

THE USE OF ELECTRICAL CURRENT AS AN ANESTHETIC AGENT

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In previous experiments it was demonstrated that the successful production of electrical anesthesia, or electronarcosis, could be accomplished by applying some of the techniques of modern anesthesiology. The complications of cyanosis, bradycardia, cardiac irregularities and severe muscular contractions, which had been encountered by earlier investigators of electronarcosis, were eliminated or modified by these techniques. For example, endotracheal intubation abolished the respiratory difficulties, atropine sulfate given before the production of electronarcosis prevented cardiac irregularities, and curare decreased muscular contractions to the extent that operations were performed upon animals narcotized only by the electrical current (1). Before electronarcosis could be used as an anesthetic agent in humans it was necessary that the deleterious effects of electrical currents, applied for prolonged periods, be studied in animals. After these effects were found to be minimal, the method of electronarcosis was then applied to human beings.

INVESTIGATIONS IN THE DOG

Twenty-five dogs* were narcotized for a period of three hours with a continuous alternating current of 50–100 milliamperes, 700 cycles per second, and approximately 15 volts, through electrodes placed on the head. Atropine sulfate, 0.006 mg. per kilogram of body weight, was given intravenously fifteen minutes before the production of electronarcosis. The animals were given *d*-tubocurarine chloride intravenously to assist in the introduction of an endotracheal tube, and artificial respiration with room air was given throughout the experiment by means of a solenoid apparatus. Blood samples were removed from the cephalic vein at the intervals of fifteen minutes, one, two, and three hours from the beginning of the experiment. These samples were analyzed for glucose, urea nitrogen, serum sodium, serum potassium, serum chloride, and serum calcium. A few blood samples

* Each animal had been given an injection of distemper vaccine on entry to the experimental laboratory.

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were taken after the animals had recovered for an hour following the cessation of electronarcosis. At the end of three hours of electronarcosis the current was turned off, the animal was allowed to recover and was returned to its cage and placed on a normal regimen. Seventy-two hours later the dog was killed with a large dose of intravenous thiopental sodium. The brain and spinal cord were removed and placed in formalin solution. The brains were sectioned coronally, and complete coronal sections were prepared for microscopic study, including sections through the prefrontal, hypothalamic, thalamic and occipital regions of the cerebral hemispheres. Cross sections of midbrain, pons,

TABLE I
CHANGE IN BLOOD GLUCOSE LEVELS IN DOGS DURING PASSAGE OF CURRENT
(50 MILLIAMPERES) FOR THREE HOURS

Dog Number	Control	Blood Glucose (Mg. %)			
		15 Min.	60 Min.	120 Min.	3 Hrs.
1	75	—	99	110	104
2	93	108	104	201	182
3	74	78	90	87	—
4	73	80	—	75	78
5	72	76	95	—	—
7	—	95	83	131	107
8	56	80	85	110	117
9	61	558	64	—	88
10	80	94	107	137	130
11	67	77	83	122	92
12	68	83	87	79	66
13	69	82	82	87	76
14	55	107	90	74	251
15	76	79	91	87	86
16	79	97	94	113	139
17	68	73	70	73	66
18	71	86	78	74	93
19	74	88	137	117	107
20	84	131	104	100	91
21	63	88	159	174	193

medulla and cerebellum were also included. Segments of spinal cord generally included upper, middle and lower cervical cord, middle and lower thoracic cord segments, and at least one section of lumbar and sacral cord. The sections were stained with hematoxylin eosin, Weil, and Nissl stains.

Blood pressure studies were made on additional dogs with an arterial cannula connected to a mercury manometer which recorded on a kymograph.

A vacuum tube voltmeter was used to measure the amount of voltage present in various portions of the skin and musculature of several animals in an effort to determine the danger to the surgeon.

RESULTS

Blood Chemistry.—The serum potassium, calcium, chloride and sodium all failed to show any significant change during the passage of the electronarcotic current for three hours.

A marked hyperglycemia occurred (table 1) which was statistically significant according to the *t* test for correlated pairs.† Although the average changes showed a steady rise during the passage of the current, there was a general tendency for each dog to give a maximum response at the 15-minute interval and fall thereafter. Other dogs reached a maximum at one hour and others at two and three hours. In only 3 dogs did a three-hour level become less than the initial one.

