 90 | 75 | 85 | 66 |
| 60 " " | 75 | 80 | 70 | 80 | 60 |

TABLE XXIII showing that no spontaneous increase in the blood sugar was occurring in the half hour preceding the shock. Blood sugar in mgms %.

which shows clearly that no significant change occurring in the blood sugar before the fit and therefore exludes the possibility of any spontaneous increase in blood sugar having occurred. Even more marked is the increase in blood sugar that follows the fit when it is induced in patients during insulin coma. Comparison has been made of these increases, in the same patient, when the fit was induced during coma, and on a rest day from insulin treatment. This was shown in figure 13.

Similar changes in the blood sugar have been observed by Bömer (1930), and Demole and Bersot (1937) in experimental animals during cardiazol fits, and by Georgi and Strauss (1937)

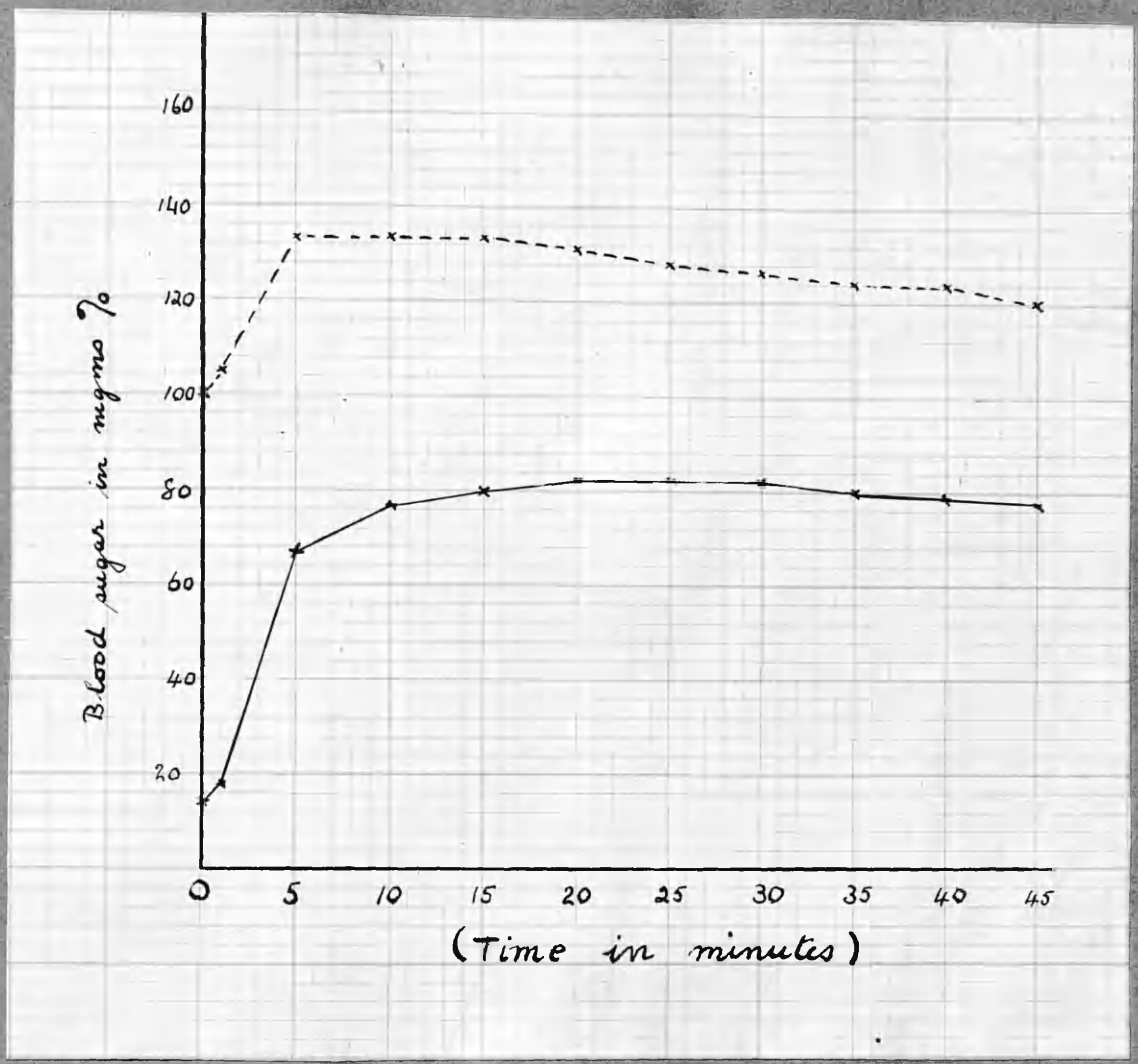



Figure 13. Showing the effect of the application of the current on the blood sugar of the same patient when in insulin coma and on a rest day from insulin treatment.

———— Coma.
-------- Rest day.

and Harris (1938) in schizophrenic patients undergoing cardiazol treatment. Harris, who considers that the cardiazol convulsion is a mixed sympathetic and parasympathetic discharge, expressed the opinion that it is this which produces the rise in blood sugar. Holmstrom (1924) and Lennox, O'Connor and Bellinger (1927), however, observed an increase in the blood sugar of epileptics after spontaneous convulsions. This, the latter workers considered was due to muscular exertion and asphyxia. This explanation, while it might account for the increased blood sugar after a major fit induced by the electrical method, hardly suffices to account for the blood sugar increase after the abortive fit, for in this there may be no muscular movements at all, and although cyanosis frequently develops it only does so after an interval, which allows of blood specimens being taken. These also showed a rise in blood sugar. It is therefore reasonable to exclude muscular exertion and asphyxia as the causes of this increase and to suppose that it is mediated by the sympathetico-adrenal system, through stimulation of the brain stem, which is known to be capable of influencing glocygenolysis, or of the hypothalamus which recent work has shown exerts an influence on carbohydrate metabolism.

Effect on the gastric juice.

