Edinburgh University and Derbyshire House, Manchester, both have general practitioner teaching units attached to the university to enable students to study both disease and health of people in their normal environment. Cruickshank, at St. Mary's Hospital, London, instituted family visiting by senior medical students followed by discussions at which a paediatrician, psychiatrist, epidemiologist, almoner and nurse visitor were also present. General practitioners were not directly involved.

At Queen's College, Dundee, however, a project was set up under Alexander Mair, in which students followed up a hospital patient and studied the socio-psychological picture. Patients were mostly un-warned and the student had to make the approach. A twice weekly conference was held, with the patient's own general practitioner in a leading rôle. Mair himself was chairman, others present being a hospital almoner, health visitor, social worker and the students. A circular to the 87 doctors in the city brought forth a good response and the scheme was very popular with both student and general practitioner. Besides imparting an understanding of the social, diagnostic and therapeutic problems with which a general practitioner is so familiar, it gave an idea of the general practitioner's approach to problems as against the consultant's. In this case, one particular disease—rheumatoid arthritis—was followed up and much valuable research data was obtained. (Reid, J. J. A., 1958. *Practitioner*, 181, 65.

The basic professional education recommended to those concerned in undergraduate education by the Royal College of Physicians is an admirable objective. However, there are features of the course which can introduce the student to general practice. The subject of public health, along with the week's practical work is a great help. The thesis could be organised more along the lines of the Dundee experiment, although there are features of the present scheme which are satisfactory. For instance, the student would benefit by writing the results of his investigation in thesis form as this experience will prove valuable later. It is the study of the socio-psychological background of patients in their own environment which is so useful.

The Virginian scheme, apart from being un-practical in this country, does not force the student to make the approach and inquiry himself.

Out-patient and casualty training are valuable especially if only one or two students are present and they can take an active part in the proceedings. Varied vacation work gives an understanding of different classes of people and a tolerance of different philosophies. Medical jurisprudence points out a doctor's behaviour, responsibilities, and safeguards in modern society.

The general practitioner week shows the student for the first time just what kind of life general practice really entails.

The lectures in sixth year when given by speakers with experience and ability to pin-point the important factors, are most interesting and instructive, especially when illustrated by actual patients or case histories.

In summing up I may say it is my opinion that it would be wrong to emphasise general practice too much before graduation. By all means make the student aware of the special approach and problems of the general practitioner and give him an idea of the type of life it offers, but the primary object of undergraduate medical education, as laid down by the College of Physicians should be the aim of medical education in this country. After graduation is the time for more intensive general practitioner training as pointed out by Craddock.

Useful hospital work, overseas experience and travel, assistance and guidance by the College of Physicians, medical journals, etc., and apprenticeship and advice from experienced general practitioners are all important. Lastly, but most important of all, in the training of a general practitioner is experience of general practice itself.

If necessity is the mother of invention, it must surely be also the father of wisdom.

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Cerebral Lesions from Electric Shock Treatment

By I. M. ALLEN (*Wellington*)

It was shown in an earlier paper (1951) that the use in electric shock treatment of currents that were light and of short duration did not prevent the production of changes in the brain due to passage of the current. Observations included those made in experimental work on animals; pathological examination of brains of patients who had died after treatment from conditions unrelated to it, those precipitated by it or the treatment itself; and clinical examination of patients who had been treated. Illustrative cases were reported, and it was shown that enough damage was produced in the brain to give rise to neurological variations; the damage might be reversible and these variations disappear in some cases; and it might persist and the variations continue in other cases.

Now, the apparent freeing and even removal of limitations on the use of the method, the much wider use of it even for conditions for which it was formerly thought to be unjustified, the accumulation of observations on the effects of it on the brain, and the increasing frequency with which patients appear with neurological variations after it make it necessary to review these observations and re-examine the relation of electric shock treatment to lesions of the brain.

This study is based, therefore, on observations summarised in the earlier paper, others reported since then and those made during clinical study of 18 patients after electric shock treatment.

Experimental Observations

Experimental work on the brains of animals showed that changes are produced in the ganglion cells of the brain almost immediately, develop for a time and then subside; haemorrhages occur into the substance of the brain, often in a particular part but also in a lesser degree elsewhere; there is destruction of nerve cells in and about the areas affected by the haemorrhages; and organisation of these parts may be accompanied by further changes in nerve cells which are irreversible.