TABLE 2
CHANGES IN BLOOD UREA NITROGEN IN DOGS AFTER PASSAGE OF CURRENT
(50 MILLIAMPERES) FOR THREE HOURS

Dog Number	Control	Urea Nitrogen (mg. %) after 3 Hours
1	15	12
3	15	8
5	11	18
6	13	20
7	15	13
8	9	5
9	5	14
10	12	24
11	17	16
12	14	17
13	15	16
14	9	16
15	13	13
16	13	12
17	14	13
19	19	26
20	30	26
21	16	19

When the current was turned off, a definite decrease occurred within one hour.

Although it appears from table 2 that there is little change in the blood urea nitrogen during the three-hour period of electronarcosis, there is actually an increase which is significant when analyzed statistically. This increase in the blood urea nitrogen probably indicates decrease in kidney function during the passage of the current.

Neuropathology.—Of the brains and spinal cords of 24 dogs examined grossly and microscopically by one of us (F.Y.T.) for evidences of neurological damage by the electrical current, 17 were entirely free from distemper and inflammatory changes. In these 17 animals no morphological changes resulting from the electrical current were

† A. E. Treloar, Professor Biostatistics Division, University of Minnesota, made the statistical studies.

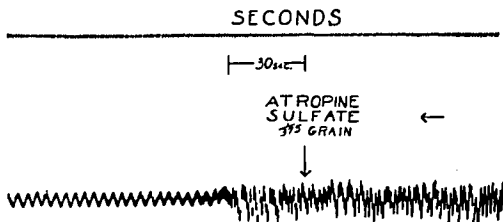


FIG. 1. This kymograph tracing shows marked cardiac irregularities from a current of 50 milliamperes, which were abolished by intravenous atropine sulfate. (Read from right to left.)

found. Seven dogs were excluded from the study because 4 showed severe inflammatory changes suggestive of distemper and 3 other dogs were excluded on the basis of poor processing and staining techniques.

Blood Pressure Studies.—Figure 1 demonstrates the remarkable protective effect which atropine sulfate has upon the heart of a dog receiving electrical current through the head. Before the intravenous administration of atropine sulfate, marked cardiac irregularities and bradycardia were present as shown in the mean arterial blood pressure tracing. Thirty seconds after the administration of the atropine sulfate the heart rhythm and rate became normal. It was suggested by Dr. Van Bergen that gallamine triethiodide (Flaxedil®) might be used in a dual role, providing muscular relaxation and blocking the vagal influence upon the heart. Five dogs were given electro-narcosis (50 milliamperes, 700 cycles per second, 15–20 volts), and the effect of intravenous gallamine triethiodide upon the mean arterial blood pressure studied. In all 5 dogs irregularities of the heart seen in tracings were decreased or abolished. Figure 2 is a typical tracing of

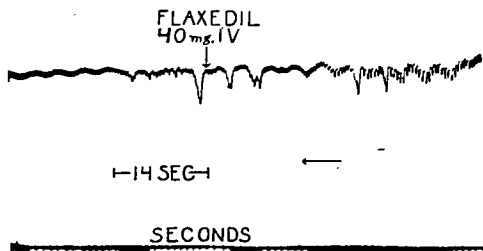


FIG. 2. The dog was receiving a current of 50 milliamperes, and the kymograph tracing shows bradycardia and cardiac irregularities. Flaxedil® abolished these in about 14 seconds. (Read from right to left.)

the response observed in the 5 dogs. It appeared that gallamine triethiodide was as effective as atropine sulfate in decreasing the cardiac irregularities resulting from passage of the electronarcotic current.

The mean arterial blood pressure rose abruptly as soon as the electronarcotic current passed through the animal (fig. 3). Initially the blood pressure rose from 30 to 40 mm. of Hg and then over a period of a half hour decreased slowly about 10 mm. of Hg, but always remained above the original level. If the current was turned off, the blood pressure dropped to the original level within 10 to 15 seconds.

