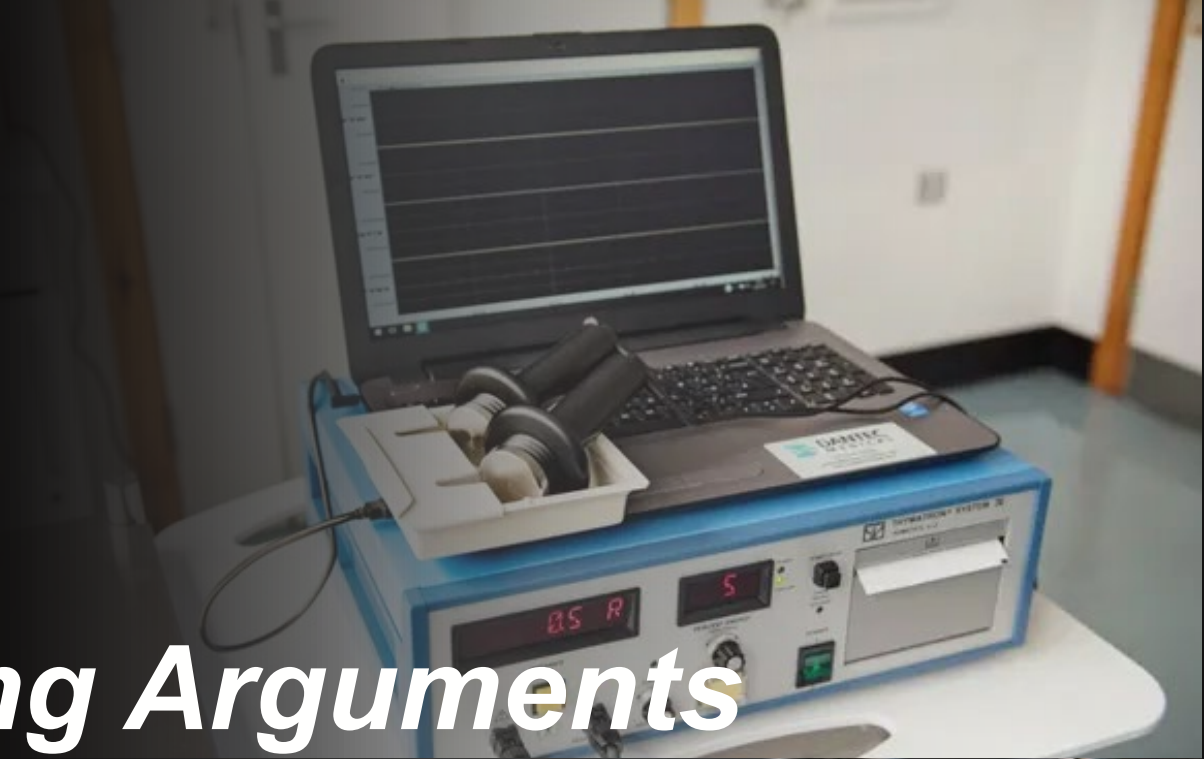


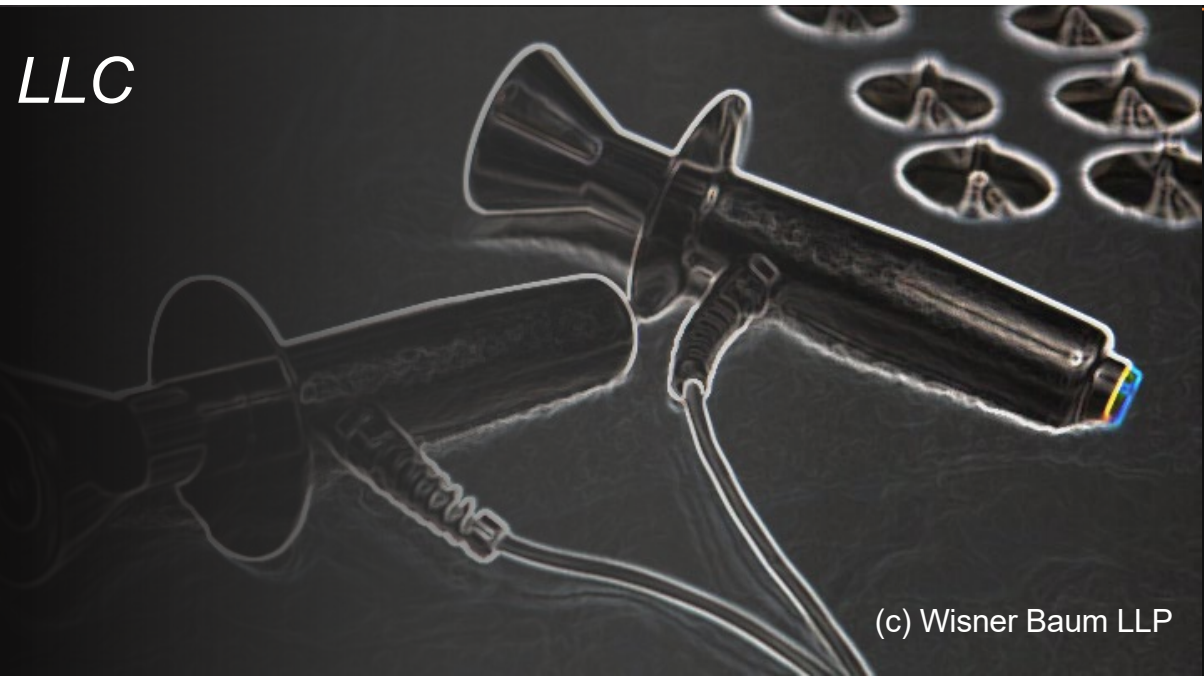
Plaintiff Closing Arguments

Thelen, Jeffery v. Somatics, LLC

June 8, 2023

Department 14A

Hon. Tom P. Barber

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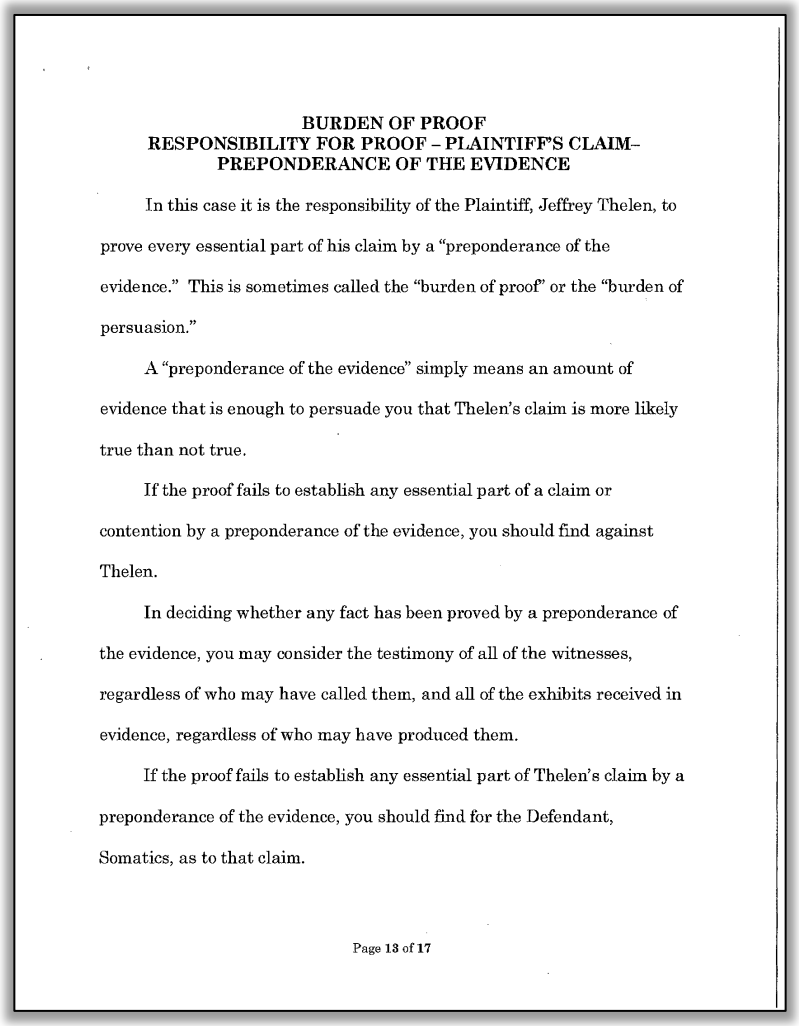

BURDEN OF PROOF
RESPONSIBILITY FOR PROOF – PLAINTIFF'S CLAIM–
PREPONDERANCE OF THE EVIDENCE

In this case it is the responsibility of the Plaintiff, Jeffrey Thelen, to prove every essential part of his claim by a "preponderance of the evidence." This is sometimes called the "burden of proof" or the "burden of persuasion."

A "preponderance of the evidence" simply means an amount of evidence that is enough to persuade you that Thelen's claim is more likely true than not true.

If the proof fails to establish any essential part of a claim or contention by a preponderance of the evidence, you should find against Thelen.

In deciding whether any fact has been proved by a preponderance of the evidence, you may consider the testimony of all of the witnesses, regardless of who may have called them, and all of the exhibits received in evidence, regardless of who may have produced them.

If the proof fails to establish any essential part of Thelen's claim by a preponderance of the evidence, you should find for the Defendant, Somatics, as to that claim.

Page 13 of 17

COURT'S INSTRUCTIONS TO THE JURY, Pg 13

In this case it is the responsibility of the Plaintiff, Jeffrey Thelen, to prove every essential part of his claim by a "preponderance of the evidence." This is sometimes called the "burden of proof" or the "burden of persuasion."

A "preponderance of the evidence" simply means an amount of evidence that is enough to persuade you that Thelen's claim is more likely true than not true.

If the proof fails to establish any essential part of a claim or contention by a preponderance of the evidence, you should find against Thelen.

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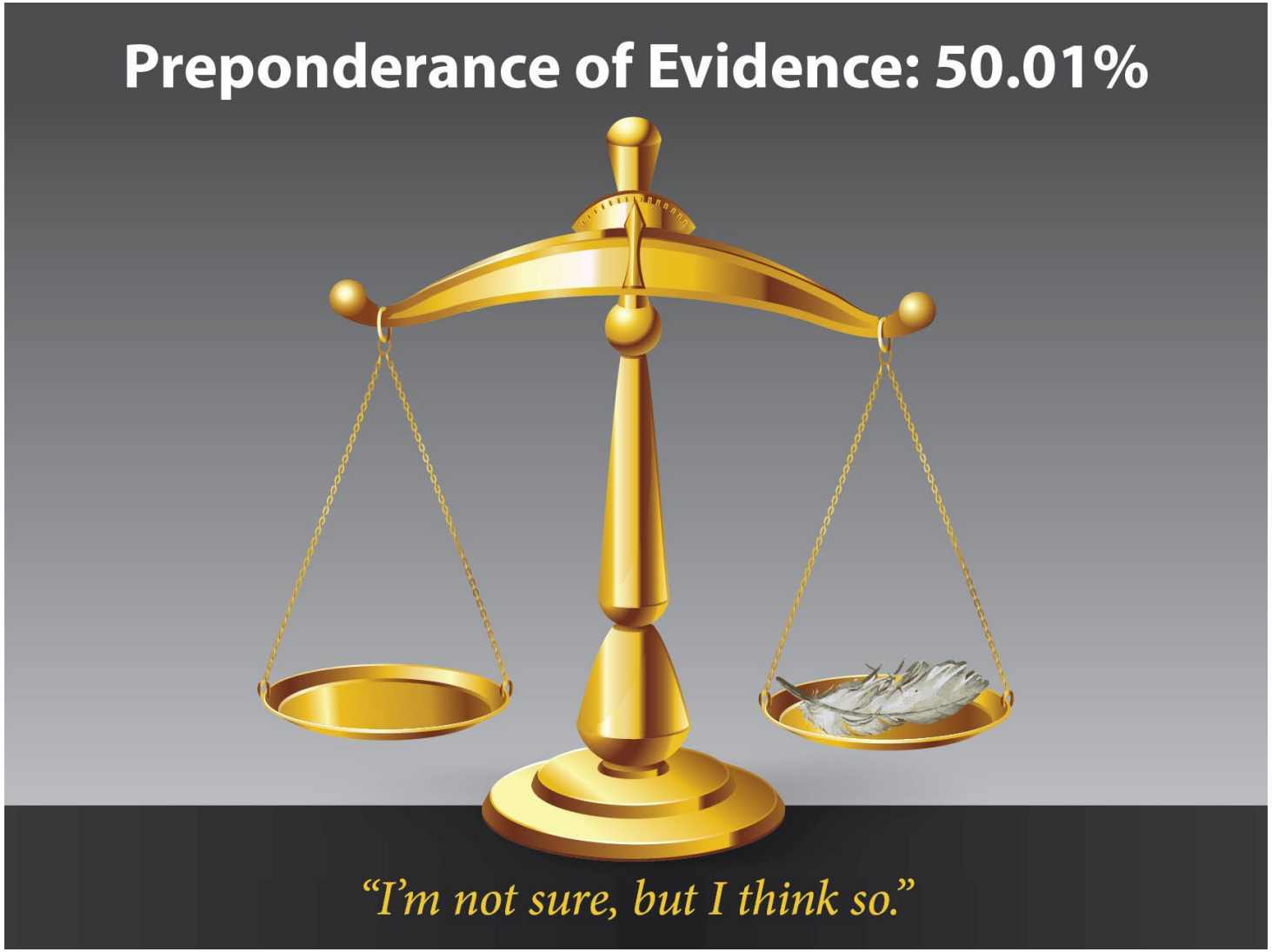
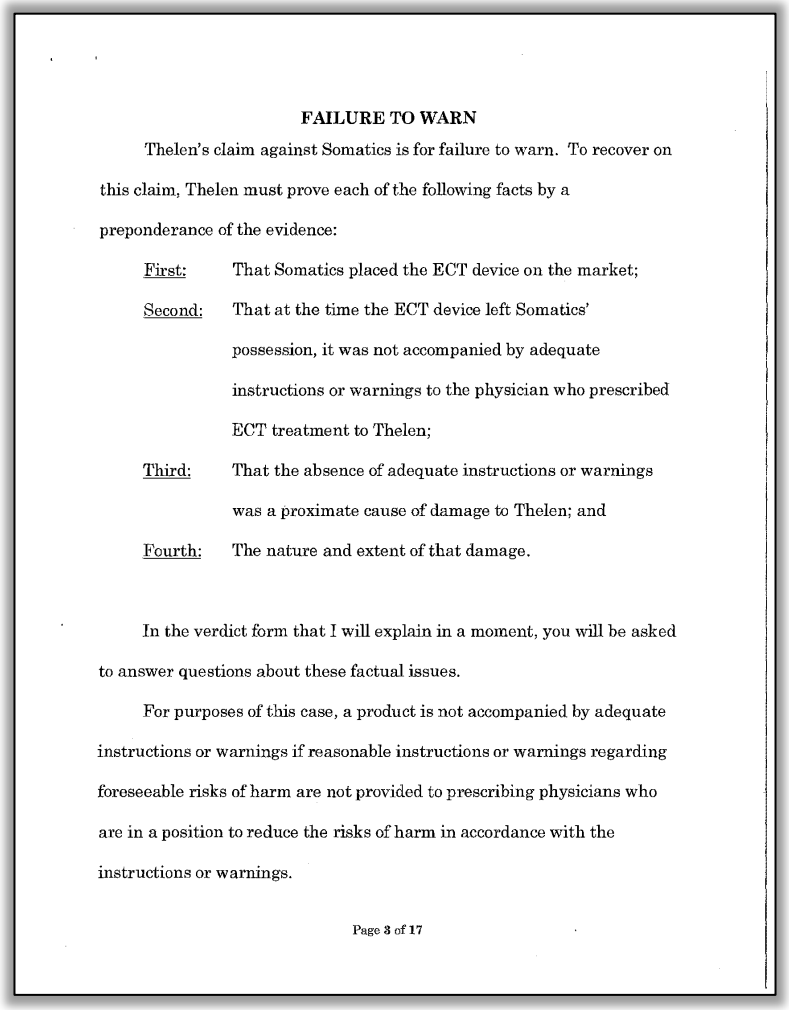

Preponderance of Evidence: 50.01%
"I'm not sure, but I think so."

Failure to Warn



FAILURE TO WARN

Thelen's claim against Somatics is for failure to warn. To recover on this claim, Thelen must prove each of the following facts by a preponderance of the evidence:

First: That Somatics placed the ECT device on the market;

Second: That at the time the ECT device left Somatics' possession, it was not accompanied by adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen;

Third: That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen; and

Fourth: The nature and extent of that damage.

In the verdict form that I will explain in a moment, you will be asked to answer questions about these factual issues.

For purposes of this case, a product is not accompanied by adequate instructions or warnings if reasonable instructions or warnings regarding foreseeable risks of harm are not provided to prescribing physicians who are in a position to reduce the risks of harm in accordance with the instructions or warnings.

Page 3 of 17

COURT'S INSTRUCTIONS TO THE JURY, Pg 3

FAILURE TO WARN

Thelen's claim against Somatics is for failure to warn. To recover on this claim, Thelen must prove each of the following facts by a preponderance of the evidence:

First: That Somatics placed the ECT device on the market;

Second: That at the time the ECT device left Somatics' possession, it was not accompanied by adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen;

Third: That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen; and

FAILURE TO WARN

Thelen's claim against Somatics is for failure to warn. To recover on this claim, Thelen must prove each of the following facts by a preponderance of the evidence:

First: That Somatics placed the ECT device on the market;

Second: That at the time the ECT device left Somatics' possession, it was not accompanied by adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen;

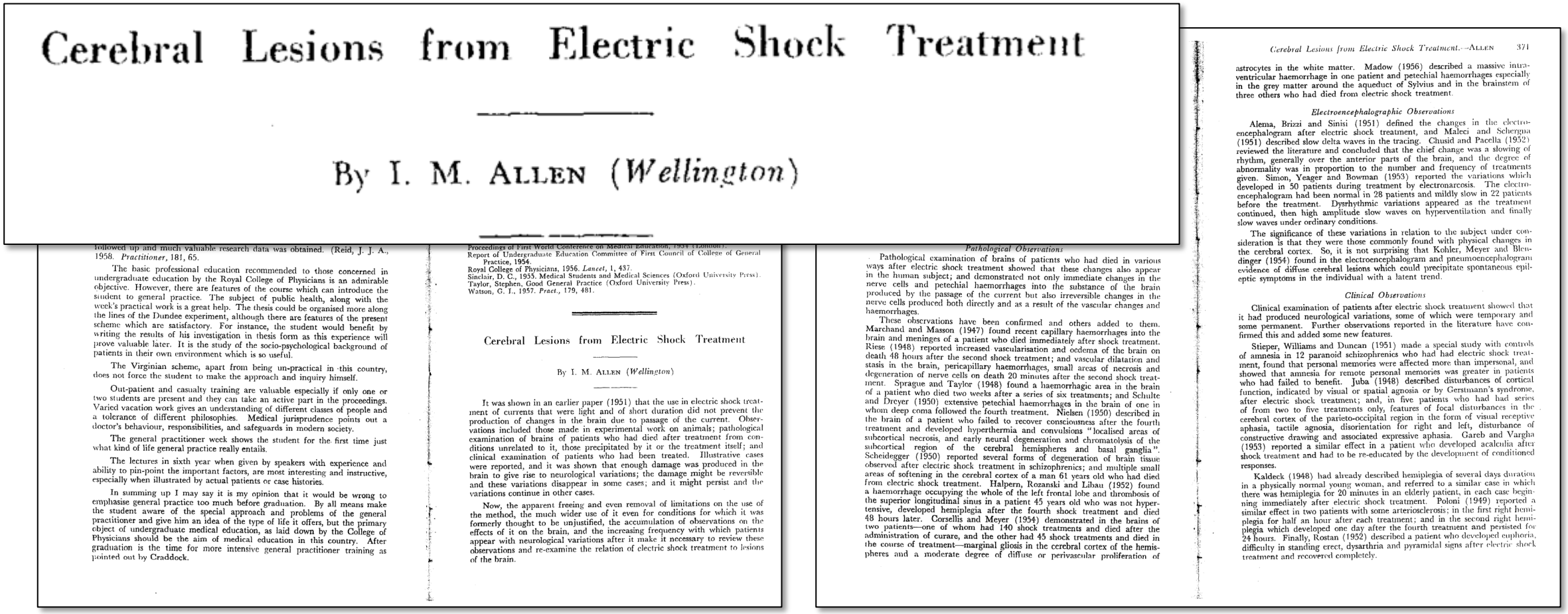
Third: That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen; and

Fourth: The nature and extent of that damage.

Stipulation

“It is here by stipulated and agreed by and between plaintiff Jeffrey Thelen, by and through his attorneys and defendant Somatics LLC by and through its attorneys that Plaintiff's Exhibit Number 1, the 2013 Thymatron System IV instruction manual does not contain the words, "brain damage." This should be taken as established by a preponderance of the evidence and no evidence regarding this fact need be presented by plaintiff at trial or any further proceedings.”

Failure to Warn



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.

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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.

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.

(1951) Allen Study

Thelen v. Somatics, LLC

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.

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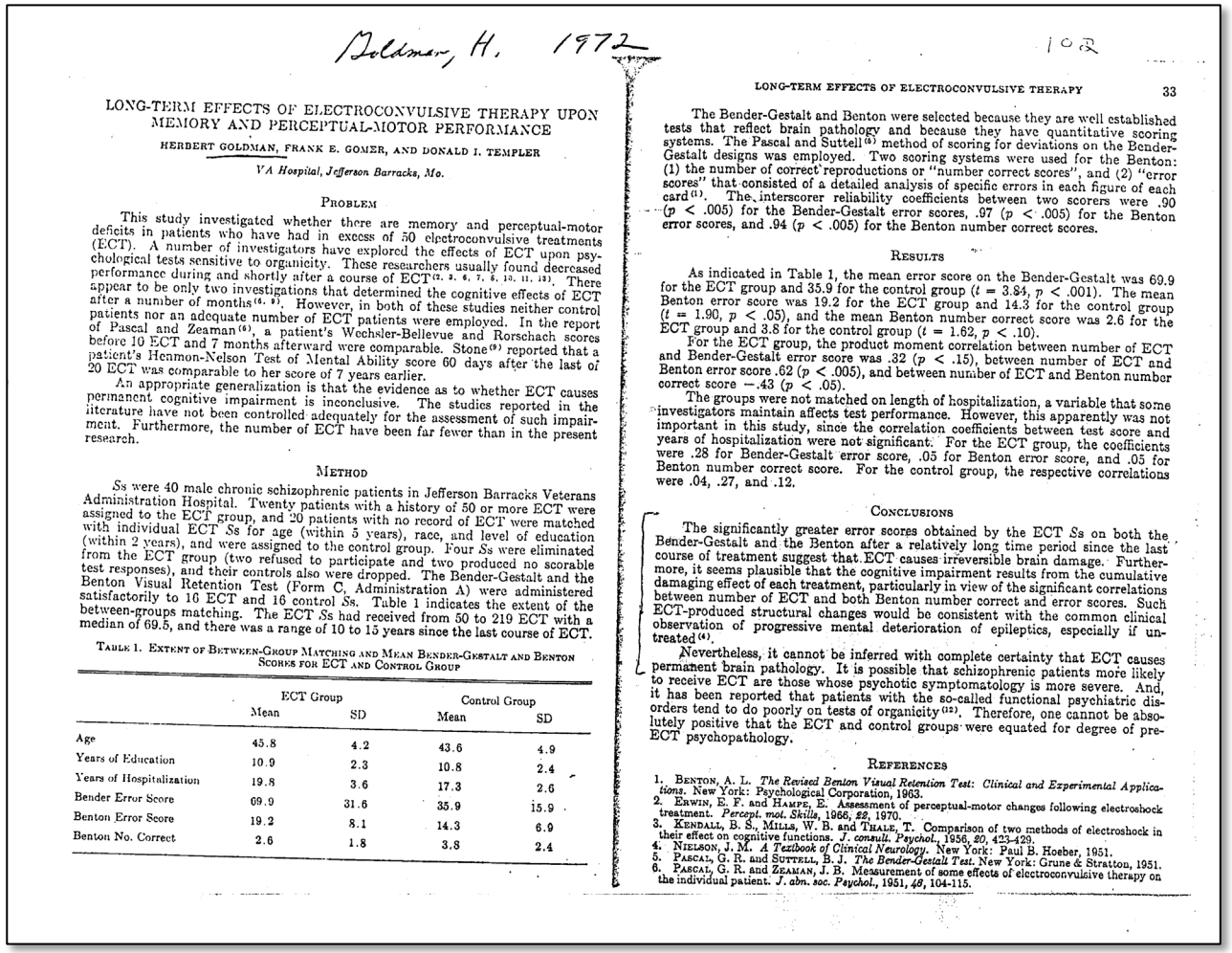

Goldman, H. 1972

102

LONG-TERM EFFECTS OF ELECTROCONVULSIVE THERAPY UPON MEMORY AND PERCEPTUAL-MOTOR PERFORMANCE

HERBERT GOLDMAN, FRANK E. GOMER, AND DONALD I. TEMPLER

VA Hospital, Jefferson Barracks, Mo.