Observations have also been made on the gastric juice before and after both major and abortive fits. With a few

exceptions these observations were made on patients undergoing insulin treatment, owing to the ease with which the gastric juice could be withdrawn while the patient was in coma. The result of these investigations showed that in every case the shock caused a marked drop in the gastric acidity. Tables XXIV and XXV show these results after major and minor fits respectively.

| Patient | % Free HCl | Total Acids | Chlorides gms.% NaCl |
|---|---|---|---|
| I. (Before shock | 0.33 | 110 | 0.84 |
| (Immed. after | 0.18 | 64 | 0.47 |
| (5 Mins. " | 0.32 | 110 | 0.80 |
| II. (Before shock | 0.41 | 130 | 0.83 |
| (Immed. after | 0.22 | 76 | 0.35 |
| (5 mins. " | 0.18 | 68 | 0.37 |
| III. (Before shock | 0.18 | 64 | 0.70 |
| (Immed. after | 0.14 | 56 | 0.62 |
| (5 Mins. " | 0.14 | 58 | 0.63 |
| IV. (Before shock | 0.36 | 120 | 0.82 |
| (Immed. after | 0.20 | 66 | 0.38 |
| (5 Mins. " | 0.14 | 52 | 0.70 |
| V. (Before shock | 0.12 | 46 | 0.59 |
| (Immed. after | Trace | 22 | 0.46 |
| (5 mins. " | Trace | 20 | 0.65 |
| VI. (Before shock | - | 8 | 0.38 |
| (Immed. after | - | 6 | 0.28 |
| (5 Mins. " | - | 6 | 0.24 |
| VII. (Before shock | 0.50 | 135 | 0.84 |
| (Immed. after | 0.40 | 80 | 0.50 |
| (5 Mins. " | 0.33 | 73 | 0.35 |

TABLE XXIV showing the effect of the shock current on the gastric juice. Numbers VI and VII are non-insulin patients, the others were in insulin coma. Major fit resulted in each case.

| Patient | | % Free HCl | Total Acids | Chlorides gms.% NaCl |
|---|---|---|---|---|
| I. | (Before shock | 0.19 | 68 | 0.52 |
| | (After " | 0.08 | 36 | 0.40 |
| II. | (Before shock | 0.44 | 140 | 0.82 |
| | (After " | 0.43 | 135 | 0.82 |
| III. | (Before shock | 0.51 | 120 | 0.68 |
| | (After " | 0.45 | 105 | 0.60 |
| IV. | (Before shock | 0.44 | 125 | 0.83 |
| | (After " | 0.38 | 115 | 0.78 |
| V. | (Before shock | 0.30 | 80 | 0.60 |
| | (After " | 0.26 | 73 | 0.52 |

TABLE XXV showing effect of shock on gastric juice when abortive fit only resulted. Patients in insulin coma.

Table XXIV includes two non-insulin patients numbers 6 and 7. Comparison of these tables shows that a major fit causes a much greater drop in acidity than the abortive fit. This point is further illustrated in table XXVI which shows the effect of two abortive fits followed by a major fit. This clearly suggests that the maximum effect on the gastric acidity is only obtained when the shock current is at or near to the convulsion threshold, for the second abortive fit which was nearer convulsion threshold than the first produced a much greater drop in gastric acidity.

This action of the electric current, or its effects, in reducing gastric acidity is of particular interest especially in the case of hypoglycaemic patients, for it has been shown by Labbe (1924), Detre and Sivo (1925) De Anciaes (1926), Fonesco

| Patient | % Free HCl | Total Acids | Chlorides as gms.% NaCl |
|---|---|---|---|
| Before | 0.44 | 140 | 0.82 |
| After 1st Abortive Fit | 0.43 | 138 | 0.82 |
| " 2nd " " | 0.33 | 104 | 0.75 |
| " Major Fit | 0.27 | 84 | 0.61 |
| 5 Mins. after Major Fit | 0.22 | 80 | 0.70 |

TABLE XXVI showing the effect of two abortive fits followed by a major fit on the gastric juice.

and Carvallo (1927), Simici , Popesco and Diculero (1927), Mustante (1929), Okada (1929), La Barre and Cespedes (1931), Boldyreff and Stewart (1932) Hopstein (1932), Freudenberg (1938), and many others that insulin increases gastric acidity. This effect of insulin on the gastric juice of 5 schizophrenic subjects is shown in Table XXVII. It will be seen that there is first of all a drop in gastric acidity, which begins to rise towards the onset of coma, until before termination the gastric acidity is much higher than the fasting level. In case number 4 table XXVII the gastric acidity is co-related with the blood chlorides and blood glucose. Opinion is divided as to the mechanism by which insulin increases gastric acidity. Mustante (1929) and others are of the opinion that it acts in the same way as histamine, which was shown by Popielski (1920) to induce copious gastric secretion by acting directly on the secretory cells of the stomach. Others, however, among whom Okada (1929),

110 units intra muscular Insulin. Coma in 130 minutes.

| | Time in minutes | 30 | 45 | 50 | 80 | 215 | 220 | 225 | 235 | 245 | 265 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| I. | Gastric Juice (Free HCl% | 0.10 | 0.06 | 0.77 | - | 0.17 | 80cc Intravenous 33% glucose | Nil | Nil | Nil | Nil |
| | Gastric Juice (Total Acidity | 47 | 27 | 31 | 13 | 61 | | 15 | 67 | 5 | 8 |
| | Gastric Juice (Chlorides (Mgms.% NaCl) | 530 | 376 | 485 | 299 | 660 | | 650 | 364 | 318 | - |
| | Blood Sugar | | | | | | | 71 mgms.% | | | |

20 units intramuscular Insulin. No coma.

| | Time in minutes | Resting | 90 | 130 | 135 | 140 | 150 | 170 | 180 |
|---|---|---|---|---|---|---|---|---|---|
| II. | Gastric Juice (Free HCl% | Nil | Nil | 0.055 | 20cc I.V. 33% glucose | Nil | Nil | Nil | Nil |
| | Gastric Juice (Total Acidity | 10 | 11 | 27.5 | | 20 | 30 | 20 | 20 |
| | Gastric Juice (Chlorides (mgms. % NaCl | 376 | 634 | 586 | | 634 | 561 | 515 | 445 |

