tive to $\dot{V}_{o_2}$ just prior to induction limits applicability of the data and is subject to misinterpretation. Theoretically, the least variance and greatest predictability would be associated with expressions which relate observed $\dot{V}_{o_2}$ to *observed* basal $\dot{V}_{o_2}$ of the individual patient. This approach, however, is inconvenient, limted in applicability, and probably unnecessarily "pure," since expressions relative to *predicted* basal $\dot{V}_{o_2}$ offer a similar degree of predictability in the present study. Assuming then that there might be agreement to express $\dot{V}_{o_2}$ during anesthesia relative to the predicted basal $\dot{V}_{o_2}$ of the individual patient, it would be necessary to define a single set of predicted values. The predicted values used in the present study (table 1) are based on the final standards suggested by Boothby and associates[5] and are in general use. Another set of predicted values which has been used[11] provides values about 4 per cent higher than those generally used.

Values for arterial P_{o_2} were more variable and less predictable than values for arterial P_{co_2}. This is believed to be based on a spread of pulmonary ventilation perfusion ratios such that the shuntlike effect (underventilated-perfused alveoli) was exaggerated and more variable than the dead spacelike effect (overventilated-perfused alveoli). The presence of sizable alveolar-arterial P_{o_2} gradients in anesthetized man has been observed previously, and ascribed in part to the prior pattern of ventilation and its relation to collapse of alveolar air spaces.[12]

SUMMARY

Oxygen and carbon dioxide exchange and arterial blood levels have been studied in 20 patients during light methoxyflurane anesthesia, controlled ventilation, and varicose vein operations. Expired air was collected and analyzed by gas chromatography. Oxygen uptake ($\dot{V}_{o_2}$) during anesthesia was about 82 per cent of basal in both a premedicated group (10 patients) and an unpremedicated group (10 patients). The basis for this reduction in $\dot{V}_{o_2}$ was not entirely clear due to prior administration of thiopental and lower-than-normal body temperatures (mean, 35.4° C.). A case is made for expressing $\dot{V}_{o_2}$ during anesthesia relative to predicted basal values (ml./min./m².). Carbon dioxide levels were consistent with adequate ventilation and fairly predictable, retrospectively. Arterial oxygen levels were variable despite a steady mean alveolar P_{o_2}. Large mean alveolar-arterial P_{o_2} gradients were present in several instances, based, it is believed, on perfusion of nonventilated or underventilated alveoli.

ACKNOWLEDGMENT

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THE CARDIOVASCULAR EFFECTS OF CRANIALLY-IMPRESSED ELECTRIC CURRENTS OF ANESTHETIC INTENSITY

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THE APPEARANCE, with increasing frequency, of articles concerning the clinical use of electrical anesthesia reflects a resurgence of interest in this procedure. For many years researchers have recognized the potential value in a form of anesthesia that could be applied or withdrawn by the simple "flip of a switch." The prospect of a safe electrical anesthesia with instantaneous induction and immediate recovery has enticed researchers since 1875, when Mach[1] "narcotized" fish by means of a constant direct current. Sporadic reports in the literature since then largely represent efforts to refine or evaluate the various technics in the production of electrical anesthesia. A presentation of the historical background is beyond the scope of this work; however, the reader interested in this phase of electrical anesthesia is directed to the recent review of Van Poznak.[2]

Various technics for the administration of electrical anesthesia are under study in at least five teaching hospitals, and Price and Dornette[3] recently reported its use on 50 surgical patients at this institution. Since the complaint of cardiovascular complications under such anesthesia is commonly voiced[3, 4] and probably constitutes a principal objection to its use, we have attempted to measure the effects of cranially-impressed electric currents on a number of cardiovascular parameters.

METHODS

For the production of these electric currents a 700 cycles per second (cps) sine wave alternating current generator was employed. The selection of this frequency was not arbitrary but was based on the reports of Hardy and associates[4] and Knutson and co-workers,[5] whose earlier studies revealed 700 cps to afford the most satisfactory electronarcosis. This instrument contains a milliammeter with a range of 0 to 150 ma. and a 0 to 50-volt A.C. voltmeter. A potentiometer on the front of the instrument case permits the operator to vary the voltage and milliamperage.