PROBLEM

This study investigated whether there are memory and perceptual-motor deficits in patients who have had in excess of 50 electroconvulsive treatments (ECT). A number of investigators have explored the effects of ECT upon psychological tests sensitive to organicity. These researchers usually found decreased performance during and shortly after a course of ECT[2, 3, 6, 7, 8, 10, 11, 13]. There appear to be only two investigations that determined the cognitive effects of ECT after a number of months[6, 9]. However, in both of these studies neither control patients nor an adequate number of ECT patients were employed. In the report of Pascal and Zeaman[6], a patient's Wechsler-Bellevue and Rorschach scores before 10 ECT and 7 months afterward were comparable. Stone[9] reported that a patient's Henmon-Nelson Test of Mental Ability score 60 days after the last of 20 ECT was comparable to her score of 7 years earlier.

An appropriate generalization is that the evidence as to whether ECT causes permanent cognitive impairment is inconclusive. The studies reported in the literature have not been controlled adequately for the assessment of such impairment. Furthermore, the number of ECT have been far fewer than in the present research.

METHOD

Ss were 40 male chronic schizophrenic patients in Jefferson Barracks Veterans Administration Hospital. Twenty patients with a history of 50 or more ECT were assigned to the ECT group, and 20 patients with no record of ECT were matched with individual ECT *Ss* for age (within 5 years), race, and level of education (within 2 years), and were assigned to the control group. Four *Ss* were eliminated from the ECT group (two refused to participate and two produced no scorable test responses), and their controls also were dropped. The Bender-Gestalt and the Benton Visual Retention Test (Form C, Administration A) were administered satisfactorily to 16 ECT and 16 control *Ss*. Table 1 indicates the extent of the between-groups matching. The ECT *Ss* had received from 50 to 219 ECT with a median of 69.5, and there was a range of 10 to 15 years since the last course of ECT.

TABLE 1. EXTENT OF BETWEEN-GROUP MATCHING AND MEAN BENDER-GESTALT AND BENTON SCORES FOR ECT AND CONTROL GROUP

	ECT Group		Control Group	
	Mean	SD	Mean	SD
Age	45.8	4.2	43.6	4.9
Years of Education	10.9	2.3	10.8	2.4
Years of Hospitalization	19.8	3.6	17.3	2.6
Bender Error Score	69.9	31.6	35.9	15.9
Benton Error Score	19.2	8.1	14.3	6.9
Benton No. Correct	2.6	1.8	3.8	2.4

The Bender-Gestalt and Benton were selected because they are well established tests that reflect brain pathology and because they have quantitative scoring systems. The Pascal and Suttell[5] method of scoring for deviations on the Bender-Gestalt designs was employed. Two scoring systems were used for the Benton: (1) the number of correct reproductions or "number correct scores", and (2) "error scores" that consisted of a detailed analysis of specific errors in each figure of each card[1]. The interscorer reliability coefficients between two scorers were .90 ($p < .005$) for the Bender-Gestalt error scores, .97 ($p < .005$) for the Benton error scores, and .94 ($p < .005$) for the Benton number correct scores.

RESULTS

As indicated in Table 1, the mean error score on the Bender-Gestalt was 69.9 for the ECT group and 35.9 for the control group ($t = 3.84$, $p < .001$). The mean Benton error score was 19.2 for the ECT group and 14.3 for the control group ($t = 1.90$, $p < .05$), and the mean Benton number correct score was 2.6 for the ECT group and 3.8 for the control group ($t = 1.62$, $p < .10$).

For the ECT group, the product moment correlation between number of ECT and Bender-Gestalt error score was .32 ($p < .15$), between number of ECT and Benton error score .62 ($p < .005$), and between number of ECT and Benton number correct score $-.43$ ($p < .05$).

The groups were not matched on length of hospitalization, a variable that some investigators maintain affects test performance. However, this apparently was not important in this study, since the correlation coefficients between test score and years of hospitalization were not significant. For the ECT group, the coefficients were .28 for Bender-Gestalt error score, .05 for Benton error score, and .05 for Benton number correct score. For the control group, the respective correlations were .04, .27, and .12.

CONCLUSIONS

The significantly greater error scores obtained by the ECT *Ss* on both the Bender-Gestalt and the Benton after a relatively long time period since the last course of treatment suggest that ECT causes irreversible brain damage. Furthermore, it seems plausible that the cognitive impairment results from the cumulative damaging effect of each treatment, particularly in view of the significant correlations between number of ECT and both Benton number correct and error scores. Such ECT-produced structural changes would be consistent with the common clinical observation of progressive mental deterioration of epileptics, especially if untreated[4].

Nevertheless, it cannot be inferred with complete certainty that ECT causes permanent brain pathology. It is possible that schizophrenic patients more likely to receive ECT are those whose psychotic symptomatology is more severe. And, it has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity[12]. Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

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Goldman Study:

LONG-TERM EFFECTS OF ELECTROCONVULSIVE THERAPY UPON MEMORY AND PERCEPTUAL-MOTOR PERFORMANCE

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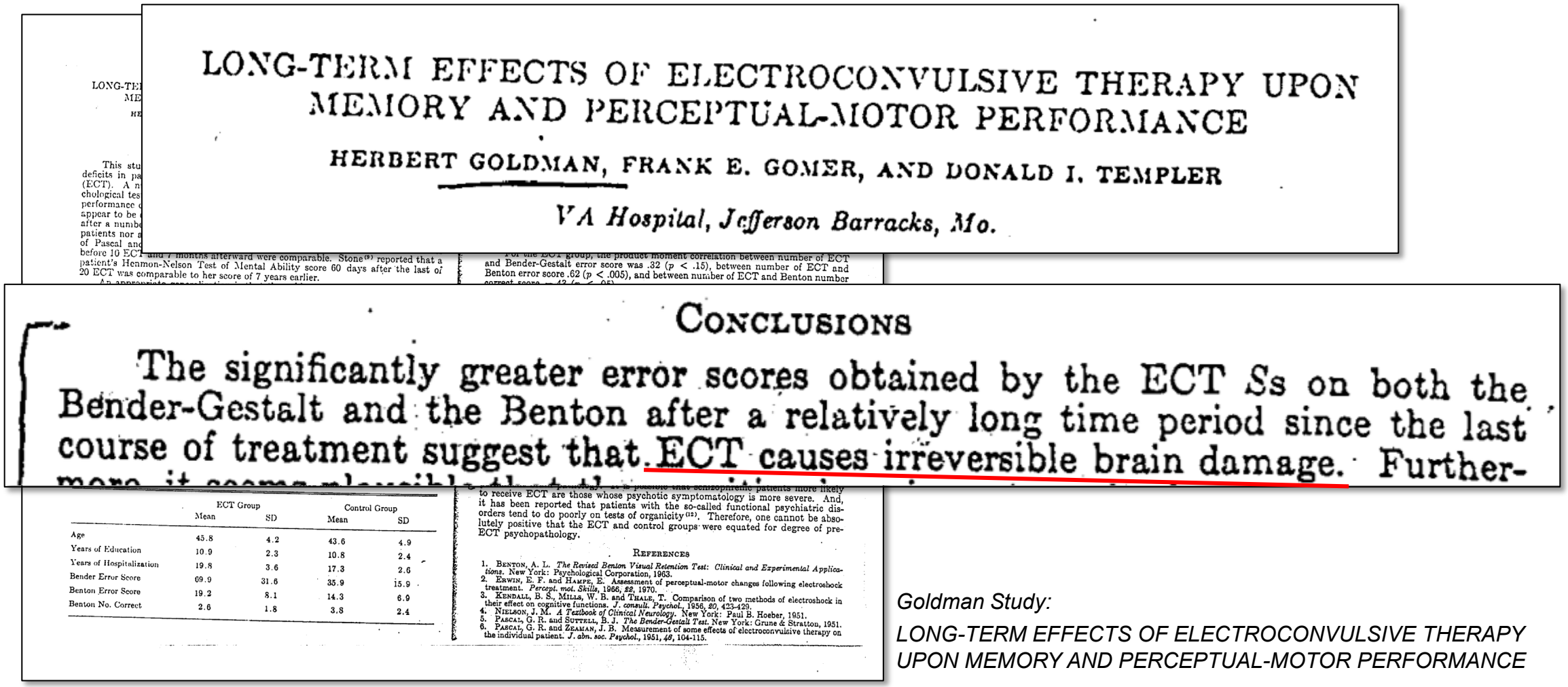

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VA Hospital, Jefferson Barracks, Mo.

CONCLUSIONS

The significantly greater error scores obtained by the ECT Ss on both the Bender-Gestalt and the Benton after a relatively long time period since the last course of treatment suggest that ECT causes irreversible brain damage. Furthermore, it seems plausible that [illegible]

	ECT Group		Control Group	
	Mean	SD	Mean	SD
Age	45.8	4.2	43.6	4.9
Years of Education	10.9	2.3	10.8	2.4
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Benton Error Score	19.2	8.1	14.3	6.9
Benton No. Correct	2.6	1.8	3.8	2.4

to receive ECT are those whose psychotic symptomatology is more severe. And, it has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity[12]. Therefore, one cannot be absolutely positive that the ECT and control groups were equated for degree of pre-ECT psychopathology.

REFERENCES

1. BENTON, A. L. *The Revised Benton Visual Retention Test: Clinical and Experimental Applications.* New York: Psychological Corporation, 1963.
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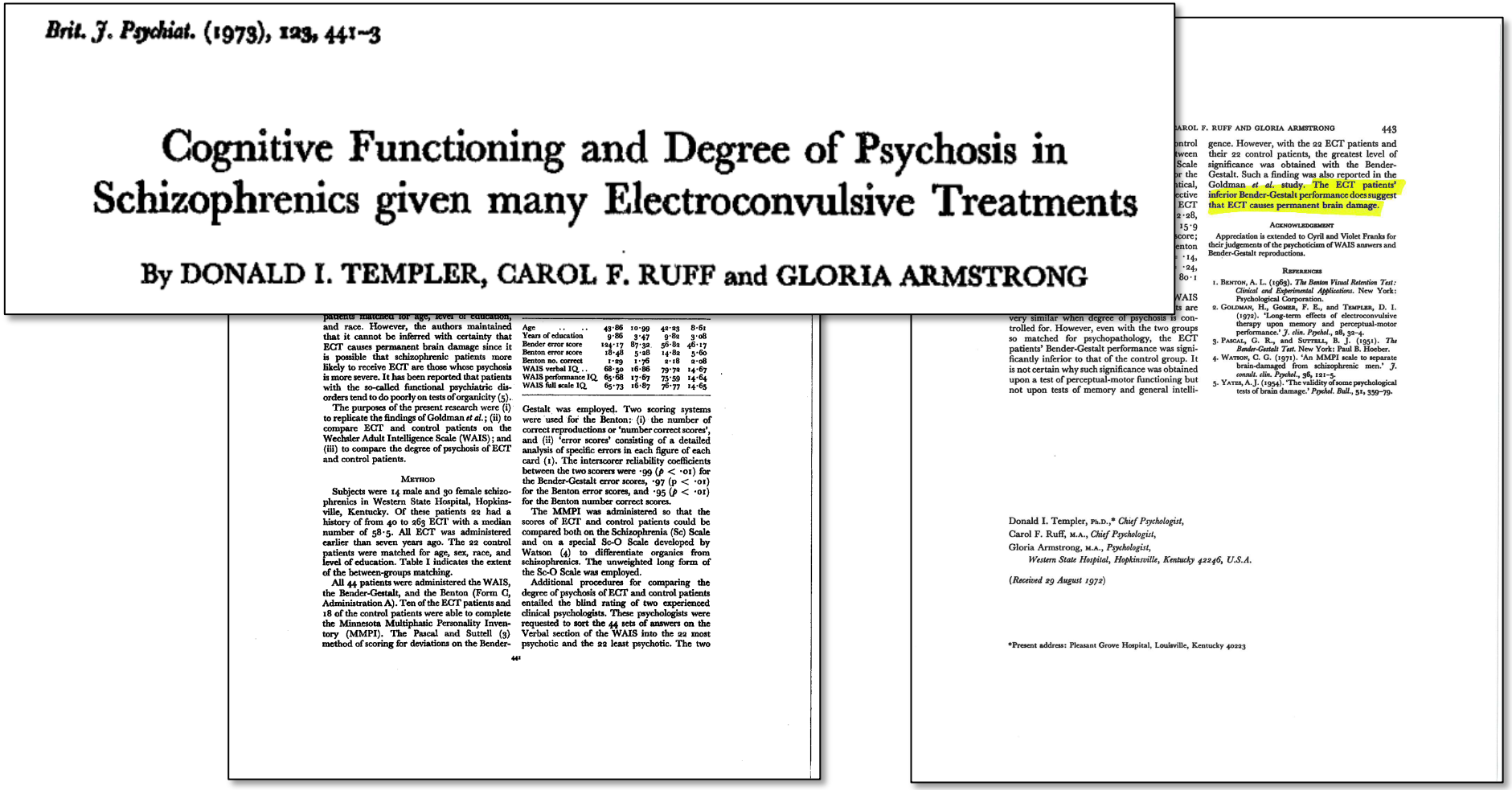

Brit. J. Psychiat. (1973), 123, 441–3

Cognitive Functioning and Degree of Psychosis in Schizophrenics given many Electroconvulsive Treatments

By DONALD I. TEMPLER, CAROL F. RUFF and GLORIA ARMSTRONG

patients matched for age, level of education, and race. However, the authors maintained that it cannot be inferred with certainty that ECT causes permanent brain damage since it is possible that schizophrenic patients more likely to receive ECT are those whose psychosis is more severe. It has been reported that patients with the so-called functional psychiatric disorders tend to do poorly on tests of organicity (5).

The purposes of the present research were (i) to replicate the findings of Goldman *et al.*; (ii) to compare ECT and control patients on the Wechsler Adult Intelligence Scale (WAIS); and (iii) to compare the degree of psychosis of ECT and control patients.

Method

Subjects were 14 male and 30 female schizophrenics in Western State Hospital, Hopkinsville, Kentucky. Of these patients 22 had a history of from 40 to 263 ECT with a median number of 58·5. All ECT was administered earlier than seven years ago. The 22 control patients were matched for age, sex, race, and level of education. Table I indicates the extent of the between-groups matching.

All 44 patients were administered the WAIS, the Bender-Gestalt, and the Benton (Form C, Administration A). Ten of the ECT patients and 18 of the control patients were able to complete the Minnesota Multiphasic Personality Inventory (MMPI). The Pascal and Suttell (3) method of scoring for deviations on the Bender-

Age	43·86	10·99	42·23	8·61
Years of education	9·86	3·47	9·82	3·08
Bender error score	124·17	87·32	56·82	46·17
Benton error score	18·48	5·28	14·82	5·60
Benton no. correct	1·89	1·76	2·18	2·08
WAIS verbal IQ	68·50	16·86	79·72	14·67
WAIS performance IQ	65·68	17·67	75·59	14·64
WAIS full scale IQ	65·73	16·87	76·77	14·65

Gestalt was employed. Two scoring systems were used for the Benton: (i) the number of correct reproductions or 'number correct scores', and (ii) 'error scores' consisting of a detailed analysis of specific errors in each figure of each card (1). The intercorer reliability coefficients between the two scorers were ·99 ($p < ·01$) for the Bender-Gestalt error scores, ·97 ($p < ·01$) for the Benton error scores, and ·95 ($p < ·01$) for the Benton number correct scores.

The MMPI was administered so that the scores of ECT and control patients could be compared both on the Schizophrenia (Sc) Scale and on a special Sc-O Scale developed by Watson (4) to differentiate organics from schizophrenics. The unweighted long form of the Sc-O Scale was employed.

Additional procedures for comparing the degree of psychosis of ECT and control patients entailed the blind rating of two experienced clinical psychologists. These psychologists were requested to sort the 44 sets of answers on the Verbal section of the WAIS into the 22 most psychotic and the 22 least psychotic. The two

441

AROL F. RUFF AND GLORIA ARMSTRONG 443

gence. However, with the 22 ECT patients and their 22 control patients, the greatest level of significance was obtained with the Bender-Gestalt. Such a finding was also reported in the Goldman *et al.* study. The ECT patients' inferior Bender-Gestalt performance does suggest that ECT causes permanent brain damage.

Acknowledgement

Appreciation is extended to Cyril and Violet Franks for their judgements of the psychoticism of WAIS answers and Bender-Gestalt reproductions.

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score as closely as possible with 10 control patients. The mean absolute difference between these ECT and control patients on the Sc Scale is 1·9 points. The mean Sc Scale scores for the ECT and control patients were almost identical, 40·90 and 40·80 respectively. The respective mean difference for these MMPI matched ECT and control patients is 76·9 and 35·9 ($t = 2·28$, $p < ·05$) for Bender-Gestalt error score; 15·9 and 14·0 ($t = 1·01$, n.s.) for Benton error score; 2·10 and 2·00 ($t = 1·00$, n.s.) for Benton number correct score; 77·1 and 82·1 ($t = ·14$, n.s.) for Verbal IQ; 78·3 and 79·8 ($t = ·24$, n.s.) for Performance IQ; and 76·1 and 80·1 ($t = ·78$, n.s.) for Full Scale IQ.

It is apparent that the Benton and WAIS performances of ECT and control patients are very similar when degree of psychosis is controlled for. However, even with the two groups so matched for psychopathology, the ECT patients' Bender-Gestalt performance was significantly inferior to that of the control group. It is not certain why such significance was obtained upon a test of perceptual-motor functioning but not upon tests of memory and general intelligence. However, with the 22 ECT patients and their 22 control patients, the greatest level of significance was obtained with the Bender-Gestalt. Such a finding was also reported in the Goldman *et al.* study. The ECT patients' inferior Bender-Gestalt performance does suggest that ECT causes permanent brain damage.

ACKNOWLEDGEMENT

Appreciation is extended to Cyril and Violet Franks for their judgements of the psychoticism of WAIS answers and Bender-Gestalt reproductions.

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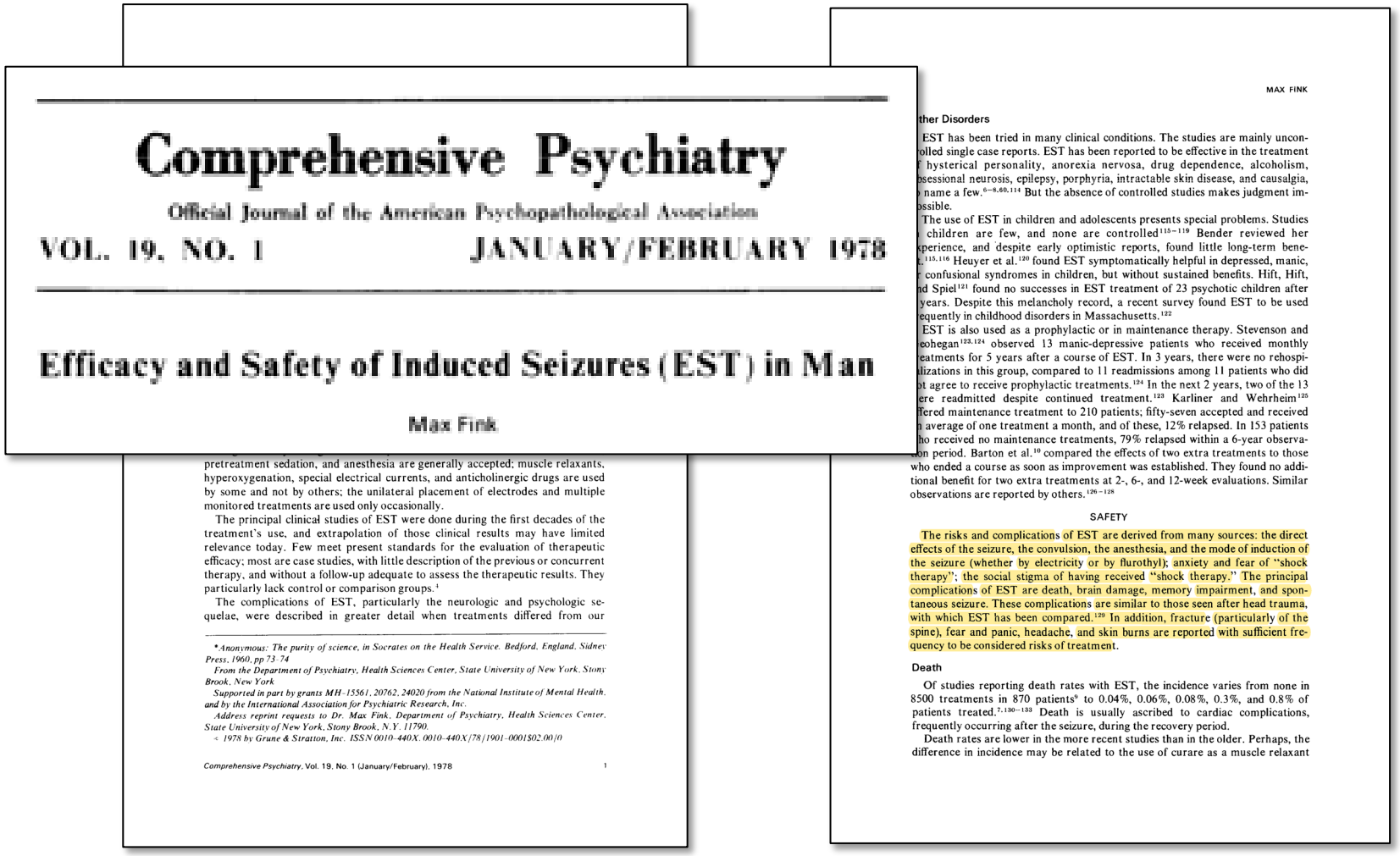

Comprehensive Psychiatry

Official Journal of the American Psychopathological Association

VOL. 19, NO. 1 JANUARY/FEBRUARY 1978

Efficacy and Safety of Induced Seizures (EST) in Man

Max Fink

pretreatment sedation, and anesthesia are generally accepted; muscle relaxants, hyperoxygenation, special electrical currents, and anticholinergic drugs are used by some and not by others; the unilateral placement of electrodes and multiple monitored treatments are used only occasionally.