66 units intramuscular Insulin.

| | Time in minutes. | Resting | 7 | 15 | 25 | 97 | 202 | 205 | 212 | 232 |
|---|---|---|---|---|---|---|---|---|---|---|
| III. | Gastric Juice (Free HCl % | 0.11 | 0.10 | 0.14 | 0.157 | Coma | 0.205 | 40cc I.V. 33% glucose | 0.12 | Nil |
| | Gastric Juice (Total acidity | 34 | 41 | 50 | 56 | | 95 | | 46 | 10 |
| | Gastric Juice (Chlorides ((Mgms.% NaCl) | - | - | - | - | | - | | - | - |

94 units intramuscular Insulin (Coma 130 mins.)

| | Time in minutes | Resting | 60 | 90 | 120 | 150 | 180 | 215 | 240 | 260 |
|---|---|---|---|---|---|---|---|---|---|---|
| IV. | Gastric Juice (Free HCl% | 0.73 | 0.14 | 0.03 | 0.051 | 0.11 | 0.234 | 60cc.I.V. glucose 33% | 0.11 | - |
| | Gastric Juice (Total acidity | 40 | 20 | 26 | 30 | 42 | 76 | | 43 | 10 |
| | Gastric Juice (Chlorides ((Mgms % NaCl) | 726 | 635 | 600 | 680 | 730 | 835 | | 515 | 542 |
| | Blood Sugar | 52 | 58 | 52 | 33 | 11 | 9 | | - | 67 |
| | " Chlorides | 565 | 587 | 550 | - | 600 | 535 | | - | 587 |

110 units intramuscular Insulin. Coma in 110 Mins.

| | Time in minutes | Resting | 30 | 60 | 90 | 120,135,150,165,210,220,230 |
|---|---|---|---|---|---|---|
| V. | Gastric Juice (Free HCl % | 0.10 | 0.073 | 0.2 | 0.2 | 0.14 - 0.175 - 0.163 20cc.I.V.Glucose 0.11 |
| | Gastric Juice (Total acidity | 35 | 30 | 64 | 68 | 50 - 62 - 58 40 |
| | Gastric Juice (Chlorides ((mgms.% NaCl) | 675 | 600 | 764 | 764 | 734 - 500 - 722 710 |
| | Blood Chloride | 468 | - | - | - | - 480 - 577,452 535 |

TABLE XXVII showing increased gastric acidity following insulin intramuscularly, and decrease in gastric acidity after intravenous glucose.

Dobreff (1931) and La Barre and Cespedes (1931) take the view that the hyperacidity that follows the exhibition of insulin is caused by the resulting hypoglycaemia. With this view, Fonesico and Guerico (1930), Meyer (1930), Hopstein (1932) and Boldyreff and Stewart (1932) who observed increased gastric acidity in the absence of hypoglycaemia do not agree. Nevertheless it appears that some degree of hypoglycaemia must be present before any increase in gastric acidity occurs for fasting subjects with relatively low blood sugars often show achlorhydria not hyperacidity. Negative confirmation of this view is obtained from the work of Bloomfield and Pollard (1933) who have reviewed the literature dealing with gastric acidity in diabetes mellitus with the results shown in Table XXVIII, which show that of a total of 357 diabetic subjects 124 were achlorhydric, of this number 48 however were suffering from pernicious anaemia. This view that increased gastric acidity is dependent on some degree of hypoglycaemia gains further support from Table XXVII, case number 4, which demonstrates that it is only after the blood sugar has reached the level of 11 mgms.% that increased gastric acidity occurs. La Barre and Cespedes (1931) showed by experiments on dogs that the hypoglycaemia resulting from insulin administration increased the gastric acidity by stimulation of the vagi. This was confirmed by Boldyreff and Stewart (1932) who showed that atropine abolished this effect of the hypoglycaemia, while the secretory

| | | | | |
|---|---|---|---|---|
| Bowen and Aaron (1926) | in 29% | of 66 | Diabetic | patients. |
| McPherson (1927) | in 40% | of 10 | " | " |
| Rabinowitch, Fowler & Watson (1931) | in 39% | of 100 | " | " |
| Wiechman (1929) | in 32% | of 25 | " | " |
| Wohl (1931) | in 33.3% | 33 | " | " |
| Root (1931) | in 24.3% | 37 | " | " |
| Moore (1932) | in 39.7% | 83 | " | " |

TABLE XXVIII showing the incidence of achlorhydria among diabetic patients. Quoted from Bloomfield and Pollard.

cells of the stomach still responded to the direct action of histamine by producing a copious acid secretion.

Returning to the problem presented by the reduction of gastric acidity effected by the electric current, or its effects, reference to Table XXVII shows that soon after the administration of intravenous glucose the gastric hyperacidity effected by insulin is reduced. It might therefore be supposed that a similar mechanism was operating in effecting the reduction in the case of the electric current, for it has already been shown that this is accompanied by an increase in blood sugar. Table XXIX shows the effect of the current on both gastric acidity and blood sugar. The increase in blood sugar effected by the electric shock is, however, so small compared with the amount of intravenous glucose given, ofbetween 20-60 ccs of 33%, that it is unlikely that it alone is responsible for the very marked

| Patient | | % Free HCl | Total acids | Chlorides | Blood Sugar |
|---|---|---|---|---|---|
| I. | (Before shock | 0.33 | 110 | 0.84 | 18 mgms.% |
| | (After shock | 0.18 | 64 | 0.47 | 24 " |
| II. | (Before shock | 0.19 | 68 | 0.52 | 17 " |
| | (After shock | 0.08 | 36 | 0.40 | 25 " |
| III. | (Before shock | 0.145 | 66 | 0.49 | 25 " |
| | (After shock | 0.140 | 54 | 0.40 | 32 " |
| IV. | (Before shock | 0.340 | 112 | 0.66 | 15 " |
| | (After shock | 0.250 | 85 | 0.56 | 40 " |
| V. | (Before shock | 0.360 | 115 | 0.72 | 22 " |
| | (After shock | 0.230 | 70 | 0.45 | 31 " |

TABLE XXIX showing the effect of the shock on both gastric acidity and blood sugar. In each case the gastric acidity is reduced and the blood sugar increases.

drop in gastric acidity effected. It therefore seems reasonable to suppose that both the increase in blood sugar and the decrease in gastric acidity resulting from the electric current are brought about in the same way, namely by stimulation of the sympathetic centres either in the brain stem or in the hypothalmus.