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On the basis of trial-and-error approach to electrode positioning, the conventional bitemporal attachment was abandoned and an improved placement of the electrodes on the roof of the mouth (palatal vault) and vertex of the skull was employed. This location reduced appreciably the amount of current traversing the motor cortices and practically eliminated the convulsive activity usually evoked by the bitemporal passage of the electric current. The electrodes consisted of two "copper" pennies firmly affixed to the end of each jaw of a pair of spring-held tongs. An energizing wire was attached to each electrode. This device (fig. 1) facilitated the rapid and firm placement of one electrode on the surface of the palatal vault and the other on the vertex of the skull. The cardiovascular effects of rostral, central, or caudal positioning of the electrodes in the mouth were determined by a simple movement of the tongs. Figure 1 illustrates the four electrode positions employed in this study and will serve to clarify the meaning of the terms electrode position A, electrode position B, etc. Since electrode position B was used predominantly throughout this study, it is to be assumed that this placement of the electrodes was employed unless otherwise specified.

Myocardial contractile force measurements were obtained with a Walton-Brodie strain gauge arch sutured to the right ventricle and recorded with a model 5c Grass polygraph. A Carolina Medical Electronics square-wave electromagnetic flowmeter was employed in conjunction with the Grass polygraph to record femoral artery blood flow. Attempts to record internal carotid artery blood flow with this instrument were unsuccessful because of the proximity of the electromagnetic pickup probe to the electrodes. The current impressed across the brain of the dog during the experiments totally disrupted the integrity of the electromagnetic field of the adjacent flow probe. This precluded use of the electromagnetic flowmeter to measure cerebral blood flow. To overcome this difficulty, internal carotid artery blood flow was measured by means