The principal clinical studies of EST were done during the first decades of the treatment's use, and extrapolation of those clinical results may have limited relevance today. Few meet present standards for the evaluation of therapeutic efficacy; most are case studies, with little description of the previous or concurrent therapy, and without a follow-up adequate to assess the therapeutic results. They particularly lack control or comparison groups.[1]

The complications of EST, particularly the neurologic and psychologic sequelae, were described in greater detail when treatments differed from our

*Anonymous: The purity of science, in Socrates on the Health Service. Bedford, England, Sidney Press, 1960, pp 73-74

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ther Disorders

EST has been tried in many clinical conditions. The studies are mainly uncon-olled single case reports. EST has been reported to be effective in the treatment hysterical personality, anorexia nervosa, drug dependence, alcoholism, bsessional neurosis, epilepsy, porphyria, intractable skin disease, and causalgia, name a few.[6-8,60,114] But the absence of controlled studies makes judgment im-ossible.

The use of EST in children and adolescents presents special problems. Studies children are few, and none are controlled[115-119] Bender reviewed her xperience, and despite early optimistic reports, found little long-term bene-.[115,116] Heuyer et al.[120] found EST symptomatically helpful in depressed, manic, confusional syndromes in children, but without sustained benefits. Hift, Hift, nd Spiel[121] found no successes in EST treatment of 23 psychotic children after years. Despite this melancholy record, a recent survey found EST to be used equently in childhood disorders in Massachusetts.[122]

EST is also used as a prophylactic or in maintenance therapy. Stevenson and eohegan[123,124] observed 13 manic-depressive patients who received monthly eatments for 5 years after a course of EST. In 3 years, there were no rehospi-lizations in this group, compared to 11 readmissions among 11 patients who did ot agree to receive prophylactic treatments.[124] In the next 2 years, two of the 13 ere readmitted despite continued treatment.[123] Karliner and Wehrheim[125] fered maintenance treatment to 210 patients; fifty-seven accepted and received average of one treatment a month, and of these, 12% relapsed. In 153 patients ho received no maintenance treatments, 79% relapsed within a 6-year observa-on period. Barton et al.[10] compared the effects of two extra treatments to those who ended a course as soon as improvement was established. They found no addi-tional benefit for two extra treatments at 2-, 6-, and 12-week evaluations. Similar observations are reported by others.[126-128]

SAFETY

The risks and complications of EST are derived from many sources: the direct effects of the seizure, the convulsion, the anesthesia, and the mode of induction of the seizure (whether by electricity or by flurothyl); anxiety and fear of "shock therapy"; the social stigma of having received "shock therapy." The principal complications of EST are death, brain damage, memory impairment, and spontaneous seizure. These complications are similar to those seen after head trauma, with which EST has been compared.[129] In addition, fracture (particularly of the spine), fear and panic, headache, and skin burns are reported with sufficient frequency to be considered risks of treatment.

Death

Of studies reporting death rates with EST, the incidence varies from none in 8500 treatments in 870 patients[9] to 0.04%, 0.06%, 0.08%, 0.3%, and 0.8% of patients treated.[7,130-133] Death is usually ascribed to cardiac complications, frequently occurring after the seizure, during the recovery period.

Death rates are lower in the more recent studies than in the older. Perhaps, the difference in incidence may be related to the use of curare as a muscle relaxant

(1978) Fink Study

Thelen v. Somatics, LLC

were readmitted despite continued treatment.[123] Karliner and Wehrheim[125] offered maintenance treatment to 210 patients; fifty-seven accepted and received an average of one treatment a month, and of these, 12% relapsed. In 153 patients who received no maintenance treatments, 79% relapsed within a 6-year observation period. Barton et al.[10] compared the effects of two extra treatments to those who ended a course as soon as improvement was established. They found no additional benefit for two extra treatments at 2-, 6-, and 12-week evaluations. Similar observations are reported by others.[126–128]

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Death rates are lower in the more recent studies than in the older. Perhaps, the difference in incidence may be related to the use of curare as a muscle relaxant

Failure to Warn

Electroconvulsive Shock Induces Neuron Death in the Mouse Hippocampus: Correlation of Neurodegeneration with Convulsive Activity

I. I. Zarubenko, A. A. Yakovlev, M. Yu. Stepanichev, and N. V. Gulyaeva

totic nuclei (the TUNEL method). In addition, the activity of caspase-3, the key enzyme of apoptosis, was measured in brain areas immediately after completion of electric shocks. The number of neurons decreased significantly in field CA1 and the dentate fascia, but not in hippocampal field CA3. The numbers of cells in CA1 and CA3 were inversely correlated with the intensity of convulsions. Signs of apoptotic neuron death were not seen, while caspase-3 activity was significantly decreased in the hippocampus after electric shocks. These data support the notion that functional changes affect neurons after electric shock and deepen our understanding of this view, providing direct evidence that there are moderate (up to 10%) but significant levels of neuron death in defined areas of the hippocampus. Inverse correlations of the numbers of cells with the extent of convulsive activity suggest that the main cause of neuron death is convulsions evoked by electric shocks.

KEY WORDS: convulsive activity, electric shocks, hippocampus, apoptosis, caspase-3.

Data on selective cell death in the brain in conditions of convulsive activity were first obtained some 100 years ago [34]. Despite many studies in this area, there is still no unambiguous answer to the fundamental question of whether convulsions are the consequence or cause of cell death in the brain. A number of studies have demonstrated that convulsive activity can develop as a result of brain damage, though there is support for the notion that in some situations, convulsive activity induces brain damage. This latter depends on many factors, primarily the characteristics of the organism (including age), as well as on the type and duration of convulsive activity. For example, status epilepticus induces significant damage in the brain, while repeated convulsive activity in a number of models did not result in cell death [22].

Institute of Higher Nervous Activity and Neurophysiology, Russian Academy of Sciences, 5a Butlerov Street, 117485 Moscow, Russia.

Convulsive activity can induce neuron death in various parts of the brain. From this point of view, the best studied structure is the hippocampus, as this is believed to be selectively sensitive to the damaging effects of convulsions [7, 36, 42]. In adult animals, status epilepticus induces neuron death in hippocampal fields CA1 and CA3, granule cells in the dentate fascia, and in the hilus. Cell damage is mediated by excess release of neurotransmitters activating NMDA receptor Ca^{2+} channels and opening of potential-dependent Ca^{2+} channels, leading to increased Ca^{2+} influx into the cell. Excess intracellular Ca^{2+} leads to a cascade of biochemical events (excessive generation of active forms of oxygen, activation of nitric oxide synthase, uncoupling of oxidation and phosphorylation in mitochondria, and activation of lipases, proteases, endonucleases, and other catabolic enzymes), resulting in cell death [23]. This was demonstrated in animal experiments using a variety of models of convulsive activity, including single-episode status epilepticus induced by kainic acid [2, 7, 36, 39] and by repeated

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10%) but significant level of neuron death in particular parts of the hippocampus due to shocks. The inverse correlations between the numbers of cells and the severity of convulsions suggests that convulsions are the main cause of neuron death induced by electric shocks. The approaches used here did not allow us to establish definitively whether neuron death occurs by apoptosis, though they exclude necrotic processes.

This study was supported by the Russian Fund for Basic Research (Grant No. 01-04-49476).

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the dentate fascia [18].

CONCLUSION

The data reported here support the concept that significant functional changes following repeated electric shocks do occur; they deepen our understanding of this phenomenon by providing direct evidence for a moderate (up to 10%) but significant level of neuron death in particular parts of the hippocampus due to shocks. The inverse correlations between the numbers of cells and the severity of convulsions suggests that convulsions are the main cause of neuron death induced by electric shocks. The approaches used here did not allow us to establish definitively whether neuron death occurs by apoptosis, though they exclude necrotic processes.

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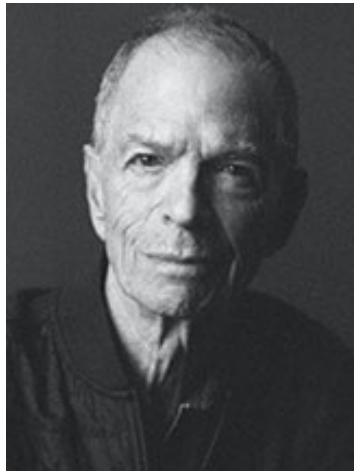
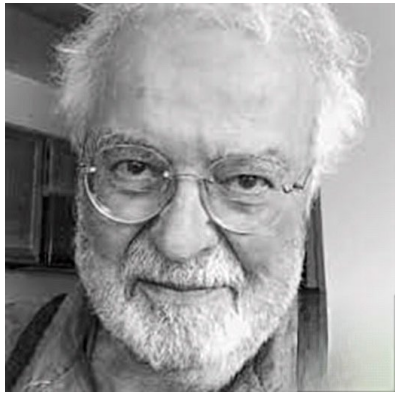
Sterling Quote



Peter Sterling
Neuroscientist at the
University Of Pennsylvania

"One can be sympathetic to psychiatry as I am and still imagine that passing 150 volts between the temples to evoke a grand mal seizure might cause brain damage. Especially, when you realize that this cure for depression, cure in quotes, requires this procedure to be repeated 10 to 20 times over a week or so. And when you talk to a friend who has been so treated and discover that a year later, she is still experiencing huge gaps in recall of major life events, you begin to worry. Finally, you discover that ECT's benefit is only temporary, so that many psychiatrists administer it chronically."



Dr. Richard Abrams
Co-Owner of Somatics LLC

Q. … is it also true that Dr. Sterling is not alone in his comments and opinions that ECT can cause brain injury and permanent memory loss?

A. He is not alone.

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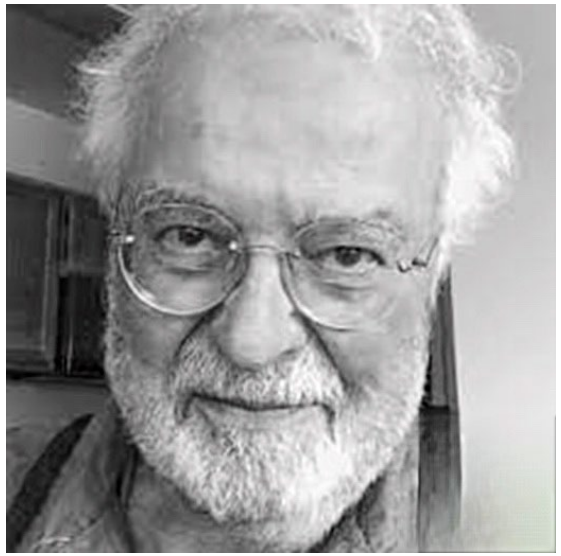

Dr. Richard Abrams
Co-Owner of Somatics LLC

Q.	At any time to the present, has Somatics initiated any studies or tests with regard to this issue of long-term side effects associated with ECT?
A.	**No.**
Q.	Any reason why not?
A.	**That's not our business.**

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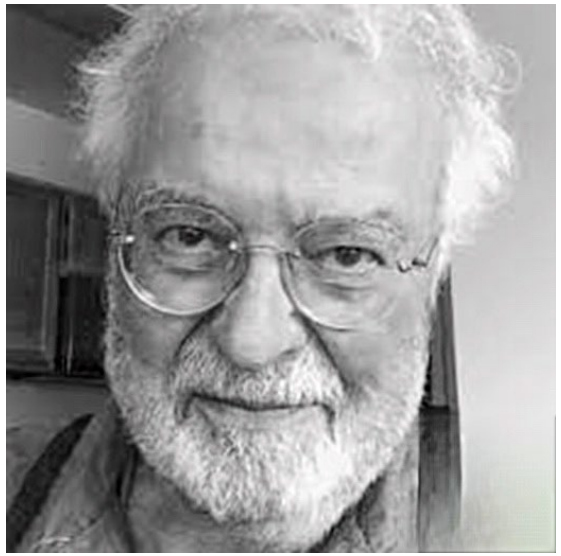

Dr. Richard Abrams
Co-Owner of Somatics LLC

Q.	Up to this point in time, had you reached any conclusions as to how ECT was working in terms of its effectiveness.
A.	**No.**

Q.	Up to the present, do you have any understanding as to the mechanics of how ECT works?
A.	**I do not.**

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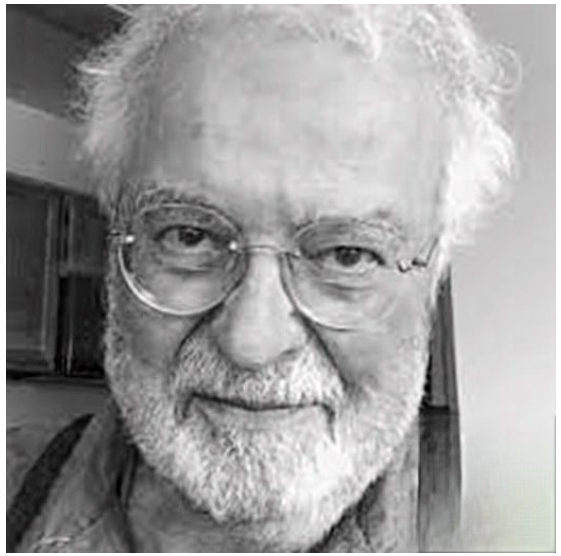

Dr. Richard Abrams
Co-Owner of Somatics LLC

Q. …do you have any understanding that anyone at Somatics has ever incorporated studies of traumatic brain injury with ECT in any way?

A. Certainly not.

Q. Do you know why?

A. There would be no reason to.

Q. Is that because you don't believe that there could be a correlation between TBI, traumatic brain injury, and ECT?

A. Well, we're not in the business of doing studies of traumatic brain injury. We sell Thymatrons.

Thelen v. Somatics, LLC

John Read, Ph.D.

John Read, Ph.D.

- Reviewed all studies that have ever been conducted that compared ECT with sham ECT.
- Gold standard way to determine if a medical treatment works, widely accepted, evidence-based medicine
- Only 11 such studies, all predating 1986
  - Studies would not pass muster today – poor quality.
  - “We can say there has never been a single study showing that ECT is better than sham ECT beyond the end of treatment, or ECT cannot be said to have any long-term benefits.”

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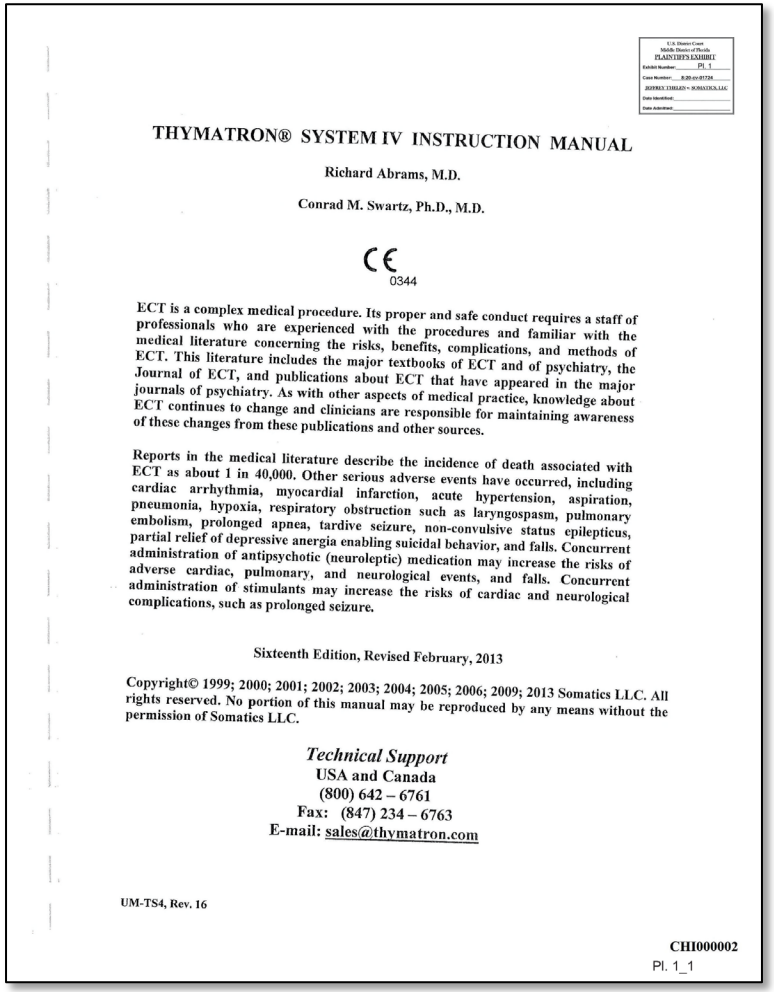

U.S. District Court
Middle District of Florida
PLAINTIFF'S EXHIBIT
Exhibit Number: Pl. 1
Case Number: 8:20-cv-01726
JEFFREY THELEN v. SOMATICS, LLC
Date Identified:
Date Admitted:

THYMATRON® SYSTEM IV INSTRUCTION MANUAL

Richard Abrams, M.D.

Conrad M. Swartz, Ph.D., M.D.

CE
0344

ECT is a complex medical procedure. Its proper and safe conduct requires a staff of professionals who are experienced with the procedures and familiar with the medical literature concerning the risks, benefits, complications, and methods of ECT. This literature includes the major textbooks of ECT and of psychiatry, the Journal of ECT, and publications about ECT that have appeared in the major journals of psychiatry. As with other aspects of medical practice, knowledge about ECT continues to change and clinicians are responsible for maintaining awareness of these changes from these publications and other sources.

Reports in the medical literature describe the incidence of death associated with ECT as about 1 in 40,000. Other serious adverse events have occurred, including cardiac arrhythmia, myocardial infarction, acute hypertension, aspiration, pneumonia, hypoxia, respiratory obstruction such as laryngospasm, pulmonary embolism, prolonged apnea, tardive seizure, non-convulsive status epilepticus, partial relief of depressive anergia enabling suicidal behavior, and falls. Concurrent administration of antipsychotic (neuroleptic) medication may increase the risks of adverse cardiac, pulmonary, and neurological events, and falls. Concurrent administration of stimulants may increase the risks of cardiac and neurological complications, such as prolonged seizure.

Sixteenth Edition, Revised February, 2013

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Plf. Exhibit 1, Thymatron System IV Manual

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U.S. District Court
Middle District of Florida
PLAINTIFF'S EXHIBIT
Exhibit Number: Pl. 31
Case Number: 8:20-cv-01724
JEFFREY THELEN v. SOMATICS, LLC
Date Identified:
Date Admitted:

CHI Health

Immanuel
BEHAVIORAL SERVICES
ELECTROCONVULSIVE INFORMATION AND EDUCATION

Sharon Lush - ECT Coordinator 572-3967

What is ECT? Electroconvulsive therapy (ECT) is a treatment for severe episodes of major depression, mania, and some types of schizophrenia. It involves the use of a brief, controlled electrical stimulus to produce a seizure within the brain. This seizure activity is believed to bring about certain biochemical changes which are expected to cause your symptoms to diminish or to even disappear. An initial course of ECT treatments, generally 6-12, given at a rate of three per week, is required to produce such a therapeutic effect; although sometimes a smaller or larger number may be necessary.

How is ECT administered? ECT is usually administered three times a week, on Monday, Wednesday, and Friday. It can be given on either an inpatient or outpatient basis. You will not eat or drink after midnight the night before each treatment. After you arrive for the treatment, a small needle is placed in a vein so that medications to put you to sleep and relax your muscles can be given. The treatment itself is given in a special ECT Treatment Room, where ECT is administered by your Psychiatrist who has had training and experience in this type of treatment. You will be brought into the treatment room on a hospital cart, after which a blood pressure cuff will be placed on your arm and a number of electrodes will be placed on your scalp, chest, and finger, so that brain waves (EEG), heart waves (EKG), and body oxygen levels can be monitored. An anesthesiologist or anesthetist will provide you oxygen to breathe by mask, and medications will be given by IV which will put you to sleep.

Within a minute after the injection of the anesthetic medication, you will be asleep, and the medication to relax your muscles will be given. A mouth guard will be placed to protect your mouth and teeth. Within one to three minutes, your muscles will be relaxed. A controlled electrical stimulus, lasting a fraction of a second to four seconds, will then be applied across the two stimulus electrodes, which will typically be placed either on both temples (bilateral ECT) or on the right temporal and top of the head (unilateral ECT). The electrical stimulus will trigger a seizure within the brain, which typically lasts around a minute. The muscular response to the seizure is greatly reduced by the muscle relaxant drug given prior to the stimulation. Very little body movement usually occurs.

Within a few minutes after the seizure, when you are breathing well on your own, you will be moved to a nearby room where, you will wake up within 5-10 minutes. Because of the anesthetic drug and the effects of having had the seizure, you will temporarily feel somewhat groggy. Usually within 20-30 minutes after leaving the treatment room, you will be brought back to your room (if you are an inpatient), or you will go to the ECT Waiting Room (if you are an outpatient) where you will wait until you are ready to leave the hospital (typically an hour or more).

Is ECT effective? Although there have been many advances in the treatment of mental disorders in recent years, ECT remains the most effective, fastest, and/or safest treatment for some individuals, particularly when alternative treatments, usually medications, are either not effective or not safe. Your doctor will discuss with you why ECT is being recommended in your case and what alternative treatments may be available.

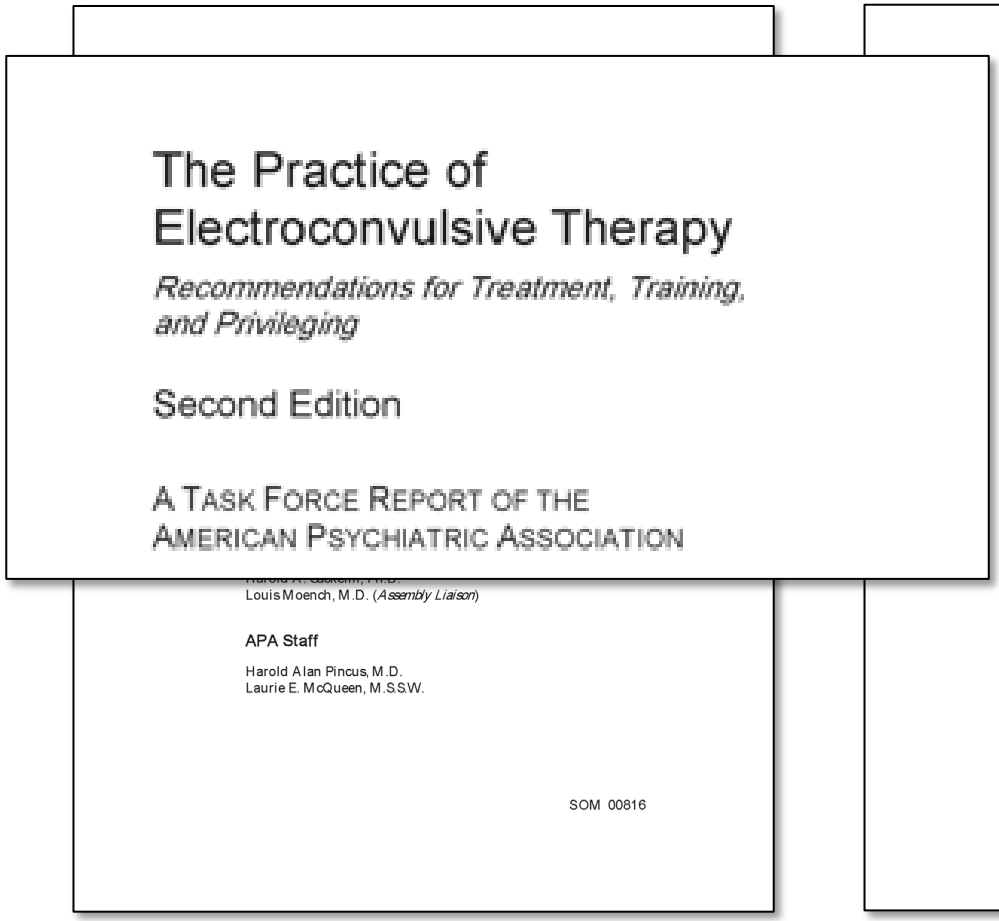
Confusion and memory problems may build up over a course of ECT, but diminish as soon as the treatments have stopped. However, because of the harmful effects that mental disorders themselves often have on memory function, some patients successfully treated with ECT actually report an improvement in memory. When memory problems occur, they vary considerably from patient to patient, but are usually greater for larger numbers of treatments or when both sides of the head are stimulated (bilateral ECT). Because of the possibility of memory loss, it is recommended that important life decisions be postponed until any major effects of ECT on memory have worn off (usually within 2-6 weeks following completion of the treatment).