Measurement of Electrical Potentials in Animals.—A vacuum tube voltmeter of great accuracy and sensitivity was used to measure the electrical potential in various portions of the dog's body. This was done in an attempt to assess the danger to the person operating upon the animals. Electronarcotic currents of 50 milliamperes and 120 milliamperes at a frequency of 700 cycles per second were used. At 50 milliamperes the voltage across the electrodes on the dog's head was

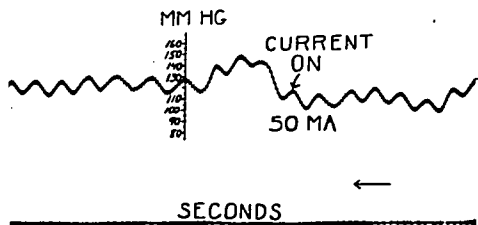


FIG. 3. On passing the electronarcotic current through the dog, the blood pressure increased abruptly by 30-40 mm. of Hg from which it gradually decreased to a level which remained higher than the initial blood pressure. (Read from right to left.)

6-5 volts. Other voltages were: head to muscle of leg, 2.8 volts; ground to muscle, 3.2 volts; ground to wound on chest, 3.4 volts; ground to mouth, 3.2 volts. When the milliamperage was increased to 120 the electrical potentials were: across electrodes on head, 18 volts; head to wound on leg, 11 volts; head to mouth, 7.5 volts; ground to wound in chest, 6.5 volts, both probes in abdomen, .007 volt; bladder to ground, 10.5 volts; right side of chest to left side of chest, .0045 volt. From these figures it is apparent that low potentials were present.

DISCUSSION OF THE INVESTIGATION IN ANIMALS

The hyperglycemia which occurred during the passage of the electrical current was expected, since Gellhorn and Kessler (2) observed that electrical convulsions produced a hyperglycemia in the rat. After observing a hypoglycemia in the adrenalectomized rat and no change in

the adrenalectomized-vagotomized animal, they concluded that the hyperglycemia was due to excitement of both the sympathetico-adrenal and vago-insulin systems. Stern, Askonas, and Cullen (3) found a hyperglycemia occurring in 28 out of 34 patients receiving electric shock therapy; and Elliott, Rivers, Elliott, and Platt (4) also reported a rapid rise in the blood glucose in electric shock convulsions. It is interesting to note from our experiments that on the average the intensity of hyperglycemia is increased as the electrical current is prolonged.

No changes owing to the prolonged passage of the electrical current through the brain were found. That electrical current may produce irreversible changes in nervous tissue is well known. Alpers and Madow (5) reported on a patient who died three hours after being given an electronarcotic treatment. The patient was given 180 milliamperes and 100 to 200 volts in three bursts of 10 seconds each over a period of one minute. Pathological examination showed hemorrhages in the caudate nuclei, the medial nucleus of the thalamus, the posterior hypothalamic nuclei, the supraoptic nucleus, the cerebellum, and the choroid plexus of the lateral ventricles. There also were smaller hemorrhages in the cerebral cortex, the corpus callosum, the optic tract, and the pineal gland. The amperage and voltage given to this patient were much greater than were used in any of our experiments, but the duration of current passage was less. In examining the evidence for nervous tissue damage following electric shock treatments, Karliner (6) stated that there is insufficient evidence at hand to indicate that in human beings structural brain changes are directly referable to electric shock treatments. It is more likely that physiologic and possibly chemical, rather than structural, changes occur. Supporting this viewpoint in a critical review, Alpers (7) believed that there was ample evidence that electric shock does not cause deaths through brain damage, but that death occurs because of cardiovascular disease.

Our experiments indicated, as a corollary to Alpers views, that when adequate oxygenation is provided and the heart is protected from noxious reflexes resulting from the passage of electrical current, no brain damage in dogs results even from prolonged passage of the narcotizing electrical current. As a result of our findings we were encouraged to try the application of electrical current to man in an attempt to produce general anesthesia.

INVESTIGATIONS IN MAN

The patients who were given electronarcosis were in a mental hospital. All had received courses of electroshock therapy previously. Patients having a normal medical history and physical examination and having the ability to communicate their impressions and feelings were chosen. Control electrocardiograms and electroencephalograms †

† The electroencephalograms were interpreted by Z. R. Miller, M.D.

were taken on all patients to determine their normalcy before the electronarcosis. Fluids were started through an 18 gauge needle before electronarcosis was begun to assure ready access to the circulatory system. Electrodes from the electronarcosis machine were placed over the temples after applying electrode paste to the skin. Cuffed endotracheal tubes were inserted in all patients following the initiation of the electrical anesthesia, and oxygen was administered by means of an anesthesia machine. Electrocardiograms and electroencephalograms were obtained as soon as the electronarcotic current was turned off. Blood pressure recordings were taken with a cuff and sphygmomanometer. The heart was auscultated with a stethoscope throughout the procedure.