Effect on Temperature.

Meduna (1935) observed slight variations in temperature following the cardiazol convulsion. These however he regarded as of no importance. Similar results have followed the electrically induced convulsion. A morning and evening

temperature chart was kept of all patients during treatment. No rise was ever noted that could not be accounted for by intercurrent disease. In a good number of patients temperature records were taken at 15 minute intervals for 1 hour after the induction of the fit, again with negative results.

Effect on the urine.

No changes have been observed in the urine on qualitative analysis of the urine of all patients undergoing this form of treatment.

## COMPLICATIONS AND CONTRA-INDICATIONS OF ELECTRICALLY INDUCED CONVULSIONS

The most constantly occurring complication occurring with the electrically induced convulsions is dislocation of the jaw. As with cardiazol this is readily reduced and no complaints have resulted from its occurrence. No fractures have occurred in our series of cases, although all patients complaining of pain or discomfort after treatment have been subjected to X-ray examination. Golla, Fleming and Walter (1940) however have reported the occurrence of vertebral fractures in two patients, so it cannot be said that this disadvantage of convulsion therapy has been eliminated. One feels, nevertheless, that the degree of severity of the electrical fit is less likely to give rise to fractures than the cardiazol fit, and it is possible that if the "start" re-action described by Golla, Fleming and Walter were eliminated that the tendency to vertebral fractures would be diminished, if not entirely eliminated.

With regard to the major disadvantage of the pharmacological convulsants that of fear, it can be asserted that this has been entirely eliminated by the electrical convulsion method. A few patients become mildly apprehensive on treatment mornings, but these can always be readily reassured. This apprehension appears to be conditioned by the amnesia which is invariably present on recovering from the fit.

In the early days of electrical convulsion treatment we were much concerned by complaints of loss of memory for normally well remembered things, such as addresses of relatives, made by a few patients. This however proved to be transitory and in every case, as far as could be determined, there was no permanent memory defect, indeed all patients who made this complaint have been discharged and are back at work.

Contra-indications.

Latent tubercle would appear to be a contra-indication for one patient a female aged 20 years undergoing combined insulin and electrical convulsion treatment, developed clinical signs of tuberculous infection of the respiratory tract, although the tubercle bacillus was not isolated. This patient had a doubtful history of tuberculous infection. In this case, the only one of the number treated to develop physical signs, the issue is complicated by the fact that she was also undergoing insulin treatment.

One apparent contra-indication to electrical convulsion treatment that does not appear to have emerged with the pharmacological convulsants is the presence of eye disease. One patient a female suffering from involutional melancholia and co-existant disease of the retinae of vascular origin was referred to the consulting ophthalmologist for an opinion as to the advisability of electrical convulsion treatment, this

was vetoed on the grounds that it might be harmful to the patient's sight, already in jeopardy.

Although the electrical convulsion has been shown to have a profound transitory effect on the function of the heart, disease of that organ does not necessarily contra-indicate treatment as the following illustrates. J.B. a male patient aged 61 years was admitted suffering from involutional melancholia. He was acutely suicidal and was placed on suicidal caution on admission. Physically he was in fair bodily health, but showed complete irregularity of the pulse and a pulse deficit. A diagnosis of auricular fibrillation was made. It was not possible to make an electro-cardiographic examination before treatment owing to his extreme agitation. After consultation with his relatives who were warned of the risk of convulsion treatment (treatment is only undertaken with the consent and co-operation of the relatives) two abortive and three major fits were induced in 4 days of treatment. Recovery was complete by this time and the patient is now awaiting his discharge. No sign of adverse effect was noted in his heart. Electrocardiographic examination has now been made. This is shown in figure 14, which shows irregularity of the Q.R.S. complex and very obscure auricular waves.

Providing the physical health of the patient is fair and there is no gross arterio-sclerosis the electrical convulsion treatment may be applied at almost any age. The oldest so far treated is 62 years of age, though there were several between 58-60 years.

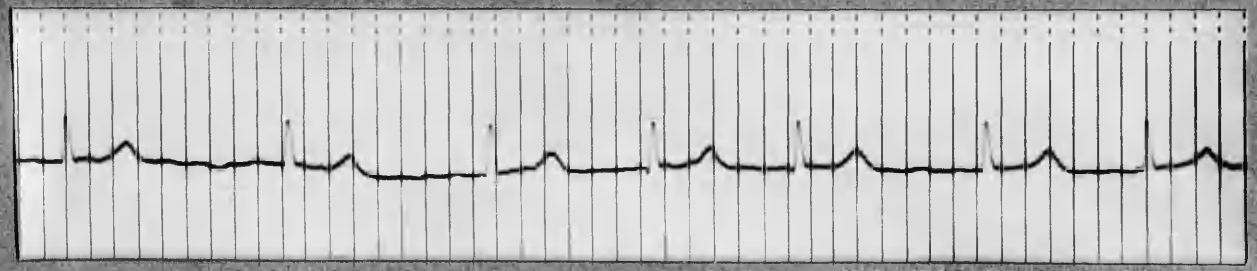


Figure 14. Showing irregularity of the Q.R.S. complex and obscure P waves.