ECT-related memory problems can be of two types: Difficulty remembering new information, and a loss of some memories from the past, particularly the recent past, e.g., during and just prior to receiving ECT. In this regard, the ability to learn and remember new information returns to one's usual level over a period of days to weeks after ECT. The ability to remember material from the past, i.e., prior to ECT, likewise tends to return to normal over a similar time period, except that in this case, some memories from the recent past, mainly days to months prior to the treatments, may be delayed in recovery or even permanently lost. Patient surveys have reported longer gaps in memory. However, patient surveys have indicated that most patients receiving ECT are not greatly disturbed by memory effects and would have ECT again if it was felt to be indicated.

Plaintiff Exhibit 31, Pg 2

Thelen v. Somatics, LLC

Failure to Warn



The Practice of Electroconvulsive Therapy

Recommendations for Treatment, Training, and Privileging

Second Edition

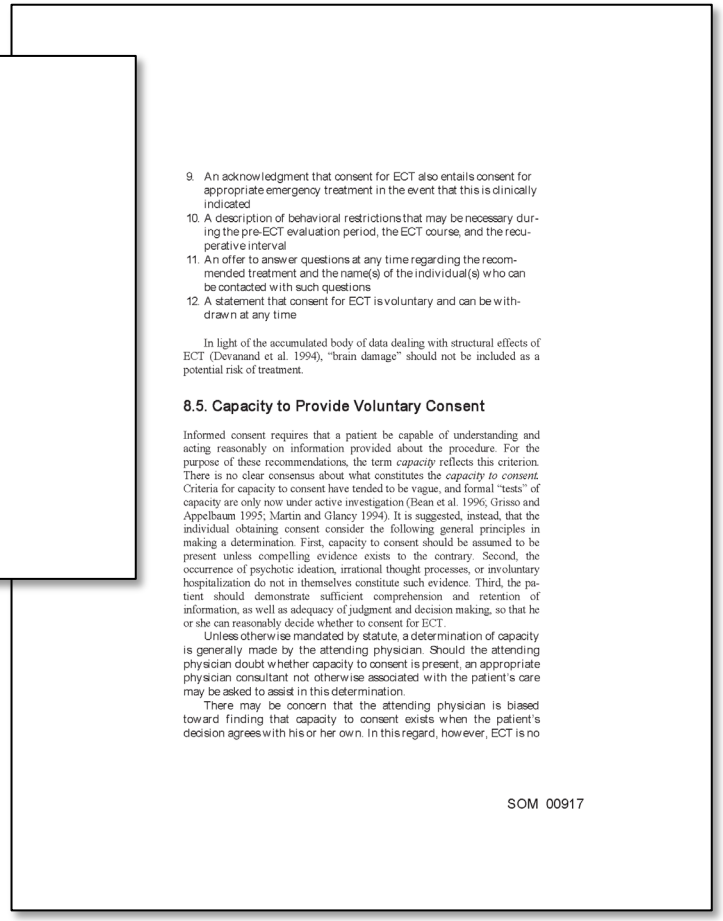
A TASK FORCE REPORT OF THE AMERICAN PSYCHIATRIC ASSOCIATION

Louis Moench, M.D. (*Assembly Liaison*)

APA Staff

Harold Alan Pincus, M.D.
Laurie E. McQueen, M.S.S.W.

SOM 00816



9. An acknowledgment that consent for ECT also entails consent for appropriate emergency treatment in the event that this is clinically indicated
10. A description of behavioral restrictions that may be necessary during the pre-ECT evaluation period, the ECT course, and the recuperative interval
11. An offer to answer questions at any time regarding the recommended treatment and the name(s) of the individual(s) who can be contacted with such questions
12. A statement that consent for ECT is voluntary and can be withdrawn at any time

In light of the accumulated body of data dealing with structural effects of ECT (Devanand et al. 1994), "brain damage" should not be included as a potential risk of treatment.

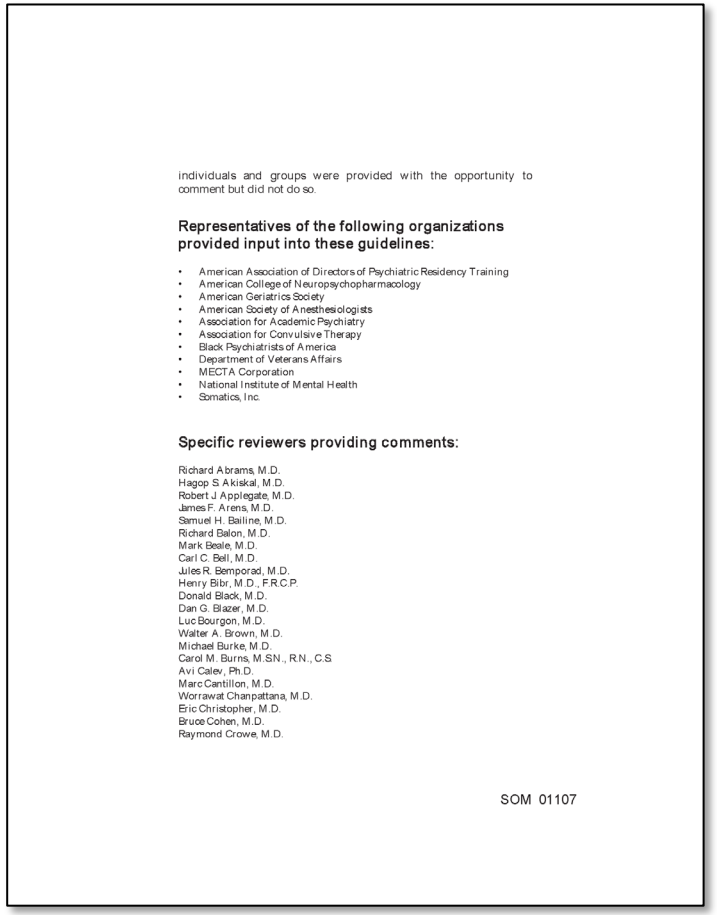
8.5. Capacity to Provide Voluntary Consent

Informed consent requires that a patient be capable of understanding and acting reasonably on information provided about the procedure. For the purpose of these recommendations, the term *capacity* reflects this criterion. There is no clear consensus about what constitutes the *capacity to consent.* Criteria for capacity to consent have tended to be vague, and formal "tests" of capacity are only now under active investigation (Bean et al. 1996; Grisso and Appelbaum 1995; Martin and Glancy 1994). It is suggested, instead, that the individual obtaining consent consider the following general principles in making a determination. First, capacity to consent should be assumed to be present unless compelling evidence exists to the contrary. Second, the occurrence of psychotic ideation, irrational thought processes, or involuntary hospitalization do not in themselves constitute such evidence. Third, the patient should demonstrate sufficient comprehension and retention of information, as well as adequacy of judgment and decision making, so that he or she can reasonably decide whether to consent for ECT.

Unless otherwise mandated by statute, a determination of capacity is generally made by the attending physician. Should the attending physician doubt whether capacity to consent is present, an appropriate physician consultant not otherwise associated with the patient's care may be asked to assist in this determination.

There may be concern that the attending physician is biased toward finding that capacity to consent exists when the patient's decision agrees with his or her own. In this regard, however, ECT is no

SOM 00917



individuals and groups were provided with the opportunity to comment but did not do so.

Representatives of the following organizations provided input into these guidelines:

- American Association of Directors of Psychiatric Residency Training
- American College of Neuropsychopharmacology
- American Geriatrics Society
- American Society of Anesthesiologists
- Association for Academic Psychiatry
- Association for Convulsive Therapy
- Black Psychiatrists of America
- Department of Veterans Affairs
- MECTA Corporation
- National Institute of Mental Health
- Somatics, Inc.

Specific reviewers providing comments:

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Robert J. Applegate, M.D.
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Mark Beale, M.D.
Carl C. Bell, M.D.
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Henry Bibr, M.D., F.R.C.P.
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Dan G. Blazer, M.D.
Luc Bourgon, M.D.
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Avi Calev, Ph.D.
Marc Cantillon, M.D.
Worrawat Chanpattana, M.D.
Eric Christopher, M.D.
Bruce Cohen, M.D.
Raymond Crowe, M.D.

SOM 01107

(2001) The Practice of Electroconvulsive Therapy

Thelen v. Somatics, LLC

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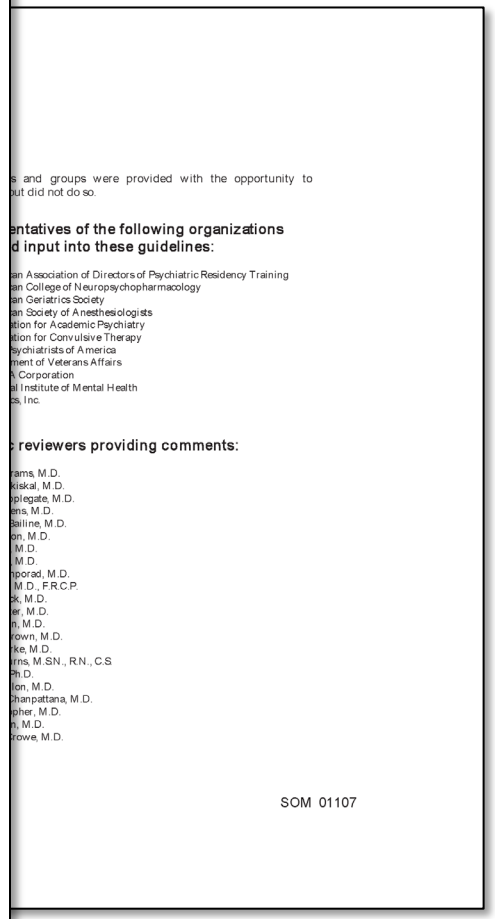

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ation for Convulsive Therapy
sychiatrists of America
ment of Veterans Affairs
Corporation
l Institute of Mental Health
s, Inc.

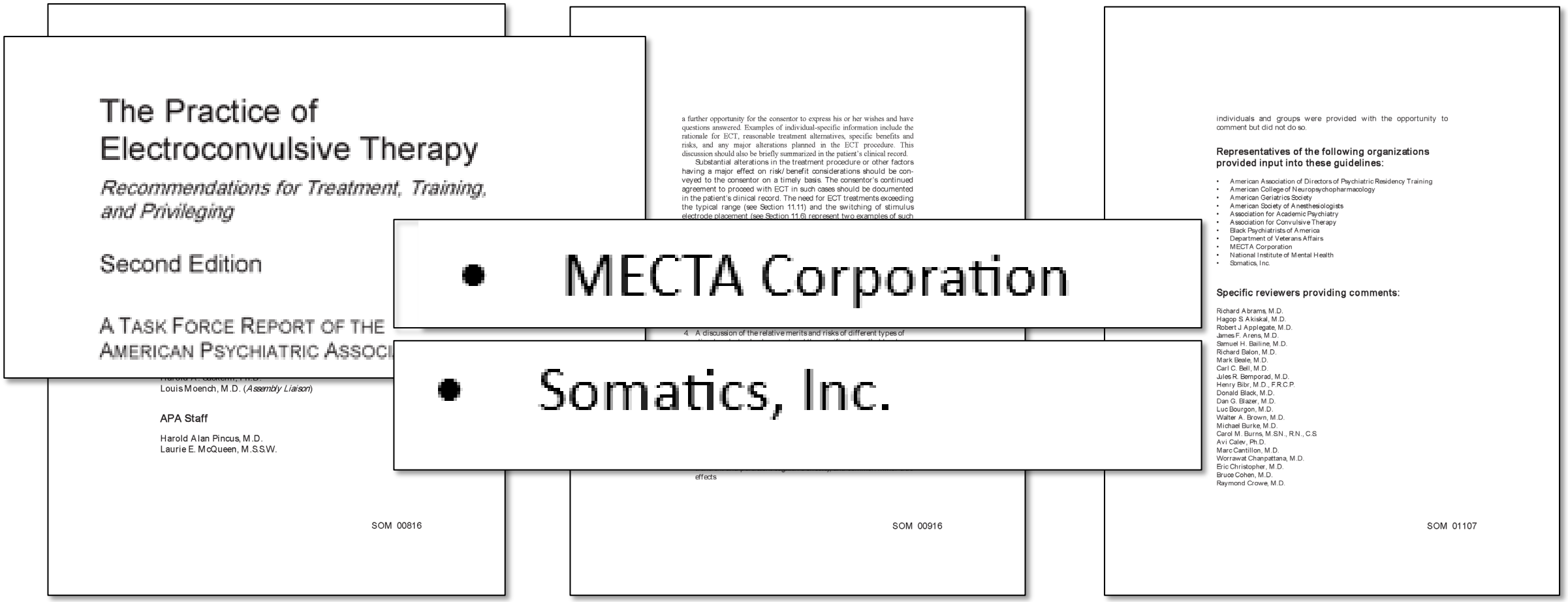
reviewers providing comments:

rams, M.D.
kiskal, M.D.
plegate, M.D.
ens, M.D.
Bailine, M.D.
on, M.D.
M.D.
M.D.
porad, M.D.
M.D., F.R.C.P.
k, M.D.
er, M.D.
n, M.D.
rown, M.D.
ke, M.D.
rns, M.S.N., R.N., C.S.
Ph.D.
on, M.D.
hanpattana, M.D.
pher, M.D.
n, M.D.
rowe, M.D.

SOM 01107

e of Electroconvulsive Therapy

Failure to Warn



(2001) The Practice of Electroconvulsive Therapy

Thelen v. Somatics, LLC

Failure to Warn

From: Conrad Swartz <cswartz@gmail.com>
Sent: Tuesday, December 12, 2006 4:34 PM
To: Holly Lisanby
Subject: book plan up to now
Attachments: ECTmonog.rtf

Hi, Holly

Attached is the up-to-date book plan. Please elucidate the last section on TMS, VNS, and DBS. Please comment on the rest as you like.

Best regards,
Conrad

Failure to Warn

The other book is American Psychiatric Press' APA Task Force report (2001), "The Practice of Electroconvulsive Therapy. Recommendations for Treatment, Training, and Privileging. Second Edition." It has 243 large-print pages plus references. This book is psychiatrist-centered and apparently aims to make permissible as much as possible. This is what a Task Force should do. To illustrate the extremity this is taken, the book section section on patient selection figuratively throws up its hands about which patients with major depression should or should not receive ECT. It is a book of administrative policy that withholds basic judgments--including clinical advice about what works best. This book probably decreases litigation risks because virtually everything is permissible. Although this is nice for clinicians it provides virtually no guidance about how to practice.

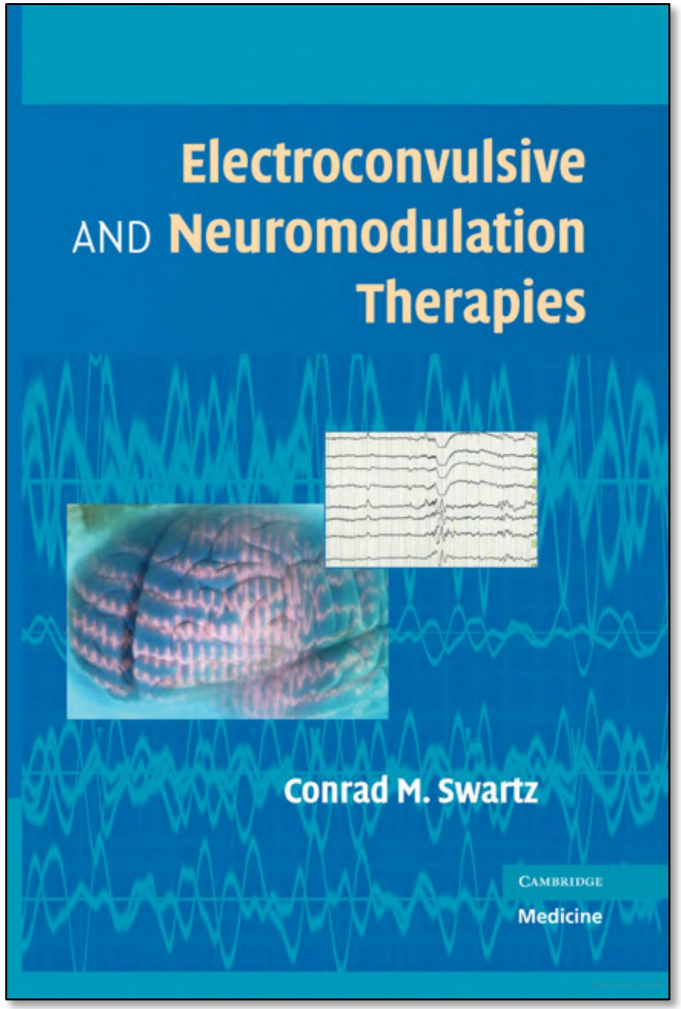
Thelen v. Somatics, LLC

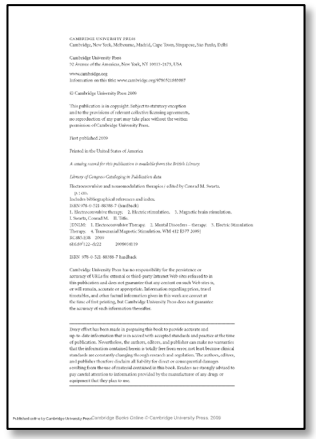
Failure to Warn

Sine Wave vs. Brief Pulse

- Generates the same electrical output.
- Same risk as brain damage exists with sine wave as brief pulse.
  - Castleman
  - Read
- Brief pulse has never been proven to safer than sine wave.

Failure to Warn





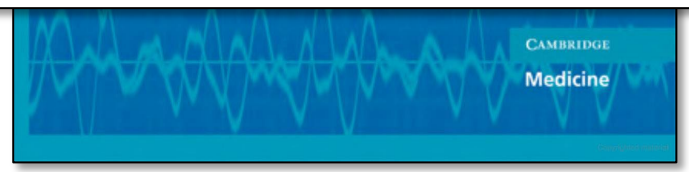
(2009) ECT and Neuromodulation Therapies

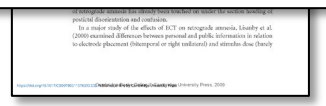
Thelen v. Somatics, LLC

mathematical relationship: *charge rate* (mC/s) = 2 × *frequency* (Hz) × *pulse width* (ms) × *current* (A).

Sine wave versus brief-pulse stimuli

The report that brief-pulse stimuli have milder side effects and use less charge than sine wave stimuli do is well known (Weiner et al., 1986). However, the result was never proven as just stated. Although the comparison found milder side effects and less charge with brief-pulse stimuli, there were large electrical differences between stimuli outside of wave shape. There are several reasons why the clinical differences





(2009) ECT and Neuromodulation Therapies

Failure to Warn

Dr. Conrad Swartz
Owner of Somatics

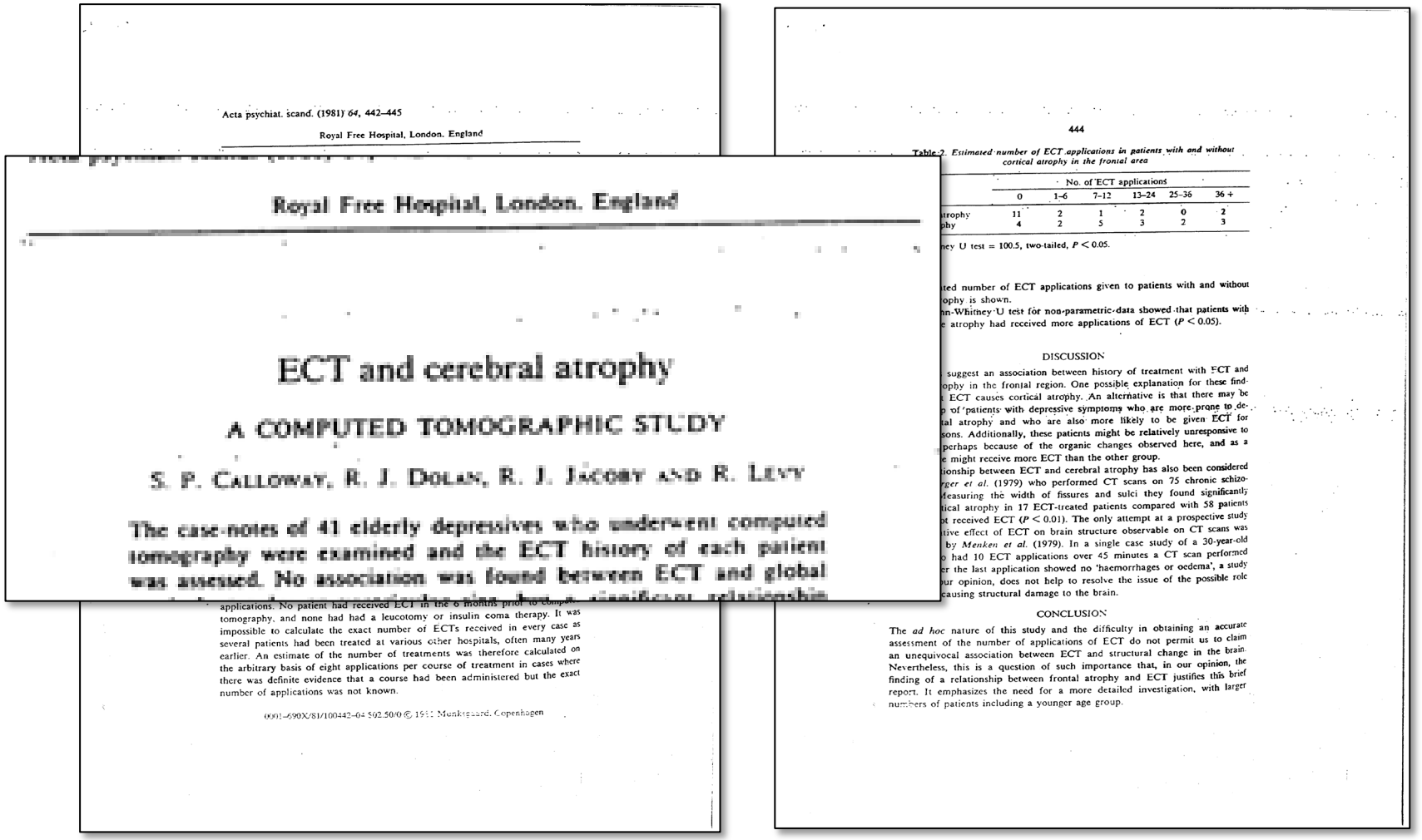
Q.	The section here, sine wave versus brief pulse stimuli. Since these were your own words, I'll let you read them please?
A.	The report that brief pulse stimuli have milder side effects and use less charge than sine wave stimuli do is well known. **However, the result was never proven as just stated**.