CASE REPORTS

Case No. 1.—The control blood pressure was 170/70 mm. of Hg, and the pulse was 90 beats per minute. The electroencephalogram before electronarcosis showed a background pattern of 8 to 10 per second moderate voltage activity with some random 5 to 6-per-second moderate voltage slowing. The electrocardiogram was normal. Atropine sulfate, 1/75 gr., was given intravenously fifteen minutes before electronarcosis. At 11:33 a.m. a current of 135 milliamperes, 700 cycles per second, 34 volts was begun. Blood pressure was approximately 170 mm. of Hg, systolic. A severe tonic contraction developed with breath-holding, moving of arms and legs, and closing of eyes. The patient became cyanotic. Flaxedil, 120 mg., was injected intravenously. The patient began to breathe after about 60 seconds and at 11:36 a.m. the endotracheal tube was inserted without difficulty. At 11:36 a.m. a tachycardia was present, and there were slight muscular twitchings of the legs and arms. Extrasystoles were heard on auscultation of the heart. The patient's eyelids opened but the gaze was staring. The pupillary reflex to light was present. The intravenous fluids infiltrated and another needle was inserted without noticeable response from the patient. At 11:44 a.m. Tensilon®, 10 mg., was given intravenously and thirty seconds later the current was turned off. The total time of electronarcosis was twelve minutes. For approximately 5-10 seconds the patient did not respond to questioning, but after this short time he answered correctly concerning time, place, and person. Deep breathing at a rate of 28 respirations per minute was present for ten minutes. The pulse was 140 beats per minute. The electrocardiogram showed numerous ventricular extrasystoles which disappeared in two minutes. An electroencephalogram was taken at 11:46 a.m. and showed no appreciable change. Nystagmus was present for ten minutes. It was apparent, on questioning him, that he was unaware of the events occurring during the passage of the current, the insertion of the endotracheal tube, or the restarting of intravenous fluids. He stated that he did not mind the experience and that he would be happy to return the following week.

Case No. 2.—Control blood pressure was 140/60 mm. of Hg, pulse, 68, and electrocardiogram, normal. Electroencephalographic tracings showed a 9-per-second background alpha pattern without abnormalities. Atropine sulfate, 1/75 gr., was given intravenously fifteen minutes before electronarcosis, and Flaxedil, 120 mg., was given one minute before. At 10:50 a.m. a current of 135 milli-

amperes, 700 cycles per second, was turned on. The blood pressure suddenly increased to 280/70 mm. of Hg, and the pulse increased to 140 beats per minute, although it remained regular. By 10:54 a.m. the blood pressure had decreased to 220/124 mm. of Hg. At 10:57 a.m. the pulse was regular and had a rate of 125 beats per minute. At 11:00 a.m. the blood pressure was 192/110 and by 11:04 a.m. the blood pressure had decreased to 176/110. Waves of "goose pimples" appeared and disappeared over his skin. At 11:07 a.m. the current decreased to 105 milliamperes, voltage 29, probably because of an increased resistance of the skin for which the machine was unable to compensate. At 11:08 a.m. he seemed to be awake although he made no movement. Three minutes later he was asked to nod his head, and he responded correctly. Tensilon®, 10 mg., was then given intravenously and at 11:14 a.m. the current was turned off. Eight minutes after the current ceased the blood pressure was normal, 140/70 mm. of Hg. On questioning the patient, it was apparent that he had no memory of the experience until the time when the current decreased from 135 milliamperes to 105 milliamperes. From that time on, the experience was unpleasant; and he described it as "burning in Hell, no pain, just the idea." The electrocardiogram was normal. The electroencephalogram showed a mild amount of 4-per-second moderate to high voltage slowing and some bursts of spike-like activity which were difficult to evaluate because of muscle and movement artifacts.