## SELECTION OF CASES

Until experience with the electrical convulsion method was gained many of the patients selected for treatment were chronic schizophrenics many of whom had failed to respond either to cardiazol, triazol or insulin treatment. Since then however patients have been selected for treatment on a more rational basis. In a discussion of the indications and contra-indications for convulsion therapy Shepley and McGregor (1940) observed that the term schizophrenia has been so widely applied as to be in danger of losing precise meaning. Support for this view is gained from the work of Langfelft (1939) who has described a great many psychotic variations under the term "Schizophreniform States". It was further observed by Shepley and McGregor that it is of fundamental importance to differentiate two types of schizophrenia, a relatively benign or exogenous form, and a malignant or endogenous form. The former group includes cases whose heredity is good and whose physical and mental constitution is well developed. In these the illness is often precipitated by environmental factors and the prognosis is good. In the latter group are found individuals of pronounced constitutional inferiority associated with poorly developed internal organs. These show physical signs of cardio-vascular hypoplasia and hypofunction together with general asthenia and poorly developed sex characteristics. These individuals morphologically tend towards the asthenico-athletic

or dysplastic groups described by Kretschmer (1925) and show in their cold blue extremities, and tendency to oedema physical signs of their poverty of organic endowment. Examination by orthodiography, in these individuals shows generalised smallness of the internal viscura and more particularly the heart and aorta. Mentally these patients show extreme apathy, self-absorption and poverty of affect. In these the prognosis is from the outset poor and they form the major portion of the average mental hospital population. This group appears to be resistant to any form of shock treatment and probably belong to what the older psychiatrists called dementia praecox.

Among the group of exogenous schizophrenics are found cases of stupor, both depressive and catatonic and a wider group designated by Shepley and McGregor (1940) as schizoid depressives. This is a group of individuals showing persistent depression without psycho-motor retardation. Physically they are for the most part of asthenico-athletic habitus. In contrast to the endogenous group of schizophrenics they are well developed and present none of the physical signs of organic asthenia which is such a prominent feature of the endogenous variety. They respond very well to convulsion treatment whether it be cardiazol, triazol or electrical.

The involutional psychoses also have a good prognosis with electrical convulsion treatment.

In selecting patients for convulsion treatment regard

is had to the physical and mental constitution of the patient, the presence of exogenous precipitating factors and the presence or absence of a strong affective element colouring the psychosis.

## NUMBER OF TREATMENTS

Meduna (1935) expressed the opinion that at least 20 major fits should be induced before regarding a case as having a bad prognosis and later (1938) he condemned those workers who considered 10 major fits enough and insisted that at least 20 - 25 major fits should be induced before regarding the case as hopeless. He frequently induced as many as 30 - 35 major convulsions at this time. In all my experience with cardiazol I have not seen one patient recover after 20 or more major convulsions unless the improvement had manifested itself at a much earlier stage of the treatment and I had reached the conclusion, in company with many other workers, that if convulsion therapy was going to prove effective it would cause considerable improvement in the patient by the third or fourth major fit, and if there was no marked improvement by the time 10 major convulsions had been induced that it was of little use carrying on any longer. Recently however my confidence in this belief was shaken when a patient undergoing electrical convulsion treatment began to improve after the 15th convulsion and made a complete recovery. This patient has now left the hospital. This, however, I regard as an exception and as a general guide it can be accepted that if a remission is going to be effected marked improvement will have occurred by the time 10 major fits have been induced. Nevertheless, I am in agreement with Meduna (1935, 1938) that it is a mistake to terminate the treatment as

soon as a remission had been obtained for to do so frequently results in an early relapse. Treatment is therefore continued until 3 or 4 more convulsions have been induced after apparent normality had been reached.

With regard to the number of convulsions that can safely be induced by the electrical method there appears to be no limit. Under the term "maintenance treatment" Shepley and McGregor (1940) described several patients who required treatment at intervals of about two weeks in order to prevent relapse. Some of these patients have now had 40 or more convulsions with no apparent adverse mental or physical effects.

Convulsions are induced twice weekly in the majority of cases. When apparent normality had been reached treatment is usually carried out once a week for 2 or 3 weeks in order to ensure stabilisation and prevent the relapse that so commonly occurs when treatment is suddenly discontinued as soon as an apparent remission has been effected. Less commonly the last 3 or 4 treatments are given at bi-weekly intervals.

## RESULTS OF ELECTRIC CONVULSION TREATMENT

During the past year 103 patients have been treated by electric convulsions. Of this number 72 were females and 31 males, and of the total 46 (44.6%) have recovered and left the hospital or are awaiting discharge. The remainder show but little change or are slightly improved. Of the 46 recoveries 31 were females and 15 males or 43% and 48.3% respectively. On further analysis, with reference to the duration of the illness, the results will be seen to be much better than the figure 44.6% for the total number of patients suggests, for of the 103 patients whose treatment has been completed 33 have been in hospital for periods of 2 to 10 years, and belong to a group of shock resistant cases who had previously failed to respond to cardiazol, triazol, or insulin treatment. If these, then, are excluded from the total of 103 patients, the percentage of remissions obtained among the remaining 70 patients is 65.7%. When the duration of the illness is still further considered these percentages will be seen to be improved on with those illnesses of short duration. This is shown in Table XXX.

When the type of illness is t aken into consideration it can be shown that the prognosis in some psychoses is much more favourable than others. This is shown in Table XXXI.

| Duration of Illness | No.of Patients. | No.of Recoveries. | % Recoveries |
|---|---|---|---|
| Over 2 years | 33 | Nil | Nil |
| Over 1 year | 9 | 2 | 22.2 |
| Over 6 months | 29 | 16 | 55.1 |
| Less than 6 months | 32 | 28 | 87.5 |

TABLE XXX showing the percentage recoveries when the duration of the illness is taken into consideration.

| Psychosis | No.of Patients Treated | No.of Recoveries. | % Recoveries. |
|---|---|---|---|
| Schizophrenia | 19 | 5 | 26.2 |
| Schizoid Depressives | 33 | 27 | 81.8 |
| Involutional Melancholia | 12 | 11 | 91.7 |
| Delusional Insanity | 3 | 1 | 33.3 |
| Manic Depressive Psychosis | 3 | 2 | 66.6 |
| | Total .. 70 | Total .. 46 | Average all groups 59.5% |

TABLE XXXI showing percentage recoveries when the type of psychosis is considered.