Structural Brain Damage

- Autopsy showed irreversible brain damage.
- Not all injuries will appear on imaging studies.
  - Dr. Read
  - Dr. Omalu
  - Calloway Article

(1981) Calloway Study

Failure to Warn



Acta psychiat. scand. (1981) 64, 442–445

Royal Free Hospital, London, England

Royal Free Hospital, London, England

ECT and cerebral atrophy

A COMPUTED TOMOGRAPHIC STUDY

S. P. Calloway, R. J. Dolan, R. J. Jacoby and R. Levy

The case-notes of 41 elderly depressives who underwent computed tomography were examined and the ECT history of each patient was assessed. No association was found between ECT and global

applications. No patient had received ECT in the 6 months prior to [illegible] tomography, and none had had a leucotomy or insulin coma therapy. It was impossible to calculate the exact number of ECTs received in every case as several patients had been treated at various other hospitals, often many years earlier. An estimate of the number of treatments was therefore calculated on the arbitrary basis of eight applications per course of treatment in cases where there was definite evidence that a course had been administered but the exact number of applications was not known.

0901–690X/81/100442–04 $02.50/0 © 1981 Munksgaard, Copenhagen

Table 2. Estimated number of ECT applications in patients with and without cortical atrophy in the frontal area

	No. of ECT applications					
	0	1–6	7–12	13–24	25–36	36 +
trophy	11	2	1	2	0	2
phy	4	2	5	3	2	3

ey U test = 100.5, two-tailed, $P < 0.05$.

ted number of ECT applications given to patients with and without ophy is shown.
nn-Whitney U test for non-parametric data showed that patients with atrophy had received more applications of ECT ($P < 0.05$).

DISCUSSION

suggest an association between history of treatment with ECT and ophy in the frontal region. One possible explanation for these find- ECT causes cortical atrophy. An alternative is that there may be of patients with depressive symptoms who are more prone to de- al atrophy and who are also more likely to be given ECT for sons. Additionally, these patients might be relatively unresponsive to perhaps because of the organic changes observed here, and as a might receive more ECT than the other group.
lionship between ECT and cerebral atrophy has also been considered ger *et al.* (1979) who performed CT scans on 75 chronic schizo- measuring the width of fissures and sulci they found significantly ical atrophy in 17 ECT-treated patients compared with 58 patients t received ECT ($P < 0.01$). The only attempt at a prospective study tive effect of ECT on brain structure observable on CT scans was by *Menken et al.* (1979). In a single case study of a 30-year-old had 10 ECT applications over 45 minutes a CT scan performed er the last application showed no 'haemorrhages or oedema', a study ur opinion, does not help to resolve the issue of the possible role causing structural damage to the brain.

CONCLUSION

The *ad hoc* nature of this study and the difficulty in obtaining an accurate assessment of the number of applications of ECT do not permit us to claim an unequivocal association between ECT and structural change in the brain. Nevertheless, this is a question of such importance that, in our opinion, the finding of a relationship between frontal atrophy and ECT justifies this brief report. It emphasizes the need for a more detailed investigation, with larger numbers of patients including a younger age group.

(1981) Calloway Study

Thelen v. Somatics, LLC

Failure

Table 2. *Estimated number of ECT applications in patients with and without cortical atrophy in the frontal area*

	No. of ECT applications					
	0	1–6	7–12	13–24	25–36	36 +
No atrophy	11	2	1	2	0	2
Atrophy	4	2	5	3	2	3

Mann-Whitney U test = 100.5, two-tailed, $P < 0.05$.

The estimated number of ECT applications given to patients with and without cortical atrophy is shown.

The Mann-Whitney U test for non-parametric data showed that patients with frontal lobe atrophy had received more applications of ECT ($P < 0.05$).

DISCUSSION

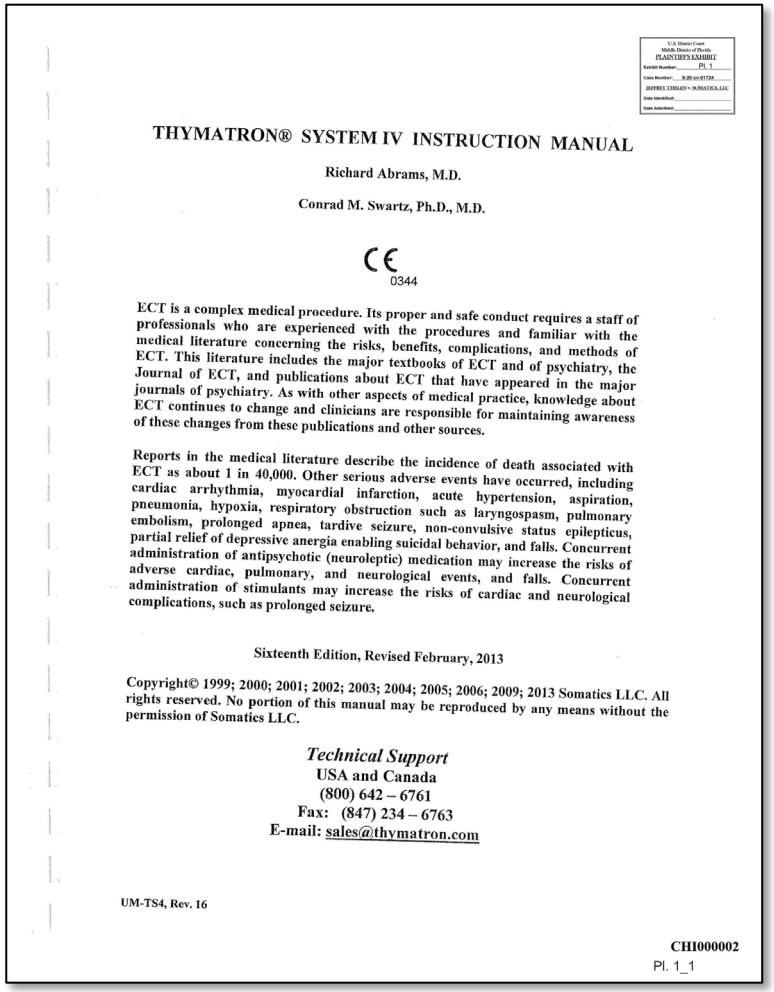
The results suggest an association between history of treatment with ECT and cortical atrophy in the frontal region. One possible explanation for these findings is that ECT causes cortical atrophy. An alternative is that there may be a sub-group of patients with depressive symptoms who are more prone to develop frontal atrophy and who are also more likely to be given ECT for clinical reasons. Additionally, these patients might be relatively unresponsive to treatment, perhaps because of the organic changes observed here, and as a consequence might receive more ECT than the other group.

The relationship between ECT and cerebral atrophy has also been considered by *Weinberger et al.* (1979) who performed CT scans on 75 chronic schizophrenics. Measuring the width of fissures and sulci they found significantly greater cortical atrophy in 17 ECT-treated patients compared with 58 patients who had not received ECT ($P < 0.01$). The only attempt at a prospective study of the putative effect of ECT on brain structure observable on CT scans was undertaken by *Menken et al.* (1979). In a single case study of a 30-year-old woman who had 10 ECT applications over 45 minutes a CT scan performed 3 hours after the last application showed no 'haemorrhages or oedema', a study which, in our opinion, does not help to resolve the issue of the possible role of ECT in causing structural damage to the brain.

CONCLUSION

...way Study

Failure to Warn



U.S. District Court
Middle District of Florida
PLAINTIFF'S EXHIBIT
Exhibit Number: Pl. 1
Case Number: 8:20-cv-01726
JEFFREY THELEN v. SOMATICS, LLC
Date Identified:
Date Admitted:

THYMATRON® SYSTEM IV INSTRUCTION MANUAL

Richard Abrams, M.D.

Conrad M. Swartz, Ph.D., M.D.

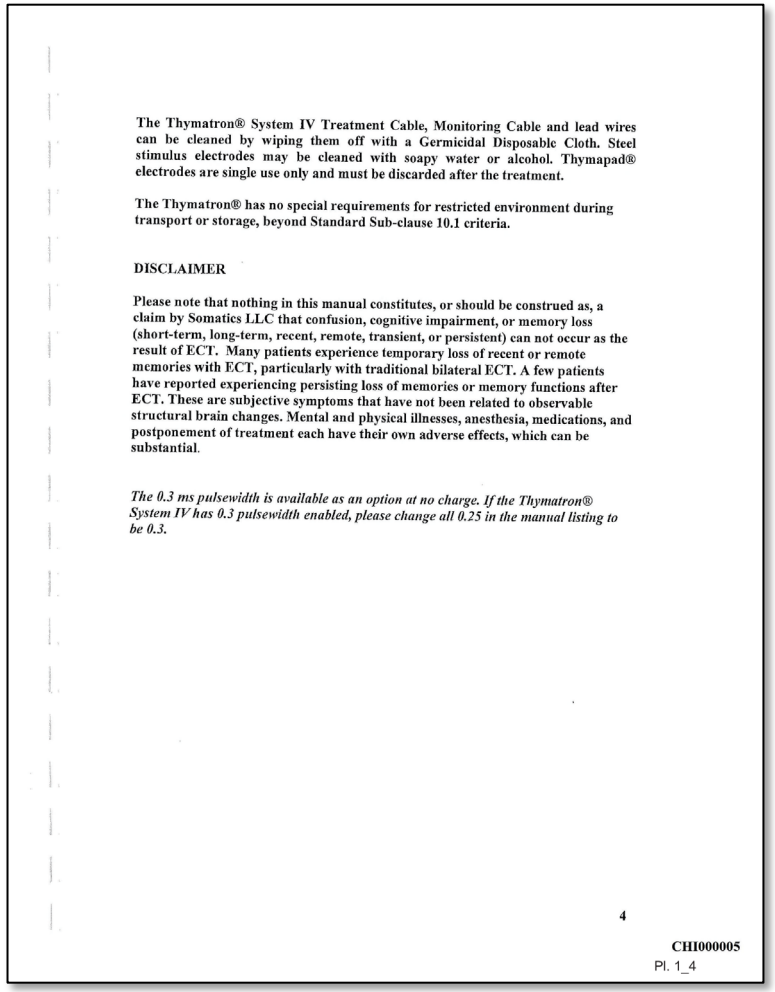
CE 0344

ECT is a complex medical procedure. Its proper and safe conduct requires a staff of professionals who are experienced with the procedures and familiar with the medical literature concerning the risks, benefits, complications, and methods of ECT. This literature includes the major textbooks of ECT and of psychiatry, the Journal of ECT, and publications about ECT that have appeared in the major journals of psychiatry. As with other aspects of medical practice, knowledge about ECT continues to change and clinicians are responsible for maintaining awareness of these changes from these publications and other sources.

Reports in the medical literature describe the incidence of death associated with ECT as about 1 in 40,000. Other serious adverse events have occurred, including cardiac arrhythmia, myocardial infarction, acute hypertension, aspiration, pneumonia, hypoxia, respiratory obstruction such as laryngospasm, pulmonary embolism, prolonged apnea, tardive seizure, non-convulsive status epilepticus, partial relief of depressive anergia enabling suicidal behavior, and falls. Concurrent administration of antipsychotic (neuroleptic) medication may increase the risks of adverse cardiac, pulmonary, and neurological events, and falls. Concurrent administration of stimulants may increase the risks of cardiac and neurological complications, such as prolonged seizure.

Sixteenth Edition, Revised February, 2013

Technical Support
USA and Canada
(800) 642 – 6761
Fax: (847) 234 – 6763
E-mail: sales@thymatron.com



The Thymatron® System IV Treatment Cable, Monitoring Cable and lead wires can be cleaned by wiping them off with a Germicidal Disposable Cloth. Steel stimulus electrodes may be cleaned with soapy water or alcohol. Thymapad® electrodes are single use only and must be discarded after the treatment.

The Thymatron® has no special requirements for restricted environment during transport or storage, beyond Standard Sub-clause 10.1 criteria.

DISCLAIMER

Please note that nothing in this manual constitutes, or should be construed as, a claim by Somatics LLC that confusion, cognitive impairment, or memory loss (short-term, long-term, recent, remote, transient, or persistent) can not occur as the result of ECT. Many patients experience temporary loss of recent or remote memories with ECT, particularly with traditional bilateral ECT. A few patients have reported experiencing persisting loss of memories or memory functions after ECT. These are subjective symptoms that have not been related to observable structural brain changes. Mental and physical illnesses, anesthesia, medications, and postponement of treatment each have their own adverse effects, which can be substantial.

The 0.3 ms pulsewidth is available as an option at no charge. If the Thymatron® System IV has 0.3 pulsewidth enabled, please change all 0.25 in the manual listing to be 0.3.

Plf. Exhibit 1, Thymatron System IV Manual

Failure to Warn

DISCLAIMER

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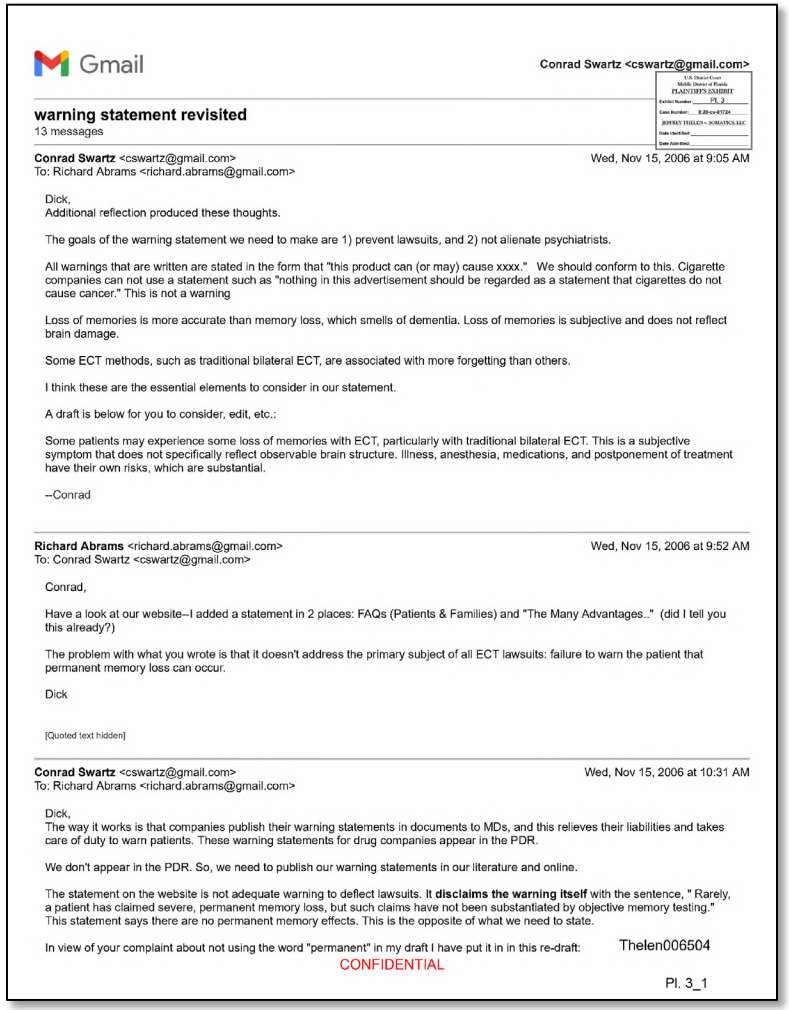
Plf. Exhibit 1, Thymatron System IV Manual

ics, LLC

Failure to Warn



Gmail — Conrad Swartz <cswartz@gmail.com>

U.S. District Court
Middle District of Florida
PLAINTIFF'S EXHIBIT
Exhibit Number: Pl. 3
Case Number: 8:20-cv-01724
JEFFREY THELEN v. SOMATICS, LLC
Date Identified:
Date Admitted:

warning statement revisited
13 messages

Conrad Swartz <cswartz@gmail.com> Wed, Nov 15, 2006 at 9:05 AM
To: Richard Abrams <richard.abrams@gmail.com>

Dick,
Additional reflection produced these thoughts.

The goals of the warning statement we need to make are 1) prevent lawsuits, and 2) not alienate psychiatrists.

All warnings that are written are stated in the form that "this product can (or may) cause xxxx." We should conform to this. Cigarette companies can not use a statement such as "nothing in this advertisement should be regarded as a statement that cigarettes do not cause cancer." This is not a warning

Loss of memories is more accurate than memory loss, which smells of dementia. Loss of memories is subjective and does not reflect brain damage.

Some ECT methods, such as traditional bilateral ECT, are associated with more forgetting than others.

I think these are the essential elements to consider in our statement.

A draft is below for you to consider, edit, etc.:

Some patients may experience some loss of memories with ECT, particularly with traditional bilateral ECT. This is a subjective symptom that does not specifically reflect observable brain structure. Illness, anesthesia, medications, and postponement of treatment have their own risks, which are substantial.

--Conrad

Richard Abrams <richard.abrams@gmail.com> Wed, Nov 15, 2006 at 9:52 AM
To: Conrad Swartz <cswartz@gmail.com>

Conrad,

Have a look at our website--I added a statement in 2 places: FAQs (Patients & Families) and "The Many Advantages.." (did I tell you this already?)

The problem with what you wrote is that it doesn't address the primary subject of all ECT lawsuits: failure to warn the patient that permanent memory loss can occur.

Dick

[Quoted text hidden]

Conrad Swartz <cswartz@gmail.com> Wed, Nov 15, 2006 at 10:31 AM
To: Richard Abrams <richard.abrams@gmail.com>

Dick,
The way it works is that companies publish their warning statements in documents to MDs, and this relieves their liabilities and takes care of duty to warn patients. These warning statements for drug companies appear in the PDR.

We don't appear in the PDR. So, we need to publish our warning statements in our literature and online.

The statement on the website is not adequate warning to deflect lawsuits. It **disclaims the warning itself** with the sentence, " Rarely, a patient has claimed severe, permanent memory loss, but such claims have not been substantiated by objective memory testing." This statement says there are no permanent memory effects. This is the opposite of what we need to state.

In view of your complaint about not using the word "permanent" in my draft I have put it in in this re-draft:

Thelen006504

CONFIDENTIAL

Pl. 3_1

Plaintiff Exhibit 3, Pg 1

Gmail

Conrad Swartz <cswartz@gmail.com>

warning statement revisited

13 messages

Conrad Swartz <cswartz@gmail.com> Wed, Nov 15, 2006 at 9:05 AM
To: Richard Abrams <richard.abrams@gmail.com>

Dick,
Additional reflection produced these thoughts.

The goals of the warning statement we need to make are 1) prevent lawsuits, and 2) not alienate psychiatrists.

All warnings that are written are stated in the form that "this product can (or may) cause xxxx." We should conform to this. Cigarette companies can not use a statement such as "nothing in this advertisement should be regarded as a statement that cigarettes do not cause cancer." This is not a warning

Loss of memories is more accurate than memory loss, which smells of dementia. Loss of memories is subjective and does not reflect brain damage.

Some ECT methods, such as traditional bilateral ECT, are associated with more forgetting than others.

I think these are the essential elements to consider in our statement.

A draft is below for you to consider, edit, etc.:

Some patients may experience some loss of memories with ECT, particularly with traditional bilateral ECT. This is a subjective symptom that does not specifically reflect observable brain structure. Illness, anesthesia, medications, and postponement of treatment have their own risks, which are substantial.

Failure to Warn

Neuropsychopharmacology (2007) 32, 244–254

www.neuropsychopharmacology.org

The Cognitive Effects of Electroconvulsive Therapy in Community Settings

Harold A Sackeim*,[1,2,3], Joan Prudic[1,2], Rice Fuller[4], John Keilp[2,5], Philip W Lavori[6] and Mark Olfson[2,7]

cognitive deficits. Thus, adverse cognitive effects were detected 6 months following the acute treatment course. Cognitive outcomes varied across treatment facilities and differences in ECT technique largely accounted for these differences. Sine wave stimulation and BL electrode placement resulted in more severe and persistent deficits.
Neuropsychopharmacology (2007) **32,** 244–254. doi:10.1038/sj.npp.1301180; published online 23 August 2006

Keywords: electroconvulsive therapy; major depression; memory; cognitive side effects; amnesia

INTRODUCTION

Electroconvulsive therapy (ECT) is widely considered the most effective antidepressant treatment, with medication resistance its leading indication (American Psychiatric Association, 2001). However, critics contend that ECT invariably results in substantial and permanent memory loss (Breggin, 1986; Sterling, 2000), with some patients experiencing a dense retrograde amnesia extending back several years (Donahue, 2000; Sackeim, 2000). In contrast, some authorities have argued that, with the introduction of general anesthesia and more efficient electrical waveforms, ECT's adverse cognitive effects are short-lived, with no persistent effects on memory (Abrams, 2002; Fink, 2004).

Shortly following the ECT course, most patients manifest deficits in retaining newly learned information (anterograde amnesia) and recalling events that occurred in the weeks or months preceding the ECT course (retrograde amnesia) (Sackeim, 1992; Squire, 1986). Randomized-controlled trials have shown more severe short-term memory deficits with sine wave compared to brief pulse stimulation (Valentine *et al*, 1968; Weiner *et al*, 1986), bilateral (BL) compared to right unilateral (RUL) electrode placement (Lancaster *et al*, 1958; Sackeim *et al*, 1986; Sackeim *et al*, 1993; Sackeim *et al*, 2000), and higher electrical dosage (McCall *et al*, 2000; Ottosson, 1960; Sackeim *et al*, 1993). These adverse effects are reduced by the use of RUL ECT with brief or ultrabrief pulse stimulation and electrical dosage titrated to the needs of the individual patient (Sackeim, 2004b). Nonetheless, a minority of US practitioners still use sine wave stimulation, approximately half do not adjust dosage relative to the

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Received 9 March 2006; revised 17 May 2006; accepted 18 May 2006
Online publication: 12 July 2006 at http://www.acnp.org/citations/Npp071206060157/default.pdf

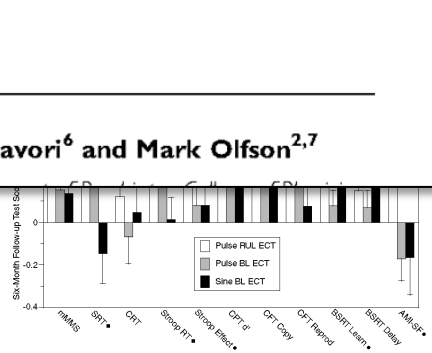


Figure 5 Scores on the 11 cognitive measures 6 months following the ECT course for patients treated with brief pulse right unilateral (RUL). ECT, brief pulse bilateral (BL). ECT, and sine wave BL ECT. Filled boxes indicate a significant effect of waveform in the ANCOVA (■ = $p<0.05$). Filled circles indicate a significant effect of electrode placement in the ANCOVA (● = $p<0.05$).