Case No. 3.—Control blood pressure was 144/86 mm. of Hg., pulse 82. Atropine sulfate, 1/75 gr., was given fifteen minutes before electronarcosis. The electrocardiogram was normal and the electroencephalogram had a background pattern of 9 to 10 per second. Moderate voltage activity was present, obscured, however, by constant muscle artifacts. Flaxedil, 180 mg., was given one minute before electronarcosis. At 10:55 a.m. a current of 135 milliamperes, 700 cycles per second was turned on. The blood pressure promptly rose to 240/140 mm. of Hg and the pulse was 140 beats per minute. By 11:02 a.m. the blood pressure had decreased to 210/118 mm. of Hg. Stimuli in the form of commands and needle pricks were given without effect. Respiration became adequate by 11:04 a.m., at which time the pupils were constricted and centrally placed. At 11:18 a.m. running movements of the legs were present. The current was increased to 150 milliamperes for 3-10 second periods and the running movements ceased. By 11:23 a.m. the blood pressure was 200/100 mm. of Hg, and the pulse was 160 beats per minute. The current was turned off at 11:27 a.m. and the patient awoke immediately. At 11:31 a.m. the blood pressure was 150/70. The electrocardiogram was normal. The electroencephalogram showed a pronounced slow record, 4 to 6-per-second moderate to high voltage slow waves from all areas, though maximal from the right motor and parietal leads. After fourteen minutes had elapsed, random and paroxysmal 5-per-second moderate to high voltage waves were still noted and were most prominent from the frontal and central leads. After thorough questioning of the patient concerning the stimuli which we had given him, we concluded that the patient had no memory of any event occurring during the electronarcotic period.

Case No. 4.—Control blood pressure was 130/80 mm. of Hg. Atropine sulfate, 1/75 gr., was given fifteen minutes before the electronarcosis, and Flaxedil, 90 mg., was given intravenously one minute before. At 11:38 a.m. a current of

135 milliamperes, 700 cycles per second, was begun. The blood pressure rose to 214/130 mm. of Hg, and the pulse increased to 198 beats per minute and was regular. By 11:46 a.m. the blood pressure was 190/100 mm. of Hg, and the pulse was 168 beats per minute. At 12:04 p.m. the patient was instructed to shake his head and he carried out the command. He also moved his right arm on command. At 12:10 p.m. the current was turned off and the patient awoke immediately. The blood pressure was 130/80, and the pulse was 148 beats per minute at 12:13 p.m. On careful questioning, it seemed probable that he was at least partially aware of the stimuli and commands given him during the passage of the current. Furthermore, the experience had been an unpleasant one for him.

Case No. 5.—Control blood pressure was 150/84 mm. of Hg and the pulse was 72 beats per minute. The electrocardiogram was normal; an electroencephalogram was not done. Atropine sulfate, 1/150 gr., was given intravenously. At 10:36 a.m. hexamethonium bromide, 25 mg., was given intravenously, in an attempt to prevent high elevations of blood pressure occurring during the passage of the current. An additional 25 mg. of hexamethonium bromide was given at 10:48 a.m. The blood pressure at 10:57 a.m. was 140/90 mm. of Hg. At 10:59 a.m. a current of 150 milliamperes, 700 cycles per second, 37.5 volts, was turned on. A minute later the blood pressure had risen to 160/110 mm. of Hg. Two minutes later the blood pressure was 200/120 and the pulse was 132 beats per minute. An additional 25 mg. of hexamethonium bromide was given intravenously and the blood pressure began to fall until at 11:10 a.m. the systolic pressure was 100. On auscultation of the chest, heart tones could not be heard. A vasopressor agent was given intravenously and the blood pressure rose rapidly. At 11:14 a.m. the current was turned off, and Tensilon® was given intravenously. At 11:30 a.m. the blood pressure was 120/60 mm. of Hg, but a great deal of difficulty was experienced in maintaining an adequate airway because of relaxed pharyngeal muscles. No cyanosis occurred, however. When the patient was questioned about the experience he said only a few words and then became inarticulate, although he was conscious. At each question he would smile faintly but give no other response. For the next 48 hours his blood pressure remained at about 90 mm. of Hg, systolic, and he continued to be unresponsive to questioning. For several months following the experience he remained in an unresponsive and suspicious state; but as a result of intensive psychotherapy he has now returned to the state existing before electronarcosis.