These figures indicate that the prognosis of true schizophrenia is only slightly improved by convulsion treatment being only slightly better than the average spontaneous

remission rate of 23.5% obtained from among untreated patients. The figures obtained in treatment of delusional insanity must be regarded as optimistic as they were obtained from so small a number of cases. In the group of schizoid depressives, and the involutional psychoses the prognosis appears to be very good and the results obtained compare very favourably with the best results of shock therapy whether cardiazol, triazol or insulin. The recovery rate among the involutional psychoses compare with those obtained by Bennett (1938) who reported relief in all of 10 depressed patients treated with cardiazol. The percentage of complete remissions in Bennett's cases was 80%. The results are also comparable with those obtained by Sogliani (1939) using the electrical convulsion method. Sogliani treated 100 patients, 73 schizophrenics of whom 15 (20%) recovered, and 27 manic-depressive patients of whom 22 (81%) recovered.

## RELAPSES

As with cardiazol and triazol relapses, it would appear, may occur with the electrical convulsion treatment although up to the present this has only occurred in one patient a female aged 26 years. This patient recovered after 6 major fits, but relapsed within two weeks. Treatment was then resumed and continued until 8 more major fits had been induced, by which time she was apparently normal. No further relapse has occurred and the patient has since taken her departure and resumed her employment. Patients undergoing "maintenance" treatment at fortnightly intervals also tend to relapse, as they did with cardiazol, if treatment is withheld for longer than that. This problem of indefinite maintenance presents no technical difficulties with the electrical method as it did with cardiazol and is unattended with adverse effects.

## THEORIES AS TO THE MECHANISM OF IMPROVEMENT

Many theories have been advanced in attempts to explain the mechanism by which the convulsion influences the course of the psychosis. Meduna (1937) to whose hypothesis reference has already been made expressed his opinion as follows "There is a biological antagonism between epilepsy and schizophrenia. If we succeeded in producing epileptic attacks in schizophrenic patients these attacks would alter the chemical, humoral and haematological milieu of the organism in such a way that a possibility of overcoming the illness might be created. because the milieu altered in this way would be unfavourable soil for schizophrenia." This theory, to my mind, does not bear close scrutiny, for in the literature dealing with the subject many references may be found to cases in which a combination of epilepsy and schizophrenia existed. These have been discussed by Nothkin (1929), Falsey (1935), Vorkastner (1918), Gruhle (1935), Harris (1938) and Esser (1938). With regard to the metabolism of carbohydrates, which Meduna stated was slowed in schizophrenia and accelerated in epilepsy, an analysis of the literature since 1916 shows that consistent results have not been obtained. Weston (1916), Wuth (1920), Bowman (1923) and others found that the fasting blood sugar of schizophrenic patients was within normal limits; while Kooy (1920), Uyematson and Soda (1921), and Labin (1927) obtained lower fasting blood

sugar levels than normal. Yet others, Smith and Hill (1927) and Bowman (1927), the latter in a second series of investigations, found a high fasting blood sugar level. In a series of 70 male and female schizophrenic subjects whose fasting blood sugar was investigated in this hospital during 1938 - 1939 it was found that the average resting sugar value was 78.3 mgm.%. This figure while on the low side is within normal limits. The literature also provides little to support Meduna's contention that the carbohydrate metabolism of epileptic subjects is accelerated. Wuth (1920), Weston (1921) and Mumford and Parkin (1923) found normal blood sugar levels during the course of their investigations. Lennox and Bellinger (1927) found that 70% of 140 epileptics had normal blood sugar curves, while 24% were high and 6% were low. Most damaging to Meduna's hypothesis, however, is the fact that other psychotic conditions are influenced by convulsion treatment and it would be fatuous to postulate an antagonism between epilepsy and these other psychoses.

Other theories are in the main psychological or physiological. Friedman (1937) believes that cardiazol is effective by breaking down the barriers to normal thought set up by the schizophrenic process. Gelhorn's (1938) theory that the convulsion produces anoxia of the brain which stimulates the sympathetico-adrenal system is not supported by the findings of Allen and his collaborators (1939) who

reported improvement in psychotic disorders after adrenalectomy. The theory of Jackson and Jackson (1938) that the convulsion produces its beneficial effect by dilating the brain capillaries and so oxygenating brain cells deficient in oxygen is partially supported by the work of Finnesinger and Cobb (1933) who showed that convulsions induced in animals by monobromide of camphor are preceded by dilation of the pial arteries. Gibbs (1933) however was unable to find any change in the cerebral blood flow of cats following convulsions induced by camphor, absinthe, picrotoxin and electrical stimulation. Ellery (1937) considers that the shock of the convulsion brings the patient so close to the outer world that he can be led back to reality by contact with the physician. Obendorf (1938) states his view as follows "It seems to me that psychological shocks and fear reactions may bring about an analagous interruption in the habitual or organic structural currents which have been established by thought habits. Such an interruption may produce a change which permits of re-organisation and readjustment of the thought flow thereby acting as a therapeutic agent." Among the theories advanced by Bennett (1938) was one suggesting that satisfaction of the "death instinct" brought about the remission in those affective psychoses marked by strong guilt feelings. Although Cohen (1939) and Cook (1940)have produced evidence showing that the results of convulsion treatment are better when no fear or apprehension is experienced by the patient

during the course of treatment, these theories are of interest for their similarity to the beliefs of psychiatrists of the early 19th century, during which, under the euphemistic title of "moral treatment" fear was much thought of as a therapeutic agent. Rush (1818) after describing the straight waistcoat, and the tranquilizer, and the use of hot and cold baths observes that if these modes of punishment fail to produce the desired effect it will be proper to resort to the "fear of death". As an example of this he describes the treatment of a troublesome patient who was placed in a bath of water and told to prepare for death. This injunction apparently induced a state of docile submission. As another instance of the influence of fear on the course of psychotic disorder, Rush cites the case of a patient whose psychosis remitted following the breaking of the rope to which he had been attached for the purpose of bathing him in a well. In this case Rush explained that the new action induced in the brain by the powerful stimulant of terror brought about the cure. Ellis (1838) observed that the object to be maintained with a view to ultimate cure was the removal of the cause by "moral treatment". Blandford (1896) commenting on the improvement seen in some psychotic subjects after treatment by the electrical method then in use expressed his opinion that the effect was moral rather than physical. With the modern method of electrical treatment all question of fear playing a part in effecting the cure is removed for one

of the most striking features of the method is the readiness with which all but the most deteriorated patients co-operate with the staff throughout the course of treatment.