SRT, Stroop effect, learning phase of the BSRT, and AMI-SF scores. Greater amnesia for autobiographical events (AMI-SF scores) was significantly correlated with the number of ECT treatments received 6-months earlier (Table 5).

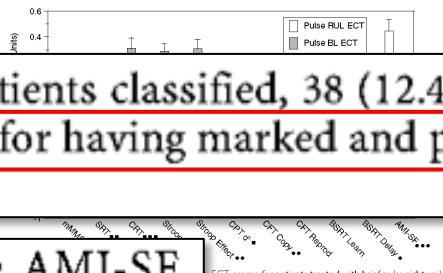
In the confirmatory analyses, there were significant linear relationships between the number of treatments administered and post-ECT AMI-SF scores for each of the three electrode placements. However, the slope of the decline in AMI-SF scores with increasing treatment number was substantially greater for BL ECT ($F(1,318) = 53.74$, slope $= -0.14$, SE $= 0.02$, $p<0.0001$) than RUL ($F(1,318) = 7.72$, slope $= -0.06$, SE $= 0.02$; $p = 0.005$) or bifrontal ECT ($F(1,318) = 8.01$, slope $= -0.09$, SE $= 0.03$, $p = 0.005$). At the 6-month time point, there continued to be a significant relationship between the number of BL ECT treatments and the extent of retrograde amnesia ($F(1,240) = 9.61$, df $= 1$, 240, slope $= -0.06$, SE $= 0.02$, $p = 0.002$), whereas there were no relationships with the RUL or BF placements. Thus, the magnitude of long-term retrograde amnesia linearly increased with longer courses of BL ECT, but was unrelated to the number of RUL or BF treatments administered.

Of the 306 patients classified, 38 (12.4%) patients met the *a priori* criteria for having marked and persistent retrograde

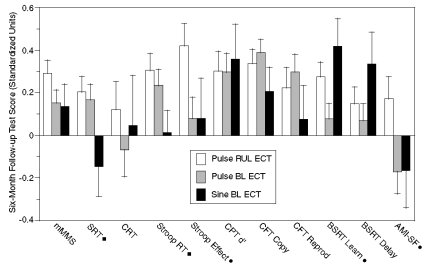
Sackeim Study (2007)

Thelen v. Somatics, LLC

Failure to Warn



Of the 306 patients classified, 38 (12.4%) patients met the *a priori* criteria for having marked and persistent retrograde amnesia on the AMI-SF.



ECT course for patients treated with brief pulse right unilateral (RUL). ECT, brief
significant effect of waveform in the ANCOVA (■ = $p < 0.05$; ■■ = $p < 0.01$;
■■■ = $p < 0.001$). Filled circles indicate a significant effect of electrode placement in the ANCOVA (● = $p < 0.05$; ●● = $p < 0.01$;
●●● = $p < 0.001$).

Figure 5 Scores on the 11 cognitive measures 6 months following the ECT course for patients treated with brief pulse right unilateral (RUL). ECT, brief pulse bilateral (BL). ECT, and sine wave BL ECT. Filled boxes indicate a significant effect of waveform in the ANCOVA (■ = $p < 0.05$). Filled circles indicate a significant effect of electrode placement in the ANCOVA (● = $p < 0.05$).

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Of the 306 patients classified, 38 (12.4%) patients met the *a priori* criteria for having marked and persistent retrograde

amnesia on the AMI-SF. In the logistic regression analysis, number of BL ECT treatments was the only treatment variable that significantly predicted membership in this subgroup, $\chi^2(1, 296) = 14.7$, $p = 0.0001$. There was also a $p < 0.02$, with a greater compared to men up.

ts were repeated now number of treatments independent variables. significant, with the e 6-month follow-up. short- and long-term ibutable to variation in d electrode placement

As seen in Tables 3 and 4, several of the covariates had powerful relations with cognitive performance at the post-ECT and 6-month follow-up time points. For every measure and at both time points, baseline scores had strong relationships with subsequent assessments, indicating strong reliability. For eight of the 11 post-ECT measures, cognitive performance was positively related to the number of days that elapsed from the end of ECT until the cognitive assessment. It is noteworthy that this effect did not occur with the AMI-SF, which assessed retrograde amnesia, the deficit thought to be most persistent. Two patient characteristics, age and the estimate of premorbid IQ, had frequent and strong relationships with cognitive outcomes, especially at the post-ECT assessment. In each instance, older patients and those with lower estimated intellectual function had more severe deficits. The gender differences, including the AMI-SF scores at the two time points, reflected greater deficits in women than men, and women were disproportionately represented in the group with marked and persistent impairment on the AMI-SF. Women have a substantially lower seizure threshold than men (Sackeim *et al*, 1987) and electrical dosage was not adjusted in most cases relative to the individual patient's seizure threshold. This pattern of gender differences might reflect the fact that electrical dosage was more markedly suprathreshold in women. Severity of depressive symptoms showed little relationship with the cognitive measures. At the post-ECT time point, none of the 11 measures were related to concurrent HRSD scores (Table 4). Findings were also negative for eight of the 11 measures at the 6-month follow-up. At this time point, lesser severity of depressive symptoms was associated with superior mMMS, delayed BSRT, and AMI-SF scores.

DISCUSSION

This was the first large-scale, prospective study of objective cognitive outcomes of patients treated with ECT. The seven hospitals differed in the magnitude of deficits at the post-ECT assessment (Figure 2), with significant differences in seven of the 11 cognitive measures. At the 6-month time point, differences among the hospitals persisted for the measure of global cognitive status (mMMS) and the primary outcome measure that assessed retrograde amnesia for autobiographical events (AMI-SF). In turn, these differences among the hospitals were largely attributable to differences in ECT technique. The use of sine wave stimulation and the BL electrode placement were both associated with greater short- and long-term deficits. In particular, sine wave stimulation had a marked effect on psychomotor response speed. Patients who received this form of stimulation were slowed at the 6-month assessment relative to patients treated with brief pulse stimulation on two of the three RT measures. In contrast, the long-term effects owing to electrode placement were expressed in the magnitude of retrograde amnesia. At both the short- and long-term time points, patients treated with BL ECT had greater amnesia for autobiographical events, and the extent of this amnesia was directly related to the number of BL ECT treatments received.

The demonstration of differences in the long-term cognitive outcomes as a function of hospital setting and treatment technique supports the conclusion that some forms of ECT have persistent long-term effects on cognitive performance. However, the findings do not indicate that the treatments with more benign outcomes are free of long-term effects. It is noteworthy, for example, that most cognitive parameters were substantially improved at 6-month follow-up relative to pre-ECT baseline, presumably because of the negative impact of the depressed state on baseline performance. Without evaluating a comparable group that did not receive ECT, it cannot be concluded, however, that the extent of improvement in any group returned to premorbid levels.

The finding that sine wave stimulation resulted in slowed RT could have reflected a speed/accuracy trade off, with patients receiving sine wave stimulation sacrificing response speed for accuracy. However, the sine wave and brief pulse groups did not differ in accuracy on any of the RT tasks where accuracy could be measured (CRT and Stroop). The fact that relative RT deficits were observed at the 6-month follow-up indicates a persistent change in the speed of information processing, motor initiation, or response execution. Randomized controlled studies have not found an advantage for sine wave stimulation with respect to efficacy (Andrade *et al*, 1988; Carney and Sheffield, 1974; Scott *et al*, 1992; Valentine *et al*, 1968; Weiner *et al*, 1986), and the American Psychiatric Association (2001) indicated that there is no justification for its continued use. The findings here raise the concern that this form of stimulation has deleterious long-term effects on elemental aspects of motor performance or information processing.

BL ECT results in broader and more severe short-term cognitive effects than RUL ECT, particularly with respect to retrograde amnesia. With respect to the AMI-SF scores, BL ECT resulted in greater retrograde amnesia than the other electrode placements and, even at the 6-month time point, this effect was linearly related to the number of BL treatments administered during the acute ECT course. The average decrement in AMI-SF scores in patients treated exclusively with BL ECT was 3.4 and 2.8 times the amount of forgetting seen in the healthy comparison groups at the post-ECT and 6-month time points, respectively, suggesting that the deficits were substantial. Furthermore, of a variety of treatment technique and patient characteristic variables, only receipt of BL treatment distinguished the group with marked and persistent retrograde amnesia. For decades, BL

Sackeim Study (2007)

preponderance of the evidence:

First: That Somatics placed the ECT device on the market;

Second: That at the time the ECT device left Somatics' possession, it was not accompanied by adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen;

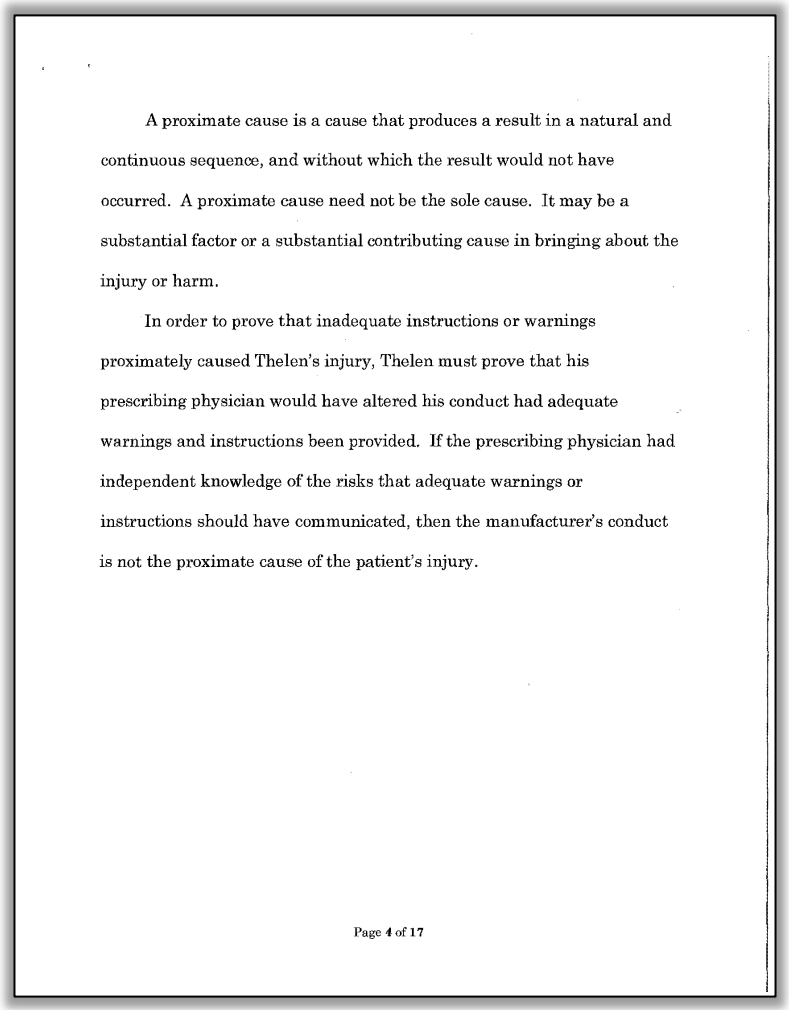
Third: That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen; and

Fourth: The nature and extent of that damage.

In the verdict form that I will explain in a moment, you will be asked to answer questions about these factual issues.

For purposes of this case, a product is not accompanied by adequate

Jury Instructions



A proximate cause is a cause that produces a result in a natural and continuous sequence, and without which the result would not have occurred. A proximate cause need not be the sole cause. It may be a substantial factor or a substantial contributing cause in bringing about the injury or harm.

In order to prove that inadequate instructions or warnings proximately caused Thelen's injury, Thelen must prove that his prescribing physician would have altered his conduct had adequate warnings and instructions been provided. If the prescribing physician had independent knowledge of the risks that adequate warnings or instructions should have communicated, then the manufacturer's conduct is not the proximate cause of the patient's injury.

Page 4 of 17

COURT'S INSTRUCTIONS TO THE JURY, Pg 4

Jury In

A proximate cause is a cause that produces a result in a natural and continuous sequence, and without which the result would not have occurred. A proximate cause need not be the sole cause. It may be a substantial factor or a substantial contributing cause in bringing about the injury or harm.

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Failure to Warn

Dr. Arun Sharma
ECT Prescribing Doctor

Q. Looking at this manual, sir, -- are -- is the very first page of that manual, descriptive of some side effects or some risks of ECT?
A. **Yes**

Q. Did you ever rely on this manual for your own assessments of patients, or is it just some -- well, let me just stop there. Do you rel -- did you ever rely on this manual for purposes of assessing a patient?
A. **Yes. Among other things. Yes.**

Q. Did you rely on this manual for purposes of talking to the patients about the certain risks and benefits of ECT?
A. **Yes.**

Failure to Warn

Dr. Arun Sharma
ECT Prescribing Doctor

Q. If Data showed that more than ten percent of people suffered permanent memory loss in the years prior to their treatment -- of memories from years prior to their treatment. Is that information you tell the patient?

A. Yes, what if the data is good, the source is good, I believe that I -- I -- of course share it with the patient.

Failure to Warn

Dr. Arun Sharma
ECT Prescribing Doctor

Q. When you say, “brain damage”, what is brain damage medically?

A. Brain damage means part of your brain is not working. That has been damaged by a procedure or activity of that sort, what means.

Failure to Warn

Dr. Arun Sharma
ECT Prescribing Doctor

A. I don't believe ECT causes brain damage. I don't believe that.

Q. And earlier, when you when you talked about common side facts and potential risks that you describe with – that you talk about with patients, ordinarily, brain damage is not something you typically discuss with patients, right?

A. No. We don't talk about brain damage.

Failure to Warn

Dr. Arun Sharma
ECT Prescribing Doctor

Q. And so if you received information that ECT treatment is associated with brain damage, you would convey that information to the patient, right?

A. Yes I would.

Q. And if after hearing that brain damage is risk of ECT treatment and the patient decided they don't want to undergo treatment, that's their decision, correct?

A. Yes. It's always their decision to do the treatment and not to do it.

Q. And if the patient decided they did not want to undergo ECT, you would not administer ECT, correct?

A. That is correct.

Q. Doctor, looking at the consent form we were looking at earlier … does the consent form indicate that brain damage is a risk of ECT treatment?

A. The informed consent does not say that.

A. Q. And earlier, when you talked about common side facts and potential risks that you describe with – that you talk about with patients ordinarily, brain damage is not something you typically discuss with patients, right?

A. No. We don't talk about brain damage.

Jury In

A proximate cause is a cause that produces a result in a natural and continuous sequence, and without which the result would not have occurred. A proximate cause need not be the sole cause. It may be a substantial factor or a substantial contributing cause in bringing about the injury or harm.

In order to prove that inadequate instructions or warnings proximately caused Thelen's injury, Thelen must prove that his prescribing physician would have altered his conduct had adequate warnings and instructions been provided. If the prescribing physician had independent knowledge of the risks that adequate warnings or instructions should have communicated, then the manufacturer's conduct is not the proximate cause of the patient's injury.

Prom royalty named

During the 1999 prom festivities, Drew Caskey and Karen Horst (center) were named king and queen. Also in the royalty were Adam Remm (far left), Chris Hoehne, Jeff Thelen (right) and Amanda Dreyer.

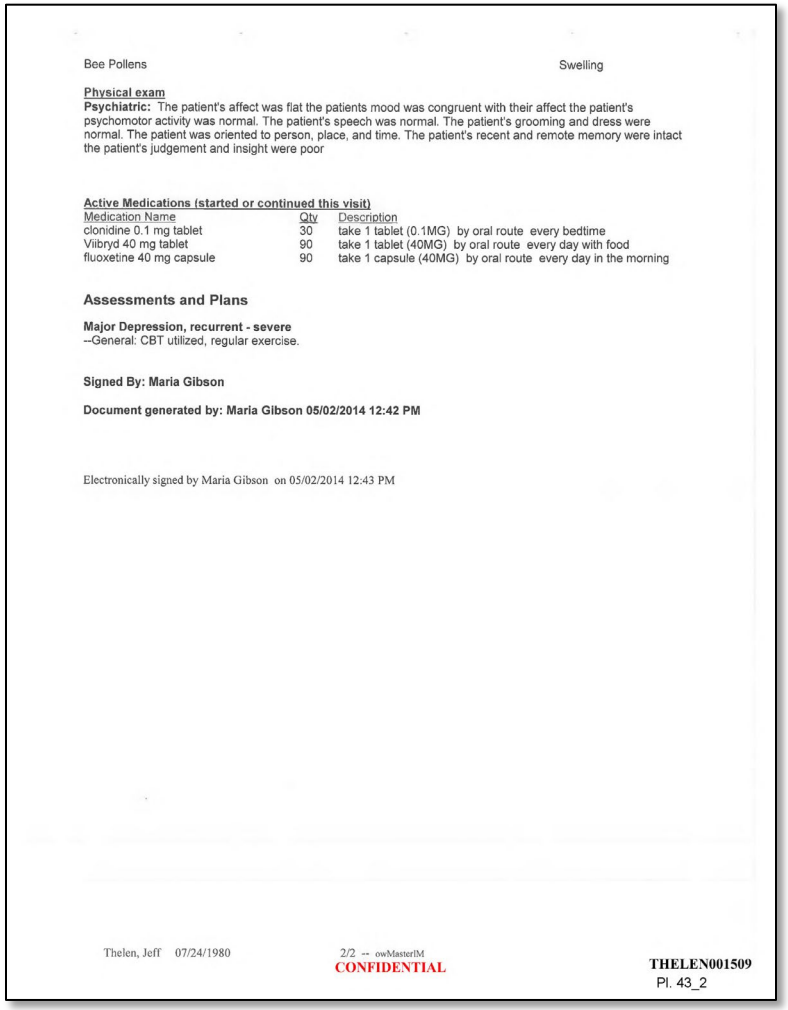
1999

JIM JAVORSKY/CORRESPONDENT

Preventive work

Oscar Flores (standing on stump) takes the end of a rope from Jeff Thelen as Chad Kingham looks on. The Nebraska Public Power District crew members recently were trimming tree branches from wires in Creighton to help prevent possible power outages from branches blowing into wires during storms.

Raymond Heller, M.D.



Bee Pollens Swelling

Physical exam
Psychiatric: The patient's affect was flat the patients mood was congruent with their affect the patient's psychomotor activity was normal. The patient's speech was normal. The patient's grooming and dress were normal. The patient was oriented to person, place, and time. The patient's recent and remote memory were intact the patient's judgement and insight were poor

Active Medications (started or continued this visit)

Medication Name	Qty	Description
clonidine 0.1 mg tablet	30	take 1 tablet (0.1MG) by oral route every bedtime
Viibryd 40 mg tablet	90	take 1 tablet (40MG) by oral route every day with food
fluoxetine 40 mg capsule	90	take 1 capsule (40MG) by oral route every day in the morning

Assessments and Plans

Major Depression, recurrent - severe
--General: CBT utilized, regular exercise.

Signed By: Maria Gibson

Document generated by: Maria Gibson 05/02/2014 12:42 PM

Electronically signed by Maria Gibson on 05/02/2014 12:43 PM

Thelen, Jeff 07/24/1980 2/2 -- owMasterIM CONFIDENTIAL THELEN001509 Pl. 43_2

Plaintiff Exhibit 43, Pg 2

Bee Pollens Swelling

Physical exam

Psychiatric: The patient's affect was flat the patients mood was congruent with their affect the patient's psychomotor activity was normal. The patient's speech was normal. The patient's grooming and dress were normal. The patient was oriented to person, place, and time. The patient's recent and remote memory were intact the patient's judgement and insight were poor

Active Medications (started or continued this visit)

Medication Name	Qty	Description
clonidine 0.1 mg tablet	30	take 1 tablet (0.1MG) by oral route every bedtime
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fluoxetine 40 mg capsule	90	take 1 capsule (40MG) by oral route every day in the morning

Assessments and Plans

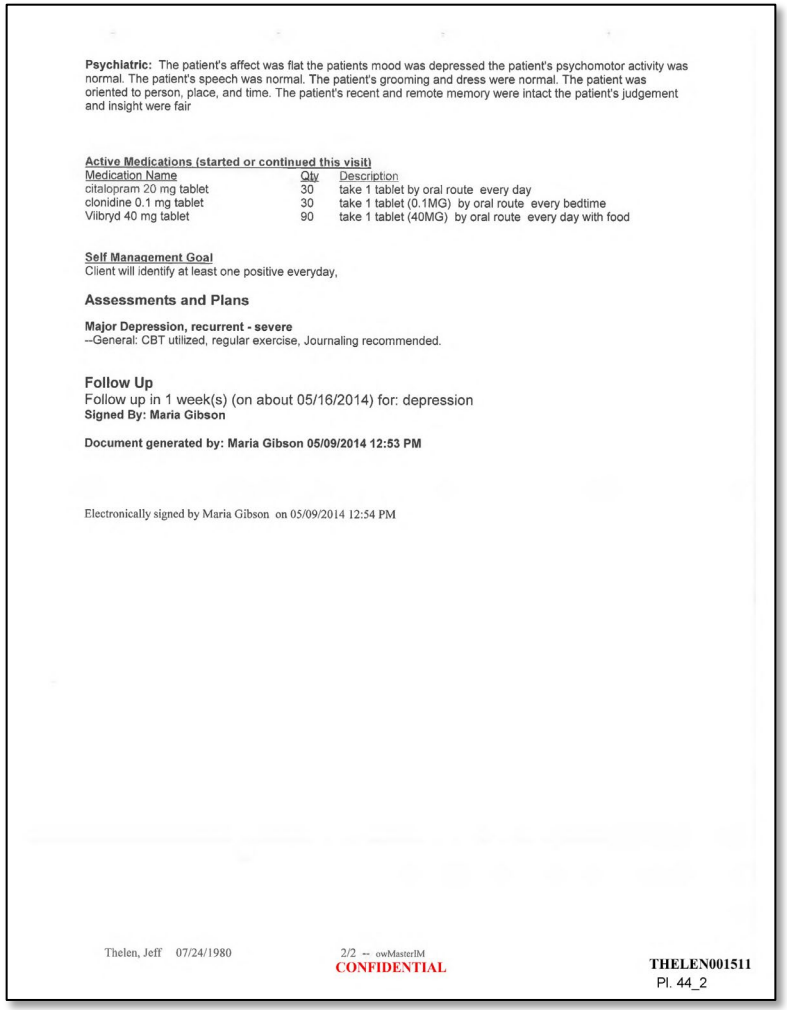
Major Depression, recurrent - severe
--General: CBT utilized, regular exercise.

Signed By: Maria Gibson

Document generated by: Maria Gibson 05/02/2014 12:42 PM

Electronically signed by Maria Gibson on 05/02/2014 12:43 PM

Raymond Heller, M.D.



Psychiatric: The patient's affect was flat the patients mood was depressed the patient's psychomotor activity was normal. The patient's speech was normal. The patient's grooming and dress were normal. The patient was oriented to person, place, and time. The patient's recent and remote memory were intact the patient's judgement and insight were fair

Active Medications (started or continued this visit)

Medication Name	Qty	Description
citalopram 20 mg tablet	30	take 1 tablet by oral route every day
clonidine 0.1 mg tablet	30	take 1 tablet (0.1MG) by oral route every bedtime
Viibryd 40 mg tablet	90	take 1 tablet (40MG) by oral route every day with food

Self Management Goal
Client will identify at least one positive everyday,

Assessments and Plans

Major Depression, recurrent - severe
--General: CBT utilized, regular exercise, Journaling recommended.