These observations have been confirmed. Ferraro and Roizin (1949) submitted 11 monkeys to daily convulsions, which varied in number from 32 to 100 in individual cases; and found in every case neurocellular changes, which they thought might be reversible. Mancini (1950), on the other hand, exposed 12 rabbits, with two controls, to one to ten consecutive shocks, killed the animals either immediately or 48 hours after the series, and found swelling of cell bodies which was sometimes acute near blood vessels, but changes in microglial cells only after many shocks.

Pathological Observations

Pathological examination of brains of patients who had died in various ways after electric shock treatment showed that these changes also appear in the human subject; and demonstrated not only immediate changes in the nerve cells and petechial haemorrhages into the substance of the brain produced by the passage of the current but also irreversible changes in the nerve cells produced both directly and as a result of the vascular changes and haemorrhages.

These observations have been confirmed and others added to them. Marchand and Masson (1947) found recent capillary haemorrhages into the brain and meninges of a patient who died immediately after shock treatment. Riese (1948) reported increased vascularisation and oedema of the brain on death 48 hours after the second shock treatment; and vascular dilatation and stasis in the brain, pericapillary haemorrhages, small areas of necrosis and degeneration of nerve cells on death 20 minutes after the second shock treatment. Sprague and Taylor (1948) found a haemorrhagic area in the brain of a patient who died two weeks after a series of six treatments; and Schulte and Dreyer (1950) extensive petechial haemorrhages in the brain of one in whom deep coma followed the fourth treatment. Nielsen (1950) described in the brain of a patient who failed to recover consciousness after the fourth treatment and developed hyperthermia and convulsions "localised areas of subcortical necrosis, and early neural degeneration and chromatolysis of the subcortical region of the cerebral hemispheres and basal ganglia". Scheidegger (1950) reported several forms of degeneration of brain tissue observed after electric shock treatment in schizophrenics; and multiple small areas of softening in the cerebral cortex of a man 61 years old who had died from electric shock treatment. Halpern, Rozanski and Libau (1952) found a haemorrhage occupying the whole of the left frontal lobe and thrombosis of the superior longitudinal sinus in a patient 45 years old who was not hypertensive, developed hemiplegia after the fourth shock treatment and died 48 hours later. Corsellis and Meyer (1954) demonstrated in the brains of two patients—one of whom had 140 shock treatments and died after the administration of curare, and the other had 45 shock treatments and died in the course of treatment—marginal gliosis in the cerebral cortex of the hemispheres and a moderate degree of diffuse or perivascular proliferation of

astrocytes in the white matter. Madow (1956) described a massive intraventricular haemorrhage in one patient and petechial haemorrhages especially in the grey matter around the aqueduct of Sylvius and in the brainstem of three others who had died from electric shock treatment.

Electroencephalographic Observations

Alema, Brizzi and Sinisi (1951) defined the changes in the electroencephalogram after electric shock treatment, and Maleci and Schergna (1951) described slow delta waves in the tracing. Chusid and Pacella (1952) reviewed the literature and concluded that the chief change was a slowing of rhythm, generally over the anterior parts of the brain, and the degree of abnormality was in proportion to the number and frequency of treatments given. Simon, Yeager and Bowman (1953) reported the variations which developed in 50 patients during treatment by electronarcosis. The electroencephalogram had been normal in 28 patients and mildly slow in 22 patients before the treatment. Dysrhythmic variations appeared as the treatment continued, then high amplitude slow waves on hyperventilation and finally slow waves under ordinary conditions.

The significance of these variations in relation to the subject under consideration is that they were those commonly found with physical changes in the cerebral cortex. So, it is not surprising that Kohler, Meyer and Blendinger (1954) found in the electroencephalogram and pneumoencephalogram evidence of diffuse cerebral lesions which could precipitate spontaneous epileptic symptoms in the individual with a latent trend.

Clinical Observations

Clinical examination of patients after electric shock treatment showed that it had produced neurological variations, some of which were temporary and some permanent. Further observations reported in the literature have confirmed this and added some new features.