DISCUSSION

It might at first appear that currents with smaller milliamperages, since they would cause smaller blood pressure elevations, would permit a less dangerous electronarcosis. And this would be true, except that there is a critical level below which the state of electronarcosis will not occur, and that critical level is within the range of currents causing serious elevations of blood pressure. Thompson *et al.* (8) have tried a high current initially (160–250 milliamperes) for thirty seconds and then decreasing the current to lower levels (60 to 75 milliamperes) and

then gradually increasing it for a period of seven minutes. In their experience the blood pressure rose to levels of 170 to 200 systolic and 100-120 diastolic which was followed by a precipitous drop, sometimes to low levels, on decreasing the current. This technique, then, also does not accomplish the desired physiologic state.

It was hoped that a ganglionic blocking agent would prevent the sharp rise in blood pressure occurring during the passage of the electrical current, but it failed to accomplish this. The precipitous fall, following the use of hexamethonium during the current passage, and the prolonged hypotension after the cessation of the current, were so unlike the usual actions of hexamethonium bromide that we would hesitate to use it again in electronarcotized patients. Perhaps an agent controllable from minute to minute, such as Arfonad[®], could be used to greater advantage.

Because of the seriousness of the cardiovascular complications in man (hypertension, cardiac irregularities, and tachycardia), we have discontinued our electronarcosis investigations in human beings. This is not to say, however, that we believe electrical anesthesia to be impossible of achievement, for in our work we have actually investigated only one more frequency (700 cycles per second) in man. It may well be that a higher frequency, or a different wave form, or a different combination of the variables of current, potential and frequency, will produce a satisfactory narcosis without the frightening side effects. In order to investigate the problem logically, though, the work should be accomplished with man as the subject, since there is a marked difference in the reaction to electrical currents among different species. Human volunteers who would be able to report accurately their experience during the narcosis would be the most valuable subjects.

SUMMARY

Twenty-five dogs were given electronarcosis for three hours with a continuous alternating current of 50 milliamperes, 700 cycles per second, 16-20 volts. Pathological studies of the brains and spinal cords revealed no evidence of neurological damage owing to the continuous passage of the current. A marked hyperglycemia which tended to increase during the electronarcosis was present. The blood urea nitrogen rose significantly during the three-hour period of electronarcosis. No changes occurred in the serum calcium, sodium, chloride and potassium.

Electrical anesthesia was administered to 5 patients by means of an alternating current of 135 milliamperes, 700 cycles per second. Marked hypertension and tachycardias developed during the passage of the current.

REFERENCES

1. Knutson, R. C.: Experiments in Electronarcosis; Preliminary Study, *ANESTHESIOLOGY* 15: 551 (Sept.) 1954.
2. Gellhorn, E., and Kessler, M.: Effect of Electrically Induced Convulsions on Vago-insulin and Sympatho-adrenal System, *Proc. Soc. Exper. Biol. & Med.* 46: 64 (Jan.) 1941.
3. Stern, K., Askonas, B. H., and Cullen, A. M.: Influence of Electroconvulsive Treatment on Blood Sugar, *Am. J. Psychiat.* 105: 585 (Feb.) 1949.
4. Elliott, K. A. C., Rivers, T. C., Elliott, F. H., and Platt, B.: Effects of Insulin, Metrazol, and Electrical Shock on Blood Pyruvate, Lactate, and Glucose, *J. Lab. & Clin. Med.* 26: 1928 (Sept.) 1941.
5. Alpers, B. J., and Madow, L.: Changes in Brain Associated with Electronarcosis, *Arch. Neurol. & Psychiat.* 60: 366 (Oct.) 1948.
6. Karliner, W.: Neurologic Signs and Complications of Electric Shock Treatments: Review, *J. Nerv. & Ment. Dis.* 107: 1 (Jan.) 1948.
7. Alpers, B. J.: Brain Changes Associated with Electric Shock Treatment: Critical Review, *Journal Lancet* 66: 363 (Nov.) 1946.
8. Thompson, G. N., McGinnis, J. E., Van Harreveld, A., Wiersma, C. A. G., and Tietz, E. B.: Electronarcosis: Clinical Comparison with Electroshock, *War Med.* 6: 158 (Sept.) 1944.