Follow Up
Follow up in 1 week(s) (on about 05/16/2014) for: depression
Signed By: Maria Gibson

Document generated by: Maria Gibson 05/09/2014 12:53 PM

Electronically signed by Maria Gibson on 05/09/2014 12:54 PM

Thelen, Jeff 07/24/1980 2/2 -- owMasterIM CONFIDENTIAL THELEN001511 Pl. 44_2

Plaintiff Exhibit 44, Pg 2

Psychiatric: The patient's affect was flat the patients mood was depressed the patient's psychomotor activity was normal. The patient's speech was normal. The patient's grooming and dress were normal. The patient was oriented to person, place, and time. The patient's recent and remote memory were intact the patient's judgement and insight were fair

Active Medications (started or continued this visit)

Medication Name	Qty	Description
citalopram 20 mg tablet	30	take 1 tablet by oral route every day
clonidine 0.1 mg tablet	30	take 1 tablet (0.1MG) by oral route every bedtime
Viibryd 40 mg tablet	90	take 1 tablet (40MG) by oral route every day with food

Self Management Goal

Client will identify at least one positive everyday,

Assessments and Plans

Major Depression, recurrent - severe

--General: CBT utilized, regular exercise, Journaling recommended.

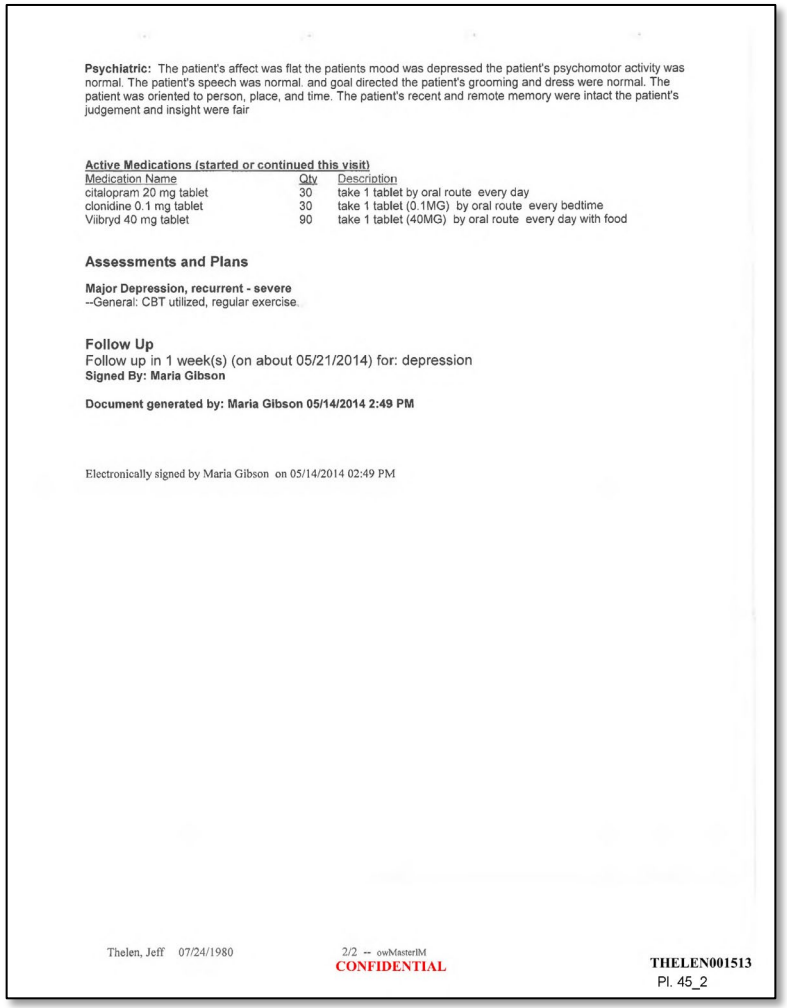
Follow Up

Follow up in 1 week(s) (on about 05/16/2014) for: depression

Signed By: Maria Gibson

Document generated by: Maria Gibson 05/09/2014 12:53 PM

Raymond Heller, M.D.



Psychiatric: The patient's affect was flat the patients mood was depressed the patient's psychomotor activity was normal. The patient's speech was normal. and goal directed the patient's grooming and dress were normal. The patient was oriented to person, place, and time. The patient's recent and remote memory were intact the patient's judgement and insight were fair

Active Medications (started or continued this visit)

Medication Name	Qty	Description
citalopram 20 mg tablet	30	take 1 tablet by oral route every day
clonidine 0.1 mg tablet	30	take 1 tablet (0.1MG) by oral route every bedtime
Viibryd 40 mg tablet	90	take 1 tablet (40MG) by oral route every day with food

Assessments and Plans

Major Depression, recurrent - severe
--General: CBT utilized, regular exercise.

Follow Up
Follow up in 1 week(s) (on about 05/21/2014) for: depression
Signed By: Maria Gibson

Document generated by: Maria Gibson 05/14/2014 2:49 PM

Electronically signed by Maria Gibson on 05/14/2014 02:49 PM

Thelen, Jeff 07/24/1980 2/2 -- owMasterIM CONFIDENTIAL THELEN001513 Pl. 45_2

Plaintiff Exhibit 45, Pg 2

Psychiatric: The patient's affect was flat the patients mood was depressed the patient's psychomotor activity was normal. The patient's speech was normal. and goal directed the patient's grooming and dress were normal. The patient was oriented to person, place, and time. The patient's recent and remote memory were intact the patient's judgement and insight were fair

Active Medications (started or continued this visit)

Medication Name	Qty	Description
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clonidine 0.1 mg tablet	30	take 1 tablet (0.1MG) by oral route every bedtime
Viibryd 40 mg tablet	90	take 1 tablet (40MG) by oral route every day with food

Assessments and Plans

Major Depression, recurrent - severe
--General: CBT utilized, regular exercise.

Follow Up

Follow up in 1 week(s) (on about 05/21/2014) for: depression
Signed By: Maria Gibson

Document generated by: Maria Gibson 05/14/2014 2:49 PM

Electronically signed by Maria Gibson on 05/14/2014 02:49 PM

After ECT - June 17, 2017

FAITH REGIONAL HEALTH SERVICES
2700 West Norfolk Ave. * Norfolk, NE, 68701

U.S. District Court
MDL Thelen v. Somatics
PLAINTIFF'S EXHIBIT
Exhibit Number: PL 121
Case Number: 8:18-cv-01724
JEFFREY THELEN v. SOMATICS, LLC
Date Identified:
Date Admitted:

ADMISSION DATE: 06/04/2017
SERVICE DATE: 06/17/2017
DATE OF BIRTH: 07/24/1980

HISTORY OF PRESENT ILLNESS
Mr. Thelen is a 36 year old gentleman seen today for followup. By nursing staff report, he reported depression and anxiety yesterday and had to be encouraged to get out of bed and attend groups. He did attend, sits on the periphery of groups, and is minimally participating. Last night, he reported some continued depression and anxiety but denied wanting to harm himself or others. He stated his visit with his mother went well. He denied any other concerns. He went to bed at 2300 and slept 7 hours. He refused breakfast, which has been his norm since his stay as he does not eat breakfast and will not in the hospital either, but he ate 100% of his other meals. He describes his appetite as all right. The patient had questions about memory loss from his ECT treatments. He stated he thought he may have had a 40 or more, but he cannot have the records released because it costs too much money, and he states it is difficult to fill out the paperwork. His best recollection is that he thinks he had weekly ECTs for approximately three quarters of the year. He states it has since been difficult for him to keep any job. When questions of his memory impairment come up, he is not accepted for positions he would really like to get. He states he has felt depressed for some time now. He states the Nardil was started 2 weeks ago, and he is hopeful it will help, but he isn't feeling much improvement yet. He denies any medication side effects. We talked about the possibility of TMS as an option for his treatment, and he was open to this. He required trazodone at 2100 last night to help him sleep as well as some hydroxyzine for anxiety. He denied any pain today but staff reported he was reporting significant pain in the morning, so they were surprised when he denied any pain today from his burn wound on his hand. He contracted for safety today but stated he still feels significantly depressed. He also had questions about whether or not he would be able to make his appointment with Dr. Sharma on June 22nd. I told him we could work with the Social Work team and based on his response to treatment to see about rescheduling that visit or if he would be able to make it. He continues to report his depression as "the same." His current medications were reviewed. He indicates he is tolerating his medicine without any issues.

REVIEW OF SYSTEMS
A full review of systems was conducted and with the exception noted previously was negative.

EXAMINATION
VITAL SIGNS: Temperature today 98.3, pulse 64, respirations 18, blood pressure 125/69.
MUSCULOSKELETAL: Gait and station intact. Strength and tone intact.
GENERAL APPEARANCE: Consistent with age. He is dressed casually with adequate grooming.
BEHAVIOR: Cooperative.
MOTOR: No psychomotor slowing or agitation present.
SPEECH: Normal rate and tone.

Patient Name: THELEN, JEFFREY	**MRN:** 131809
Dictating Provider: LANGENFELD, ROBERT J., MD	**Acct#:** 00112671748
Date of Admission:	

not in the hospital either, but he ate 100% of his other meals. He describes his appetite as all right. The patient had questions about memory loss from his ECT treatments. He stated he thought he may have had a 40 or more, but he cannot have the records released because it costs too much money, and he states it is difficult to fill out the paperwork. His best recollection is that he thinks he had weekly ECTs for approximately three quarters of the year. He states it has since been difficult for him to keep any job. When questions of his memory impairment come up, he is not accepted for positions he would really like to get. He states he has felt depressed for some time now. He states the Nardil was started 2 weeks ago, and he is hopeful it will help, but he isn't feeling much improvement yet. He denies any medication

Plaintiff Exhibit 121

After ECT - August 2017

Mark Hannappel, Ph.D.

- Evaluated Mr. Thelen in August 2017 and diagnosed Mr. Thelen with a neurocognitive disorder.
- June 2020 – begins treating Mr. Thelen on a weekly basis for Cognitive Behavioral Therapy

Q. Yeah, neurocognitive disorder implies that there's something neurologically impaired that causes the cognitive problems.

A. Yeah, neurocognitive disorder implies that there's something neurologically impaired that causes the cognitive problems.

Dr. Walter Duffy, June 28, 2018 EEG

U.S. District Court
Middle District of Florida
PLAINTIFF'S EXHIBIT
Exhibit Number: Pl. 159
Case Number: 8:20-cv-01724
JEFFREY THELEN v. SOMATICS, LLC
Date Identified:
Date Admitted:

Patient: 48000 - Jeffery P. Thelen
DOB: 07/24/1980
SSN: *******4193

Date: 06/28/2018 09:42
Provider: Duffy, Walter II, MD
Encounter: EEG – Interpretation

NOTES
Interpretation of EEG:

EEG recording completed on: 6.26.18

Comparison to previous EEG completed on date: n/a

Relevant Medications:
We do not have an updated list of his medications and Pt was unable to name his medications for me.

Indication: R41.89 Oth symptoms and signs w cognitive functions and awareness
Final Diagnosis: R41.844 Frontal lobe and executive function deficit

Impression:
Abnormal Study. Based on a comprehensive digital analysis of this task-specific EEG, there is electrophysiologic evidence of dysfunction in neuronal processing circuits responsible for attention networks.

There is evidence of significant changes in the following neuronal processing centers: attention, working memory. There is evidence of mild changes in the following neuronal processing centers: sensory.

4/7 neurocognitive biomarkers are positive as compared to previous study (4/7).

Medication and other treatment effect should be taken into consideration.

Recommendation:
Continue to analyze follow up studies to monitor neurophysiological changes to any treatment changes.

Summary:
Event related potential analysis of the individual networks and the comprehensive EEG include:

Sensory Processing:
Amplitude: 76.00
Absolute time: 97.00
Relative time: 92.74
Connectivity: 88.58

Final Diagnosis: R41.844 Frontal lobe and executive function deficit

Impression:
Abnormal Study. Based on a comprehensive digital analysis of this task-specific EEG, there is electrophysiologic evidence of dysfunction in neuronal processing circuits responsible for attention networks.

There is evidence of significant changes in the following neuronal processing centers: attention, working memory. There is evidence of mild changes in the following neuronal processing centers: sensory.

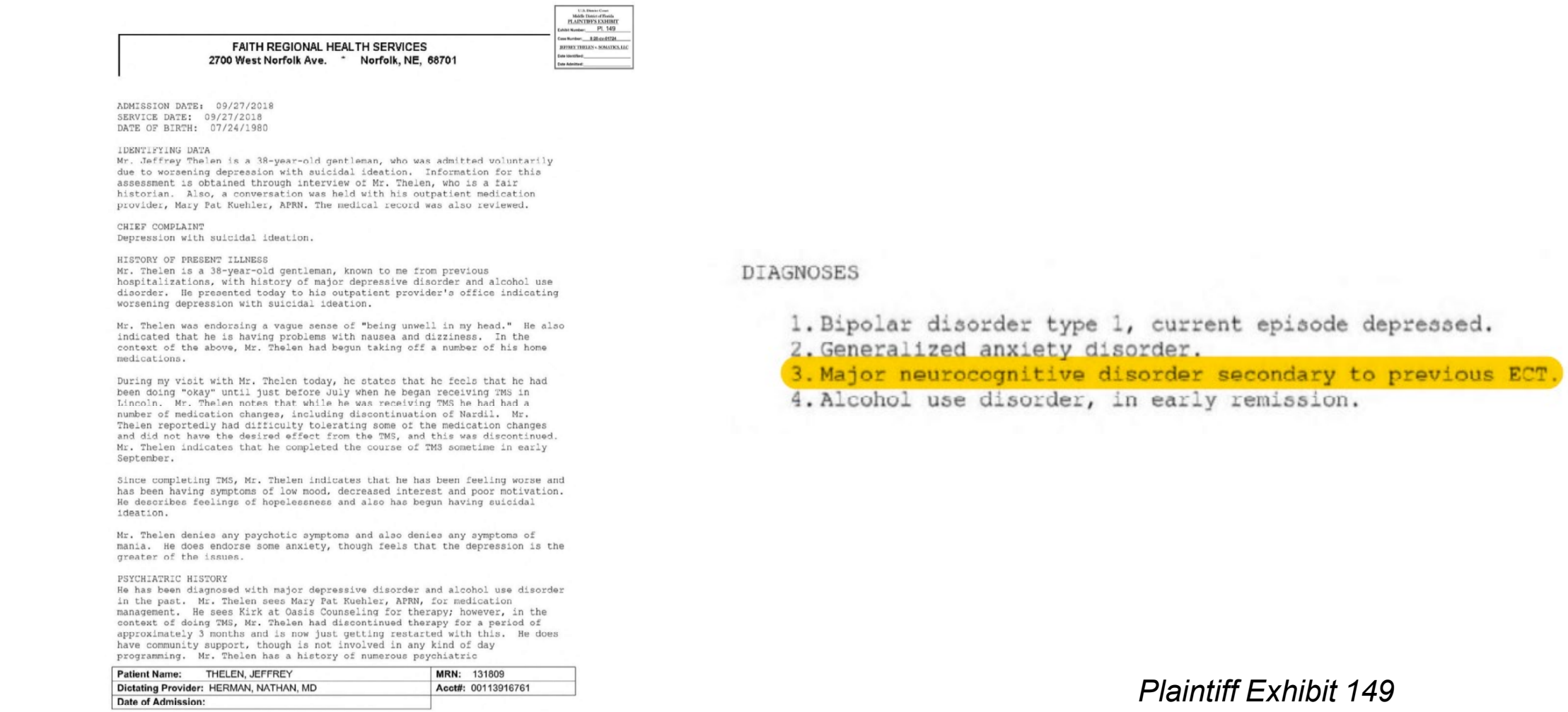
4/7 neurocognitive biomarkers are positive as compared to previous study (4/7).

Medication and other treatment effect should be taken into consideration.

Plaintiff Exhibit 159

Thelen v. Somatics, LLC

Dr. Nathan Herman, September 27, 2018



FAITH REGIONAL HEALTH SERVICES
2700 West Norfolk Ave. * Norfolk, NE, 68701

U.S. District Court
Middle District of Florida
PLAINTIFF'S EXHIBIT
Exhibit Number: Pl. 149
Case Number: 8:20-cv-01724
JEFFREY THELEN v. SOMATICS, LLC
Date Identified:
Date Admitted:

ADMISSION DATE: 09/27/2018
SERVICE DATE: 09/27/2018
DATE OF BIRTH: 07/24/1980

IDENTIFYING DATA
Mr. Jeffrey Thelen is a 38-year-old gentleman, who was admitted voluntarily due to worsening depression with suicidal ideation. Information for this assessment is obtained through interview of Mr. Thelen, who is a fair historian. Also, a conversation was held with his outpatient medication provider, Mary Pat Kuehler, APRN. The medical record was also reviewed.

CHIEF COMPLAINT
Depression with suicidal ideation.

HISTORY OF PRESENT ILLNESS
Mr. Thelen is a 38-year-old gentleman, known to me from previous hospitalizations, with history of major depressive disorder and alcohol use disorder. He presented today to his outpatient provider's office indicating worsening depression with suicidal ideation.

Mr. Thelen was endorsing a vague sense of "being unwell in my head." He also indicated that he is having problems with nausea and dizziness. In the context of the above, Mr. Thelen had begun taking off a number of his home medications.

During my visit with Mr. Thelen today, he states that he feels that he had been doing "okay" until just before July when he began receiving TMS in Lincoln. Mr. Thelen notes that while he was receiving TMS he had had a number of medication changes, including discontinuation of Nardil. Mr. Thelen reportedly had difficulty tolerating some of the medication changes and did not have the desired effect from the TMS, and this was discontinued. Mr. Thelen indicates that he completed the course of TMS sometime in early September.

Since completing TMS, Mr. Thelen indicates that he has been feeling worse and has been having symptoms of low mood, decreased interest and poor motivation. He describes feelings of hopelessness and also has begun having suicidal ideation.

Mr. Thelen denies any psychotic symptoms and also denies any symptoms of mania. He does endorse some anxiety, though feels that the depression is the greater of the issues.

PSYCHIATRIC HISTORY
He has been diagnosed with major depressive disorder and alcohol use disorder in the past. Mr. Thelen sees Mary Pat Kuehler, APRN, for medication management. He sees Kirk at Oasis Counseling for therapy; however, in the context of doing TMS, Mr. Thelen had discontinued therapy for a period of approximately 3 months and is now just getting restarted with this. He does have community support, though is not involved in any kind of day programming. Mr. Thelen has a history of numerous psychiatric

Patient Name: THELEN, JEFFREY	**MRN:** 131809
Dictating Provider: HERMAN, NATHAN, MD	**Acct#:** 00113916761
Date of Admission:	

DIAGNOSES

1. Bipolar disorder type 1, current episode depressed.
2. Generalized anxiety disorder.
3. Major neurocognitive disorder secondary to previous ECT.
4. Alcohol use disorder, in early remission.

Plaintiff Exhibit 149

Thelen v. Somatics, LLC

Bennet Omalu, M.D.

Bennet Omalu, M.D.

"My opinion, conclusion and diagnosis is that Mr. Jeff Thelen suffered brain damage, yes, sir."

Q. And did your differential diagnosis [identify] what the cause of the brain damage injury is?

A. Yes, sir, I performed a [differential diagnosis] of the data before me, and I made a determination … of a substantial and significant factor that caused his brain damage.

Q. And what was that?

A. Multiple electroconvulsive therapies, yes sir.

"Just like in [other] types of dementia, the brain damage here is on the cellular level, sub cellular level … So a C.T. scan and an MRI translational study cannot identify such microscopic findings."

Second: That at the time the ECT device left Somatics' possession, it was not accompanied by adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen;

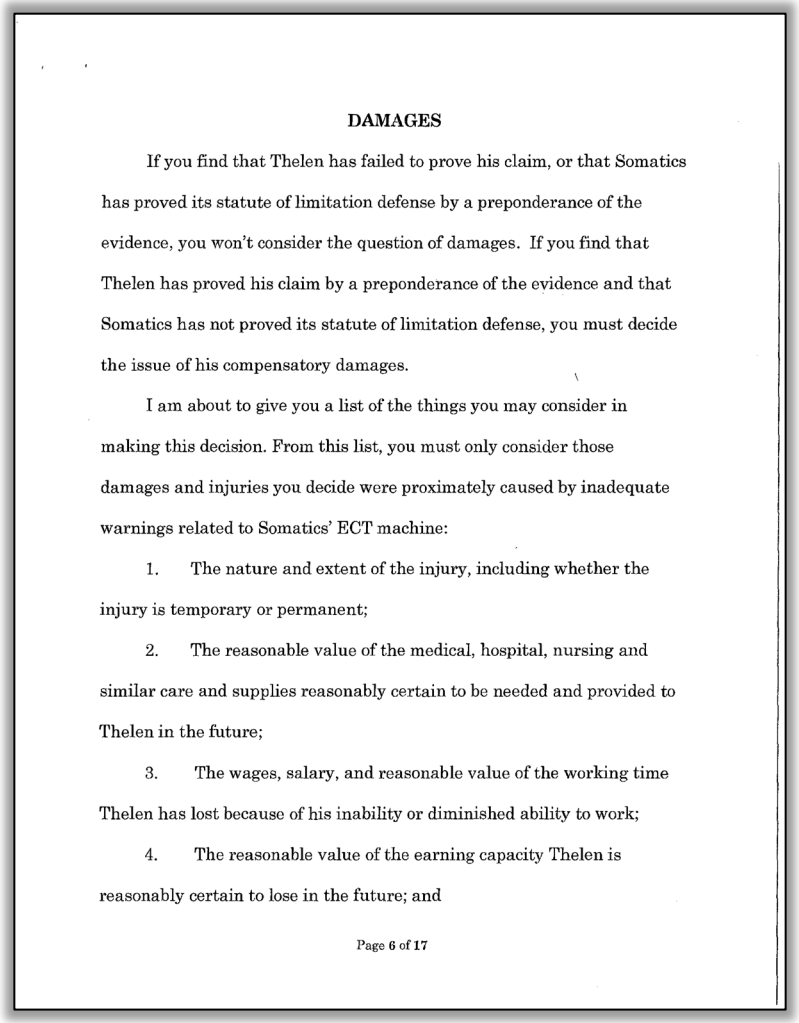
Third: That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen; and

Fourth: The nature and extent of that damage.

In the verdict form that I will explain in a moment, you will be asked to answer questions about these factual issues.

For purposes of this case, a product is not accompanied by adequate instructions or warnings if reasonable instructions or warnings regarding foreseeable risks of harm are not provided to prescribing physicians who

Jury Instructions



DAMAGES

If you find that Thelen has failed to prove his claim, or that Somatics has proved its statute of limitation defense by a preponderance of the evidence, you won't consider the question of damages. If you find that Thelen has proved his claim by a preponderance of the evidence and that Somatics has not proved its statute of limitation defense, you must decide the issue of his compensatory damages.

I am about to give you a list of the things you may consider in making this decision. From this list, you must only consider those damages and injuries you decide were proximately caused by inadequate warnings related to Somatics' ECT machine:

1. The nature and extent of the injury, including whether the injury is temporary or permanent;

2. The reasonable value of the medical, hospital, nursing and similar care and supplies reasonably certain to be needed and provided to Thelen in the future;

3. The wages, salary, and reasonable value of the working time Thelen has lost because of his inability or diminished ability to work;

4. The reasonable value of the earning capacity Thelen is reasonably certain to lose in the future; and

Page 6 of 17

COURT'S INSTRUCTIONS TO THE JURY, Pg 6

Jury

Somatics has not proved its statute of limitation defense, you must decide the issue of his compensatory damages.