Stieper, Williams and Duncan (1951) made a special study with controls of amnesia in 12 paranoid schizophrenics who had had electric shock treatment, found that personal memories were affected more than impersonal, and showed that amnesia for remote personal memories was greater in patients who had failed to benefit. Juba (1948) described disturbances of cortical function, indicated by visual or spatial agnosia or by Gerstmann's syndrome, after electric shock treatment; and, in five patients who had had series of from two to five treatments only, features of focal disturbances in the cerebral cortex of the parieto-occipital region in the form of visual receptive aphasia, tactile agnosia, disorientation for right and left, disturbance of constructive drawing and associated expressive aphasia. Gareb and Vargha (1953) reported a similar effect in a patient who developed acalculia after shock treatment and had to be re-educated by the development of conditioned responses.

Kaldeck (1948) had already described hemiplegia of several days duration in a physically normal young woman, and referred to a similar case in which there was hemiplegia for 20 minutes in an elderly patient, in each case beginning immediately after electric shock treatment. Poloni (1949) reported a similar effect in two patients with some arteriosclerosis; in the first right hemiplegia for half an hour after each treatment; and in the second right hemiplegia which developed one day after the fourth treatment and persisted for 24 hours. Finally, Rostan (1952) described a patient who developed euphoria, difficulty in standing erect, dysarthria and pyramidal signs after electric shock treatment and recovered completely.

In confirmation of the warning by Kohler, Meyer and Blendinger (1954) that diffuse cerebral lesions demonstrated in the electroencephalogram and pneumoencephalogram could precipitate spontaneous epileptic symptoms in the individual with a latent trend, there have been reports of cases in which that actually occurred. Fattovich (1948), who had already published three similar cases, described the case of a patient who developed and continued to have spontaneous fits after a series of 25 electric shock treatments. Delgado (1951) reported the development of spontaneous epileptic fits in 12 of 794 patients who had had electric shock treatment and shown some predisposition to such symptoms. Ruggeri and Somazzi (1955), on the other hand, described the development of spontaneous epileptic fits in 13 cases of schizophrenia in which there had been no clinical indications before the treatment; and Blumenthal (1955) that of spontaneous epilepsy in 12 patients who had not had epilepsy before and in four of which the electroencephalogram had been normal. The patients in the last series had been given an unusually large number of shock treatments, from 11 to 192 with an average of 71. With changes demonstrated in the brain in various ways after electric shock treatment, it was evident that it was the permanent damage to the brain, as with that due to other causes, and not the history of having had artificially induced convulsions which was responsible for the appearance of spontaneous epileptic symptoms after the treatment in these cases.

Personal Observations

Personal observations were made on 18 cases in which electric shock treatment had been used and there was evidence of lesions of the brain. This series represented only a small proportion of the cases in which this form of treatment must have been used and probably of those in which there may have been lesions of the brain.

The patient had been examined at various times before the treatment in eight cases and found to have no evidence of lesions of the brain; and it was evident from the history of the patient and from examination by others that there had been no previous physical condition of the brain in the other 10 cases. The patient was examined in detail personally on one or more occasions after the treatment in all 18 cases.

The time that had elapsed from the end of the treatment to the first observations on the patient varied considerably, from six weeks at one extreme to 10 years at the other. It was three months or less in seven cases; between three and six months in three cases; between six months and one year in two cases; between one and two years in three cases; and over two years in three cases. It was long enough in 11 cases for the effects of any reversible changes in the brain to have disappeared, and at the later examinations in the remaining seven cases for these effects to have subsided.

The original condition for which shock treatment had been used, though not advised in the cases seen shortly before it, also varied considerably. It was autonomous depression in six cases, in five of which there had been previous phases from which the patient had recovered spontaneously within a reasonable time, and in one there had been no previous phase and the duration of tht condition before the treatment had been very short. It was a chronic anxiety state in which various though not severe phases of depression had supervened from time to time in seven cases, and a more clearly defined phase of anxiety depression in one case. It was an anxiety state with obsessive and compulsive features in one case; and a pattern of one or more phases of depression which were not severe and covered a schizophrenic trend in four cases.

The number of treatments given had been small in comparison with that in cases reported by Blumenthal (1955) and others. It had been less than five in two cases; between five and ten in four cases; and an average of eight in seven cases. It was ten or more in three cases, in two of which it had been fifteen. There had been two courses of treatment in two cases: in the first two series of twelve each; and in the second a first series of eight and a second series of five.

The clinical effects in relation to the central nervous system were both recent and late. As, however, no patient was examined within six weeks of the end of the treatment, this summary of them is concerned chiefly with the latter.