None of the theories so far considered is supported by any real weight of evidence and convulsion treatment is still regarded as empirical. Recently, however, the claims of Bennett (1938), Menninger (1938), Youngs and Young (1939) and Wilson (1939) that better results are obtained in treating depressive states with cardiazol than schizophrenia suggests that this drug influences the affective centres. Indeed Pfister (1937) suggested that schizophrenia was a "system disease of the vegetative control mechanism" and expressed the opinion that cardiazol stimulated the vegetative nervous system. A similar observation was made by Bennett, in considering the mechanism by which cardiazol influenced the depressive psychoses, who suggested that the circulatory change resulting from the convulsion affected the emotional centres in the brain. Recent work suggests that these centres may be situated in the hypothalamus. Goltz (1892) who studied a decorticated dog over a period of 18 months found that stimulation of the animal produced all the outward signs of rage. Similar findings were obtained by Dusser de Barrene (1920) and Schaltenbrand and Cobb (1930) in experiments on cats, and in a dog by Rothman (1923). These reactions were designated "sham rage" by Cannon and Britton (1927) and were

shown by Bard (1934) to be dependent on the integrity of the hypothalamus for transection behind this structure, as in the Sherrington decerebrate animal, caused these reactions to disappear. Bard also demonstrated that normally the hypothalamus is under cortical control.

Clinico-pathological studies also suggest that the hypothalamus is closely linked with the emotions. Gagel (1936) drew attention to the fact that emotional changes were the most constant of the symptoms associated with tumours in the hypothalamic area. Foerster and Gagel (1933) reported that electrical stimulation of the hypothalamus in a patient whose brain was exposed at operation gave rise to restlessness, euphoria and other signs of mania. These workers are also of the opinion that the hypothalamus influences the cerebral cortex, its functions being stimulated from the anterior, and inhibited from the posterior portions of the hypothalamus respectively. Alpers (1937) reported personality changes in a man aged 39 years who had a teratoma in the region of the third ventricle which was invading the hypothalamus, and Cox (1937) found similar changes in a patient with a pituitary cyst indenting the hypothalamus. According to Fungeld and Kleist quoted by Grinker (1939) personality is a function derived from the activity of the diencephalon and Ranson and Ingram (1938) have postulated the existence of a centre of consciousness in the diencephalon. From this evidence there

appears to be little doubt that the hypothalamus participates in the complex expression of the emotions.

The experiments of Bard (1929) clearly indicate that normally the hypothalamus is under control of the cortex. There is also evidence as was suggested by Foerster and Gagel (1933) showing that the hypothalamus exerts an influence over the cortex. Grinker (1938) experimenting on cats showed that the hypothalamus gave rise to typical action potentials, and demonstrated that the hypothalamus could be stimulated by electric currents. Later Grinker and Serota (1938) showed that the method could be applied to man, and demonstrated that electrical stimulation of the hypothalamus was followed by the generalised visceral effects on blood pressure, respiration, pupils, heart rate, sweat glands, bladder and temperature seen in decorticated animals. Frequently too, the patients showed emotional disturbance, anxiety occurring quite commonly. In one patient uncontrollable sobbing resulted, while another felt his whole life pass before him. None of their patients showed the emotional concomitants of anger. With regard to the action potentials these workers showed that in both cat and man the typical cortical alpha wave appeared at about 10 waves per second and approximately 50 to 70 microvolts in amplitude. The hypothalmic waves appeared at the much slower rhythm of 4 to 5 per second. In amplitude these were only 75 per cent of the amplitude of the cortical waves. Superimposed on these were

small waves appearing at 13 per second. Cortical rhythm often permeated into the normal resting hypothalamus resulting in an alpha type of wave which was less regular and of lower amplitude than the normal alpha cortical wave. When the hypothalamus was destroyed to the point of extinction of the hypothalamic waves, the cortical alpha waves became clear and of the usual amplitude. Electrical stimulation of the hypothalamus evoked a massive discharge which resulted in the disappearance of the cortical alpha rhythm which normally entered into the resting hypothalamus. The cortical responses to hypothalamic stimulation were quite striking. Large rounded waves of 100 to 150 microvolts in amplitude made their appearance at the rate of one in 2 or 3 seconds. The normal cortical rhythm had disappeared. This result is similar to that obtained by Fleming, Golla and Walter (1939) in making electro-encephalographic records after the induction of a major epileptiform fit by electricity. These workers found that for some ten seconds after the fit the cortex showed little sign of electrical activity, after that, however, large slow waves made their appearance and these increased in amplitude until 30 seconds from the end of the fit they were several hundred microvolts. The frequency of these was 1 to 2 per second and their form was irregular. Grinker and Serota also showed that cardiazol evoked a short lived excitation of the hypothalamus.

In schizophrenia, according to Singer (1938) there is a

structural lesion of the autonomic system, and Gelhorn (1938) to whose theory reference has already been made believes that the cardiazol convulsion brings about a remission of the schizophrenic symptoms by stimulating the autonomic centres. Grinker (1939) expresses the opinion that in schizophrenia there is a deficiency of some central autonomic co-ordinating process which manifests itself as defective homeostasis. He also holds the belief that the difficulty which the schizophrenic subject experiences in relieving his tensions increases to a point where cortical inhibitions, conditioned by the demands for adaptation made by reality fail and hypothalamic activity forces itself into direct expression. This results in violent emotional outbreaks or, in terms of physiology, control of cortical activity by the hypothalamus. In other types of schizophrenia with cold blue extremities, and the other signs of what has been referred to as the endogenous form of schizophrenia, Grinker suggests that in these the driving power of the hypothalamus is reduced to a minimum where cortical activity is no longer maintained. In some of these types it has been found that the cortical action potentials are similar to those found in organic disease of the cortex.

Briefly then, there is evidence to show that the cortex and hypothalamus have reciprocal functions, and although the evidence in support of the belief that the hypothalamus is

implicated in the psychoses is of a speculative nature it appears reasonably certain that stimulation of this area produces restlessness and excitement while destructive lesions produce opposite effects.