I am about to give you a list of the things you may consider in making this decision. From this list, you must only consider those damages and injuries you decide were proximately caused by inadequate warnings related to Somatics' ECT machine:

1. The nature and extent of the injury, including whether the injury is temporary or permanent;

2. The reasonable value of the medical, hospital, nursing and similar care and supplies reasonably certain to be needed and provided to Thelen in the future;

3. The wages, salary, and reasonable value of the working time Thelen has lost because of his inability or diminished ability to work;

Jury

making this decision. From this list, you must only consider those damages and injuries you decide were proximately caused by inadequate warnings related to Somatics' ECT machine:

1. The nature and extent of the injury, including whether the injury is temporary or permanent;

2. The reasonable value of the medical, hospital, nursing and similar care and supplies reasonably certain to be needed and provided to Thelen in the future;

3. The wages, salary, and reasonable value of the working time Thelen has lost because of his inability or diminished ability to work;

4. The reasonable value of the earning capacity Thelen is reasonably certain to lose in the future; and

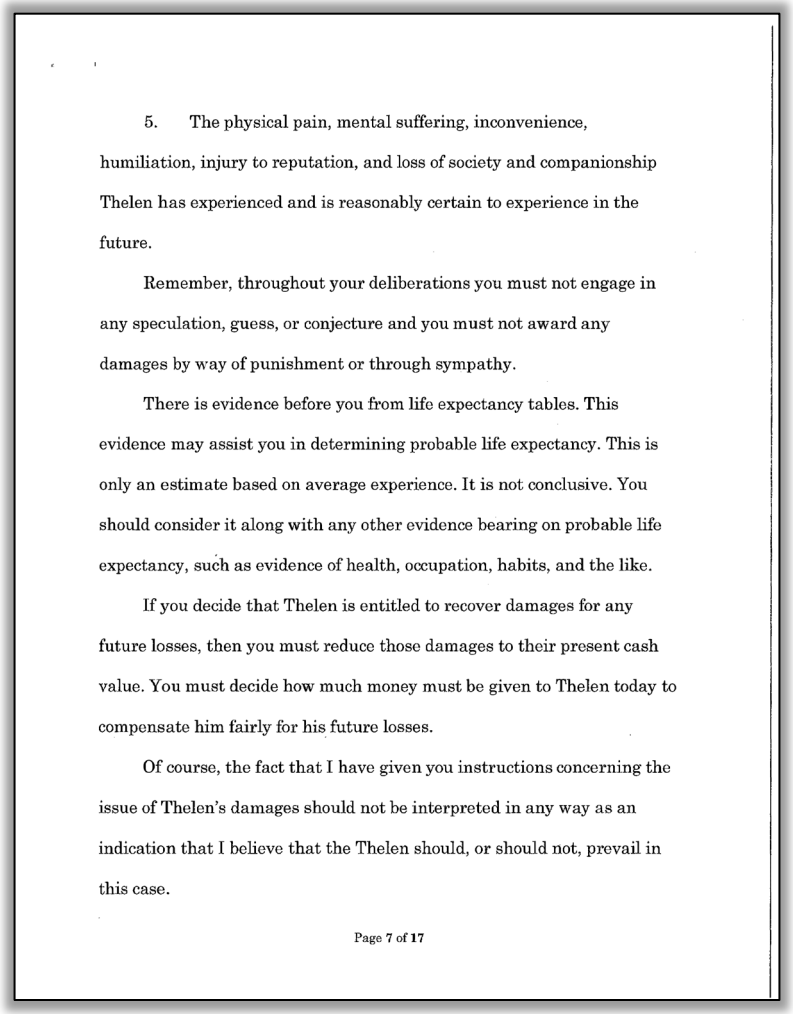
2. The reasonable value of the medical, hospital, nursing and similar care and supplies reasonably certain to be needed and provided to Thelen in the future;

3. The wages, salary, and reasonable value of the working time Thelen has lost because of his inability or diminished ability to work;

4. The reasonable value of the earning capacity Thelen is reasonably certain to lose in the future; and

COURT'S INSTRUCTIONS TO THE JURY, Pg 6

Jury Instructions



5. The physical pain, mental suffering, inconvenience, humiliation, injury to reputation, and loss of society and companionship Thelen has experienced and is reasonably certain to experience in the future.

Remember, throughout your deliberations you must not engage in any speculation, guess, or conjecture and you must not award any damages by way of punishment or through sympathy.

There is evidence before you from life expectancy tables. This evidence may assist you in determining probable life expectancy. This is only an estimate based on average experience. It is not conclusive. You should consider it along with any other evidence bearing on probable life expectancy, such as evidence of health, occupation, habits, and the like.

If you decide that Thelen is entitled to recover damages for any future losses, then you must reduce those damages to their present cash value. You must decide how much money must be given to Thelen today to compensate him fairly for his future losses.

Of course, the fact that I have given you instructions concerning the issue of Thelen's damages should not be interpreted in any way as an indication that I believe that the Thelen should, or should not, prevail in this case.

Page 7 of 17

COURT'S INSTRUCTIONS TO THE JURY, Pg 7

Jury Instructions

5. The physical pain, mental suffering, inconvenience, humiliation, injury to reputation, and loss of society and companionship Thelen has experienced and is reasonably certain to experience in the future.

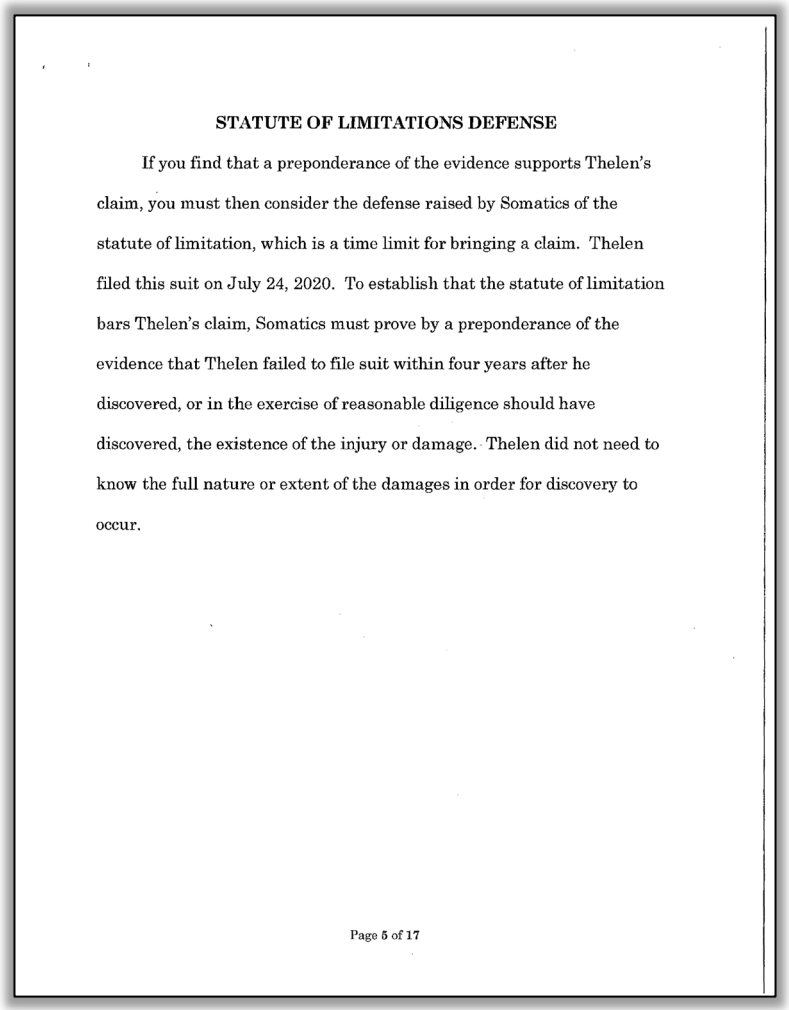
Remember, throughout your deliberations you must not engage in any speculation, guess, or conjecture and you must not award any damages by way of punishment or through sympathy.

There is evidence before you from life expectancy tables. This

Defenses

Defendants Purported Defenses

Jury Instructions



STATUTE OF LIMITATIONS DEFENSE

If you find that a preponderance of the evidence supports Thelen's claim, you must then consider the defense raised by Somatics of the statute of limitation, which is a time limit for bringing a claim. Thelen filed this suit on July 24, 2020. To establish that the statute of limitation bars Thelen's claim, Somatics must prove by a preponderance of the evidence that Thelen failed to file suit within four years after he discovered, or in the exercise of reasonable diligence should have discovered, the existence of the injury or damage. Thelen did not need to know the full nature or extent of the damages in order for discovery to occur.

Page 5 of 17

COURT'S INSTRUCTIONS TO THE JURY, Pg 5

Jury Ins

STATUTE OF LIMITATIONS DEFENSE

If you find that a preponderance of the evidence supports Thelen's claim, you must then consider the defense raised by Somatics of the statute of limitation, which is a time limit for bringing a claim. Thelen filed this suit on July 24, 2020. To establish that the statute of limitation bars Thelen's claim, Somatics must prove by a preponderance of the evidence that Thelen failed to file suit within four years after he discovered, or in the exercise of reasonable diligence should have discovered, the existence of the injury or damage. Thelen did not need to know the full nature or extent of the damages in order for discovery to occur.

s

Statute of Limitations

- Jeffrey Thelen filed suit against Somatics on July 24, 2020.
- Jeffrey Thelen's last ECT treatment was on July 25, 2016
- Although Jeffrey Thelen complained of memory loss throughout his ECT treatment, he was told his memories would return after completing ECT.

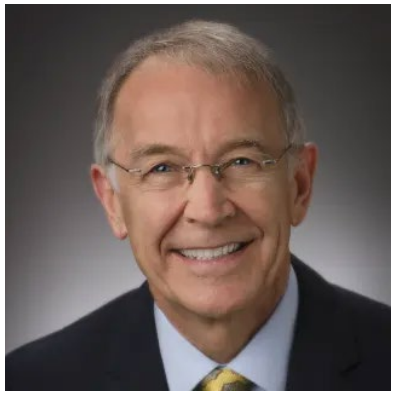
Robert M. Bilder, Ph.D.

- Someone else – who did not testify in this case – did the neuropsychological testing for him in Nebraska
- Has been paid tens of thousands of dollars by Somatics in ECT cases and has always concluded ECT did not cause the injury
- Never met or communicated with Jeffrey Thelen
- Could not make any behavioral observations of Mr. Thelen
- Agreed that Dr. Hannappel's findings from his neuropsychological testing were consistent with a major neurocognitive disorder.

Q. Dr. Bilder, is Mr. Thelen faking his memory complaints?

A. I don't think so.

Dr. Coffey



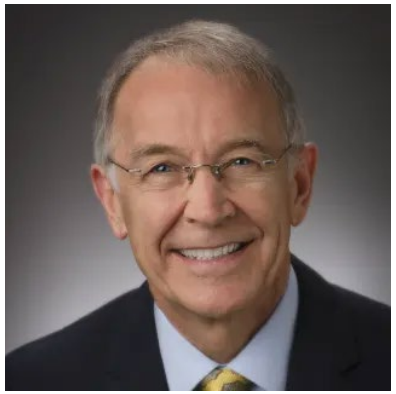
Have any of Mr. Thelen's doctors diagnosed him with neurocognitive disorder?

Yes ☐ No ☒

FALSE

Testimony 06/07/2023

Dr. Coffey



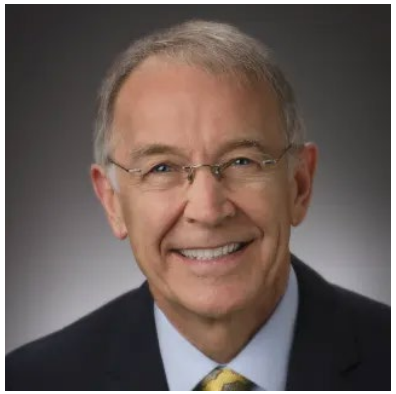
Have any of Mr. Thelen's doctors diagnosed him with major neurocognitive disorder secondary to ECT?

Yes ☐ No ☒

FALSE

Testimony 06/07/2023

Dr. Coffey



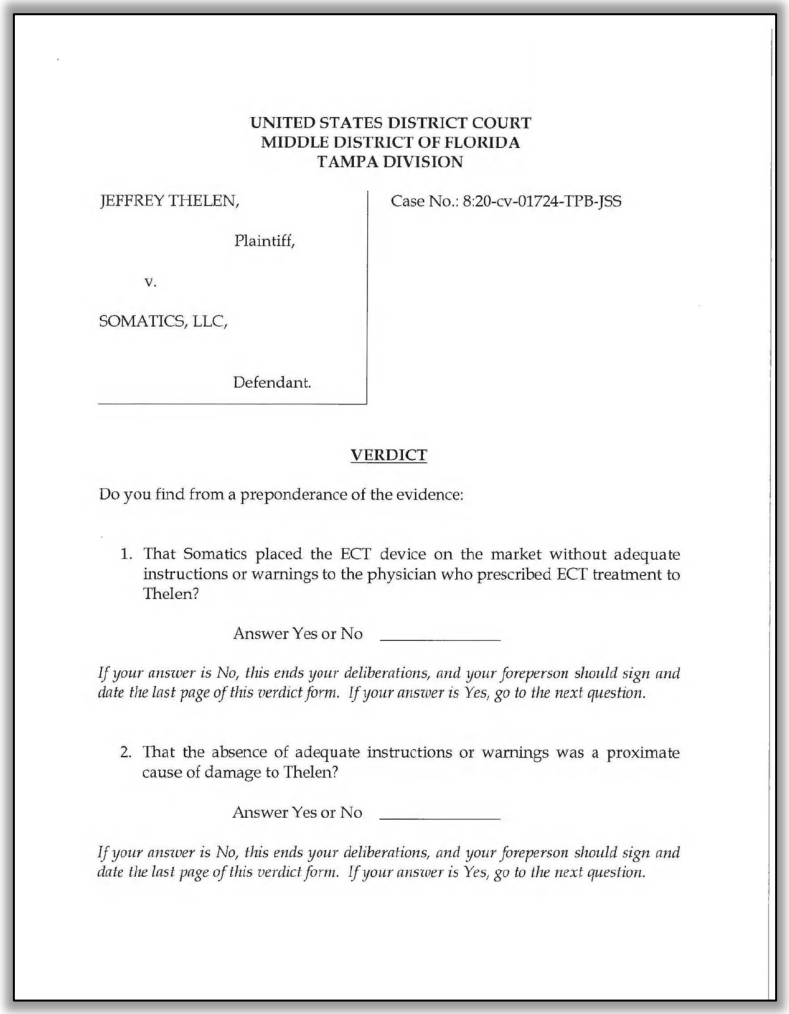
No indication in the medical records that Mr. Thelen's memory wipeout was clearly articulated to any of his providers?

Yes ☒ No ☐

FALSE

Testimony 06/07/2023

Verdict Form



UNITED STATES DISTRICT COURT
MIDDLE DISTRICT OF FLORIDA
TAMPA DIVISION

JEFFREY THELEN,

Plaintiff,

v.

SOMATICS, LLC,

Defendant.

Case No.: 8:20-cv-01724-TPB-JSS

VERDICT

Do you find from a preponderance of the evidence:

1. That Somatics placed the ECT device on the market without adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen?

Answer Yes or No _____

If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

2. That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen?

Answer Yes or No _____

If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

Verdict Form, Pg 1

Verd

SOMATICS, LLC,

Defendant.

VERDICT

Do you find from a preponderance of the evidence:

1. That Somatics placed the ECT device on the market without adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen?

Answer Yes or No _____

If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

2. That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen?

Answer Yes or No _____

SOMATICS, LLC,

Defendant.

VERDICT

Do you find from a preponderance of the evidence:

1. That Somatics placed the ECT device on the market without adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen?

Answer Yes or No YES

If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

2. That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen?

Answer Yes or No _____

1. That Somatics placed the ECT device on the market without adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen?

Answer Yes or No **YES**

If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

2. That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen?

Answer Yes or No _____

If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

Verd

1. That Somatics placed the ECT device on the market without adequate instructions or warnings to the physician who prescribed ECT treatment to Thelen?

Answer Yes or No **YES**

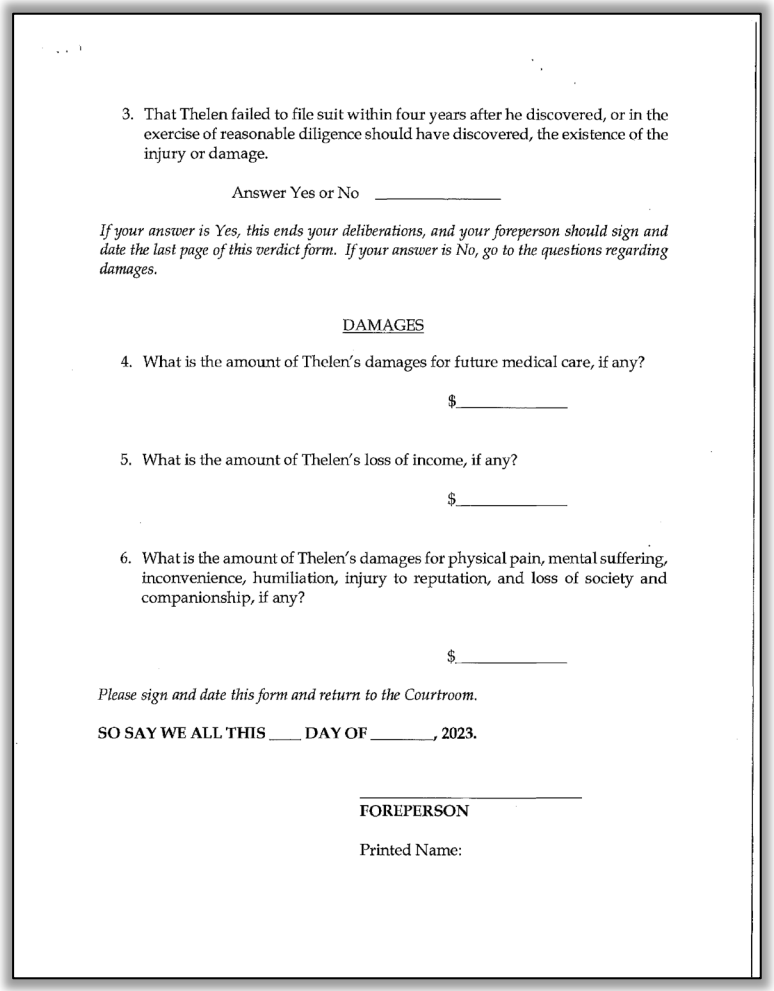
If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

2. That the absence of adequate instructions or warnings was a proximate cause of damage to Thelen?

Answer Yes or No **YES**

If your answer is No, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is Yes, go to the next question.

Verdict Form



3. That Thelen failed to file suit within four years after he discovered, or in the exercise of reasonable diligence should have discovered, the existence of the injury or damage.

Answer Yes or No _____

If your answer is Yes, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is No, go to the questions regarding damages.

DAMAGES

4. What is the amount of Thelen's damages for future medical care, if any?

$_____

5. What is the amount of Thelen's loss of income, if any?

$_____

6. What is the amount of Thelen's damages for physical pain, mental suffering, inconvenience, humiliation, injury to reputation, and loss of society and companionship, if any?

$_____

Please sign and date this form and return to the Courtroom.

SO SAY WE ALL THIS ____ DAY OF _____, 2023.

_____

FOREPERSON

Printed Name:

Thelen v. Somatics, LLC

Verdict Form

3. That Thelen failed to file suit within four years after he discovered, or in the exercise of reasonable diligence should have discovered, the existence of the injury or damage.

Answer Yes or No _____

If your answer is Yes, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is No, go to the questions regarding damages.

DAMAGES

4. What is the amount of Thelen's damages for future medical care, if any?

$_____

Verdict Form

3. That Thelen failed to file suit within four years after he discovered, or in the exercise of reasonable diligence should have discovered, the existence of the injury or damage.

Answer Yes or No **NO**

If your answer is Yes, this ends your deliberations, and your foreperson should sign and date the last page of this verdict form. If your answer is No, go to the questions regarding damages.

DAMAGES

4. What is the amount of Thelen's damages for future medical care, if any?

$_____

DAMAGES

4. What is the amount of Thelen's damages for future medical care, if any?

$_____

5. What is the amount of Thelen's loss of income, if any?

$_____

6. What is the amount of Thelen's damages for physical pain, mental suffering, inconvenience, humiliation, injury to reputation, and loss of society and companionship, if any?

$_____

Please sign and date this form and return to the Courtroom.

SO SAY WE ALL THIS ____ DAY OF _____, 2023.

_____

FOREPERSON

Verd

DAMAGES

4. What is the amount of Thelen's damages for future medical care, if any?

$949,007 - $2,085,635

5. What is the amount of Thelen's loss of income, if any?

$375,245

6. What is the amount of Thelen's damages for physical pain, mental suffering, inconvenience, humiliation, injury to reputation, and loss of society and companionship, if any?

$_____

Please sign and date this form and return to the Courtroom.

SO SAY WE ALL THIS ____ DAY OF _____, 2023.

_____

FOREPERSON

Failure t

dysfunction (especially stomach or intestines), or parasitosis. Sensory distortions or hallucinations can accompany these delusions. Middle-aged or elderly patients with psychotic melancholia often have constipation or other gastrointestinal complaints (Parker et al., 1991).

Psychotic melancholia is one of several common types of psychotic depression, most of which are treatable with ECT, but for some types ECT is not suitable (Swartz and Shorter, 2007). Some other types of psychotic depression are bipolar mixed depression, deteriorative psychotic depression, catatonic psychotic depression, psychotic-equivalent demented depression, tardive psychotic depression, drug-induced psychotic depression, and coarse brain disease psychotic depression. ECT should help patients with these types of depression but should be only a last resort in tardive and coarse brain disease psychotic depressions.

Several reports from Columbia University claim that medication-resistant major depression responds poorly to ECT (Prudic et al., 1990, 1996). Indeed, the outcome of their studies was markedly worse than expected. Response – which is a substantially lower outcome than remission – was obtained in only 35% to 65% of their patients with an average ECT course of 10 treatments, a long course. Their claim of medication resistance predisposing to poor ECT outcome contrasts markedly with other studies – some quite large – reporting that medication-resistant patients respond very well to ECT (e.g., Pluijms et al., 2002). In the DeCarolis study (Avery and Lubrano, 1979) 85% of 110 patients who failed to respond to one month of imipramine achieved remission with 8 to 10 ECT treatments. The "endogenous depression" that these patients had presumably corresponds to melancholia or simultaneous melancholia and catatonia. The inability of an acute course of ECT to treat comorbid anxiety disorders such as PTSD may explain the poor results at Columbia.

The Columbia study patients had long suffered with psychiatric conditions with averages of 15 to 20 years since illness onset. Still, the reports do not state that anxiety disorders were accounted for, disqualified, or even examined for. The simple explanation for the bad outcome is that the Columbia patients had comorbid

ECT and
modulation Therapies

Bennet Omalu, M.D.

Bennet Omalu, M.D.

"The standard of practice in medicine is you never question a patient. If a patient tells you he's having a headache, you don't tell him, no, you're not having a headache. These are called symptoms as specified by the patient."

Memory Loss

- Research shows the frequency of permanent memory loss beyond the treatment period (i.e., retrograde, autobiographical memory) is between 12 and 55% after six months.

- If memory does not come back after six months, it is considered permanent.