What appeared to be more general effects which had been apparent from the time of the treatment were still present in five cases and sometimes severe. They included defect of memory for events of six months before the treatment, which was being restored gradually by building up associations; slowness in understanding and carrying out instructions; failure in tests involving attention and recent memory; occasional expressive aphasia; and, in a more severe case, ill-sustained attention, severe defect of memory for recent events and for those of a long time before the treatment, and disturbance of time relations in regard to the latter.

There was a grasp response with or without tonic innervation on the right side in sixteen cases, and on the left side in five cases. Some features of involvement of upper motor paths were found on the right side in four cases, and on the left side in four cases. These variations were often present on one side only even in cases with more general effects, but were found on both sides in some cases. They indicated the presence of lesions especially of the anterior part of the cerebral hemispheres.

The results of treatment of the condition for which it had been given were often unsatisfactory. The acute condition had subsided but lesions of the brain were left in two cases. Depression had subsided, as it had done before spontaneously, but a schizophrenic pattern remained in one case. The more florid symptoms were less but the basic pattern persisted with the patient inadequate in all respects, even in cases of autonomous depression in which there had been spontaneous recovery from previous phases, in 10 cases. This result was also noted in patients with autonomous depression who had been so treated and had no unequivocal physical signs of lesions of the brain. The condition of the patient was unchanged in three cases; and aggravated in three cases. In one of the latter, depression which had formerly been intermittent and in relatively short phases became constant and continued for two years; in one there was deterioration with lesions of the brain and a schizophrenic state; and in one there was a severe degree of neuronic degeneration affecting the cerebral cortex and deeper parts of the brain, apparently progressive and followed by the death of the patient.

The last case, Case 15 of this series, showed many of the effects that are summarised above.

Case Report

The patient, female, age 53 years, was referred with a history of symptoms for eighteen months. From being bright, active and sociable, she rapidly lost interest, became languid and jittery, and felt that she could not do either her office or her domestic work. Her symptoms from that time were: poor memory and attention; worry attached to everything; gloom with at times more definite depression, desire to weep and sometimes weeping with relief; anxiety attached to being unable to do her work; a heaviness of the head with aching and stabs of pain in the back of it; an occasional feeling that she was going to fall; lack of desire for company; trembling of the limbs; lack of energy and slowness of the bowel.

Examination showed the following: a well-defined pyknic type; dryness of the hair: tremor of the eyelids on gentle closing; lagging of the upper eyelids behind the eyeballs on downward deviation of the latter; restless and uncertain movements of the eyeballs; in the retinae, distension of a vein distal to the crossing by an artery, angulation of a vein before and after a crossing, and kinking of a vein at a crossing with systole of the artery; stiffness and limitation of voluntary and emotional movements of the face with a tonic tremor; tremor of the extended fingers; tension of the brachial arteries; a pulse rate of 100; a blood pressure of 180 mm. systolic and 95 mm. diastolic; alternation of contraction and relaxation in movements against resistance at the knees and ankles; active knee jerks; the plantar reflexes only slightly in flexion; and other findings in detail in relation to the central nervous system normal.

The patient was then in a phase of autonomous depression of only moderate degree accompanied by slowing of functions, some conversion symptoms and effects, and occasional anxiety, and had no evidence of structural change in the brain. Treatment by other than conservative measures did not appear to be indicated.

She was referred again three years later, when her history during the interval, as given by her husband, was as follows . She remained in her former state for nine months, then recovered and was able to travel for some months. She developed symptoms again some time after she returned home, and was treated later away from her own town with a series of fifteen electric shock treatments. Her husband was startled at the deterioration in her after the treatment and said that, during the eighteen months since then, she had been worse than she had been before, done no more than potter about the house and garden, and needed constant help in the home. She got lost in her own house, needed help with dressing and undressing, and had no idea of what time or what day it was. The patient herself said that she had no confidence; did little; could not concentrate or remember; could not retain what she heard; worried about her inability to do things and got depressed; could not speak properly, often used wrong words, got muddled and forgot what she was talking about; could not dress or undress herself; could not write; walked slowly, had to look at the ground as she did so and sometimes fell; and had to support herself with her hands when rising from a chair.