Consideration of these observations of inter-relationship between the cortex and hypothalamus suggests that those exogenous psychoses described by Shepley and McGregor (1940) as inhibitory and excitatory have their origins in a disturbance of this inter-relationship. Depression of the hypothalamic affective centres brought about by cortical overactivity conditioned by unpleasant environmental events may result in the inhibited psychosis to which belong the involutional and schizoid depressives. On the other hand, release of the hypothalamus from cortical control may result in the hypermotility and excitement that characterises the excitatory psychosis. How this release is brought about is not clear. It may be, as suggested by Grinker (1939) that it is the result of failure of the cortical inhibitions, to meet the demands of adaptation made by reality thus allowing the hypothalamus direct expression. In bringing about an adjustment of these conditions the convulsion possibly stimulates the hypothalamus in the former thus freeing it from cortical activity, while in the latter as already suggested by Shepley and McGregor it furthers a release phenomenon already begun. Evidence supporting this view of hypothalamic stimulation has already been

considered in discussing the physiological concomitants of the fit when it was suggested that the increase in blood sugar and decrease in gastric acidity was the result of stimulation of the sympathetic nervous system, the centres for which the work of Camus and Roussy (1913), Bailey and Bremmer (1921), Himwich and Keller (1930), Miki (1932), Barris and Ingram (1936), Cleveland and Davis (1936) who induced an increase in blood sugar by lesions or excitation, suggests may be in the hypothalamus. Less definite is the evidence for hypothalamic stimulation afforded by the marked tendency of the patients to sleep in the post-accessual phase, although in the literature many references may be found showing an association between lesions of the diencephalon and sleep. Among those workers who have written on this subject may be mentioned Adler (1924), Hirsch (1924), Lhermitte and Tournay (1927), Fulton and Bailey (1939), Davidson and Selby (1935). The work of Hess (1932) is of considerable note in this respect for he was able to induce sleep by electrical stimulation of the hypothalamus. Harrison (1940) however, considers that sleep is only induced by electrical stimulation of the hypothalamus when that structure is damaged or destroyed.

The behaviour of the pupils, dilatation at the onset of the fit, may be the result of stimulation of the hypothalamus, for Karplus and Kreidl (1910) showed that reflex dilation of the pupil depended upon the integrity of the

hypothalamus, and not of the cerebral cortex. Nevertheless dilatation of the pupil can be produced by stimulation of the cortex, provided the hypothalamus and intrinsic nerves to the pupil are intact, and it is more likely that this is the mechanism of its production especially in the case of the electrically induced fit.

## THE FUTURE OF ELECTRIC CONVULSION TREATMENT.

During the past year the electric convulsion treatment has been demonstrated to a large number of psychiatrists and other practitioners of medicine. All have been favourably impressed and are agreed that the method will displace the pharmacological convulsants. The ready co-operation of the patient, the ease of application of the method and the almost complete absence of the disagreeable sequelae attending the use of cardiazol and triazol prompted Shepley and McGregor (1940) to forecast the use of the method in the out-patient department. This has already been practised in the case of one patient, not included in the discussion of results, who was suffering from profound depression and had been away from work for 5 months. This patient after two weeks treatment was able to return to his employment completely recovered. In this case 5 major convulsions had been induced. After each convulsion this patient recovered quite quickly and was able to go home unaided. In this connection it is of interest to note that a paper has just appeared by Strauss and McPhail (1940) in which a case is stated for the use of the electrical convulsion treatment in the out-patient department of a general hospital. The results obtained by these writers are regarded as encouraging.

Almost the only expense in connection with the method is the initial cost of the apparatus, for apart from infrequent renewal of the metal or wire portions of the electrodes, which can readily be carried out by the hospital electrician, no other outlay is necessary. The nursing staff in attendance require no special training for all that is required of them is for one to insert the gag when the mouth opens at the onset of the fit, and to s ee that the patient does not roll off the treatment couch. The nursing staff are not allowed to restrain the patient in any way during the fit for this it is felt conduces towards fractures. An ordinary bed-sheet, only, is laid over the patient during the fit. This offers no resistance to the free movements of the patient.

From a consideration of these facts it appears reasonable to predict that in general the electric convulsion method will supersede the use of massive doses of analeptic drugs, at least, for as long as convulsion treatment is practised.

## SUMMARY AND CONCLUSIONS

The technique of the electrical convulsion therapy of Cerletti and Bini has been described, and certain modifications adopted in this country, America, and Japan have been briefly considered.

The physiological effects on the blood sugar, the gastric juice and the blood pressure have been discussed. It has been shown that the application of the current is invariably accompanied by an increase in the blood sugar and a decrease in the gastric acidity. The effects on the blood pressure are less constant.

Despite the fact that electrical accidents may be attended by pathological changes in the skin, bones, eyes, heart and central nervous system no adverse effects have attended the application of the method.

During the past year 103 patients have undergone a course of treatment by the electrical method. The results obtained compare very favourably with those of other convulsion methods. The depressive states have been shown to have a very good prognosis under convulsion treatment, while it is doubtful if the prognosis of what has been called true schizophrenia is at all affected by treatment with convulsions.

The almost entire absence of complications and contra-indications to this form of convulsion therapy indicate that it

is a much more desirable method of carrying out this form of treatment than the pharmacological methods at present in wide use. Its ready applicability, and lack of need for specially trained nursing staff also suggest that it might replace the more complicated and time consuming hypoglycaemic treatment introduced by Sakel.

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I am indebted to Dr. T. P. Rees, Medical Superintendent of Warlingham Park Hospital, whose foresight was responsible for the early adoption of the electric convulsion treatment, and who has ever been ready with help and encouragement in this work. To Dr. W. H. Shepley, my senior colleague, I am indebted for assistance and permission to utilise his cases and for co-operation in carrying out the investigations. I am also indebted to Dr. A. C. West, principal of Croydon Polytechnic, for the loan of electrical apparatus, and to Mr. E. J. Bishop, laboratory technician at the hospital who assisted me in performing the laboratory tests; and also to both the female and male nursing staff of the hospital for their assistance and interest in my work.

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