Examination showed the following. When invited to sit down in a chair which was on her left, she moved as if to sit down where there was no chair. She rose from the sitting position by supporting herself with her hands on the arms of the chair. She then stood still with some flexion of the neck, trunk and thighs, and flexion of the left arm more than of the right. She walked slowly, did not swing the arms and took short steps without lifting the feet from the floor. Told to turn and walk back the way she had come, she turned to the right through about 45 degrees and remained standing in that position. She began and carried out slowly movements of the neck. On single passive movements there was slight increase of resistance in muscles of the neck; and on repeated passive or active movements this resistance increased steadily to rigidity which subsided only on cessation of the movements. The palpebral apertures were wide with at times sudden raising of the upper eyelids. She kept her gaze fixed on what was before her without blinking, and could not detach it and look in any other direction at command. When, however, a plane surface was placed before her eyes within the focal distance, she closed the eyelids or she was placed in a dark room, she could move her eyeballs and look in any direction at command provided that she did not have the opportunity of fixing gaze on an object before she tried to do so. She recognised a moving object in the right or the left temporal fields but failed to recognise either of them when both were moved at the same time. On being asked to point to a moving object she recognised in the left temporal field, she pointed straight ahead or to her right. Voluntary movements of the face were stiffer and more limited than emotional movements. She responded, during examination of sensory functions on the face, to only the first of a series of stimuli. She produced word sounds in the same tone, sometimes said only two or three words and then stopped. Other findings in the cranial nerve territory were normal.

Told to extend the upper limbs palms upward before her, she presented the left upper limb partly flexed at the elbow, the right fully extended and both deviated to the right of the middle line. Both of them then strayed steadily to the right and, on being replaced, did so again. There appeared at times hesitant jerks of the upper limbs in the line of gravity. Resistance to passive

movements was increased in all directions at both shoulders, and in the flexors of the left elbow. Told to touch the nose with the right or left index, she failed to do so; but on either the right or the left hand being touched, she did so with that one, though inaccurately with the left. The tendon reflexes of the upper limbs were abrupt and active. A grasp response was elicited in the right thumb and index. She responded to a single stimulus, touch of cotton wool or prick of a pin, on either the right or the left upper limb alone; but only to that on the right when stimuli were applied to both at the same time. Asked to indicate the site of stimulation, she said and indicated: arm for right hand; breast for right arm; left arm for left breast; and hand for left arm. Other findings in the upper limbs were normal. The pulse rate was 80; and the blood pressure was 160 mm. systolic and 100 mm. diastolic.

In the lower limbs, there was increase of resistance to single passive movements in all directions at both hips; tonic innervation was elicited at both ankles; there was a grasp response in the toes of both feet; and the tendon reflexes were active. She recognised a single touch or pin prick on either the right or the left lower limb alone; but only that on the right when it was applied to both at the same time. She localised the stimulus more proximally on the right side and more distally on the left side, saying and indicating: leg for right foot; thigh for right leg; leg for left thigh; and foot for left leg. Other findings in the lower limbs were normal.

Given a pair of gloves, one on each knee, and asked to put them on, she put on the right glove helping it in an uncertain way with the left hand, stopped and failed to continue. Though looking towards the left glove on her left knee, she seemed not to register its presence. When it was touched and she was asked to put it on, she moved the left hand aimlessly until it came into contact with the glove, picked it up with the left hand, handled it with both hands, failed to put it on and asked whether she should take off the right glove first.

She died five months later at a considerable distance, and no autopsy was obtained.

In this case, there had been at the time of her first attendance a depressive phase of moderate degree for eighteen months; there was no evidence of structural change in the brain; the condition subsided nine months later; and it recurred after an interval. There followed eighteen months before her second attendance a series of fifteen electric shock treatments, after which her condition deteriorated considerably. On examination the following variations were found: occasional expressive aphasia; exaggeration of fixation of gaze; tonic innervation and grasp responses on both sides; unilateral sensory inattention to the left for tactile and painful stimuli; lack of recognition of her left side and probably of space to her left; a gross shift of localisation of stimuli proximally and then towards the middle line on the right side, and away from the middle line and distally on the left side; and muscular rigidity of the extrapyramidal type in character and distribution. They indicated the presence of lesions throughout the cerebral cortex on both sides and in the brain down to the level of the substantia nigra. These followed a long course of electric shock treatment, appeared in a patient who had no evidence of structural change in the brain before, may have been progressive, and were followed by the death of the patient.

Discussion

The observations recorded in this paper left no doubt about the conclusions to be drawn from them and call for little comment. Their clinical implications demand, however, the most careful consideration.

They confirmed the appearance of irreversible physical changes in the brain after and as a result of electric shock treatment. They gave no indication of the frequency with which these changes occurred; but suggested that physical changes, some of which might be irreversible, were inseparable from such treatment. They supported the conclusion that the changes might

be more severe and more liable to be irreversible with increase in the number and frequency of treatments, but showed at the same time that they could appear and be irreversible after very few treatments.

The observations on changes in the brain of animals after electric shock treatment added little to those summarised in the previous paper (1951). Those made on the pathological and histological examination of brains of patients who had died from various causes during and after the treatment confirmed the occurrence of changes, some of which might have been reversible but many of which were irreversible, in the brain after electric shock treatment; and directed attention to other complications, usually vascular in origin, which might appear. The changes reported in the electroencephalogram during and after treatment were those usually found with physical changes in the cerebral cortex and supported the conclusion that changes were produced in the cerebral cortex by passage of the current. The various physical signs and disturbances of function following electric shock treatment, reported in the literature and found in the cases on which this paper is based, left no doubt that definite neurological variations, which could be found on detailed examination and many of which were irreversible, were left after electric shock treatment. They also showed that damage to the brain was not necessarily limited to the anterior part of the cerebral hemispheres; it might involve the posterior part of the hemispheres and produce disturbances of function which were not so readily appreciated during the few weeks after the treatment ended; it might be widespread throughout the cerebral cortex and produce specific variations such as those included in Gerstmann's syndrome with or without physical signs of involvement of the fronto-parietal areas; and it might affect grey matter at a level deeper than that of the cerebral cortex. The presence of these neurological variations was associated with relative lack of success in the treatment of the condition for which the method was used, even when it was one which had subsided spontaneously on former occasions. It could, as shown in some reports, have provided an additional factor, as in the case of lesions of the brain from any cause, leading to the appearance of spontaneous epileptic symptoms in an individual with a latent trend and formerly free of them. The physical changes in the brain appeared, though it could not be finally proven, to have become progressive in Case 15.

Summary and Conclusions

Observations made in the course of experimental study, and in pathological examination of brains, electroencephalograms of patients and clinical examination of patients after electric shock treatment are reviewed.

These observations showed that damage to the brain, sometimes reversible but often irreversible, occurred in the course of electric shock treatment.

Observations on eighteen patients who had evidence of lesions of the brain after electric shock treatment are reported.

The frequency of damage to the brain from electric shock treatment is not known; but it is probable that some damage, which may be reversible but is often irreversible, is inseparable from this form of treatment.

The clinical implications of the association of irreversible changes in the brain with electric shock treatment demand more serious consideration.

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Muscle Spasms due to Perphenazine (*Trilafon*)

By O. L. DINE AND P. FLEISCHL (*Napier*)

Perphenazine (Trilafon-Schering) is a new phenothiazine derivative which would appear to have some advantages over chlorpromazine (Largactil) in that jaundice, severe hypotension and toxic effects on bone marrow have not yet been reported. Used in comparable dosage to chlorpromazine, the side effects of perphenazine on motor tone, producing extra-pyramidal rigidity, occur more frequently, as also involuntary muscle spasms which are encountered only very rarely with chlorpromazine. These effects may be alarming to patient and physician alike and their importance lies in early recognition.

Case Report

M. D. a 24-year-old married woman presented in the fourth week of her first pregnancy with hyperemesis gravidarum. All food ingested was returned and nausea was continuous. Dimenhydrinate (Dramamine) 50 mgm. twice daily was administered for one week without any beneficial effect. After a lapse of a further two days perphenazine was commenced. An intramuscular injection of 5 mgm. was given in the morning followed by one 4 mgm. tablet taken at noon, at night and in the morning of the following day. No vomiting occurred during treatment and relief of the nausea was complete.

About noon of the second day after a total dose of 17 mgm. had been given the patient complained of an increasing tightness in the jaw muscles which was at first relieved by opening the mouth widely but later progressed to complete trismus. The spasm was accompanied by pain in the masseter muscles. Swallowing was not affected and control of the tongue was normal. There were no other abnormal features. Intermittent spasm of the muscles of mastication was observed particularly on the left side. The patient was alert and apprehensive throughout the whole episode which lasted for six hours. Recovery occurred soon after the administration of 3 gr. of sodium amytal.