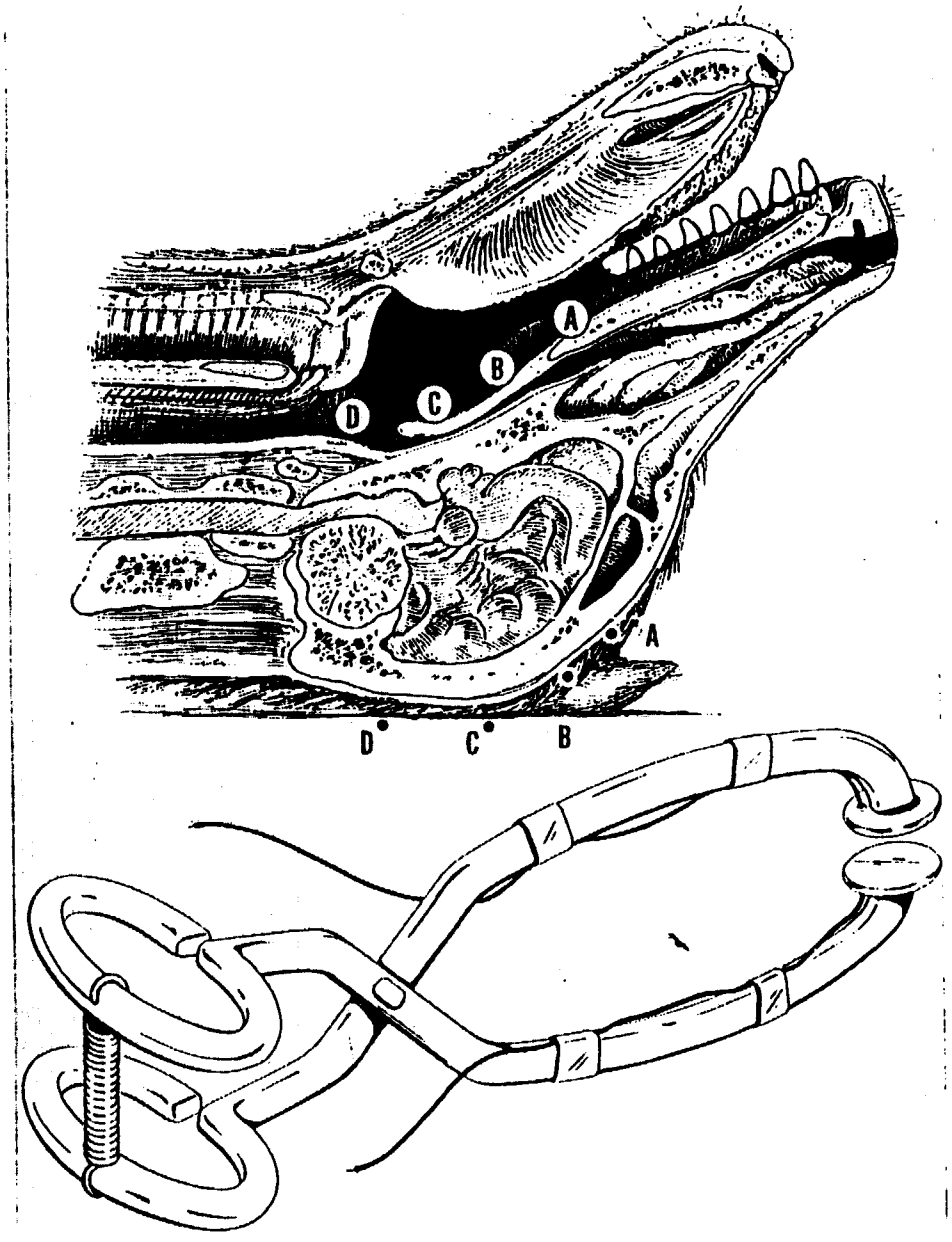

Fig. 1. Midsagittal section through head of dog indicating approximate electrode positions A, B, C, and D used in this study. Points marked A on palatal vault and vertex of skull approximate locations of the two electrodes when electrode position A was employed. Points B, C, and D indicate the other positions. Lower portion of figure shows electrode holder. Disc electrodes were adequately insulated from metallic body of tongs by a heavy layer of plastic.

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of a Shipley-Wilson rotameter, an instrument unaffected by the cerebrally-impressed electric current.

Skin temperature measurements were made with a Minneapolis-Honeywell (Brown Instruments Division) recording potentiometer equipped with six copper-constantan thermocouple leads. Blood pressure in these animals was measured by means of a Statham pressure transducer in communication with a polyethylene tubing inserted into the femoral artery. After cannulation or intubation of the trachea, artificial ventilation, just adequate to eliminate spontaneous breathing movements, was routinely employed throughout the experiments.

Forty-five adult, mongrel dogs (12 to 21 kg.) under pentobarbital sodium (30 mg. per kg. intravenously) anesthesia were used for these experiments.

The cardiovascular effects of cranially-impressed currents were studied in pentobarbitalized animals because:

1. Control studies revealed little difference between pentobarbitalized and nonpentobarbitalized dogs in their cardiovascular responses to electric currents of anesthetic intensity.

2. Clinically, electrical anesthesia is frequently superimposed upon an intravenous barbiturate induction.[3]

3. We were mainly concerned with the cardiovascular effects of passing electric currents through the brain, and an evaluation of the anesthetic effectiveness of these currents was not of primary importance in this study. Moreover, the current amplitude employed in this work was considered adequate for the production of canine electrical anesthesia on the basis of previous reports by other investigators working in this area. For example, Knutson and coworkers[5] produced electronarcosis in dogs with currents of 50 to 100 ma. (700 cps) and Hardy and associates[1] reported the achievement of adequate surgical anesthesia in dogs by the use of 35 to 40 ma. (700 cps.)

4. The ease of handling these animals was, of course, greatly facilitated by their narcotized state.

Each response in the subsequent results has been confirmed in at least three dogs, and the number of animals used in each procedure is, therefore, not repeatedly cited.

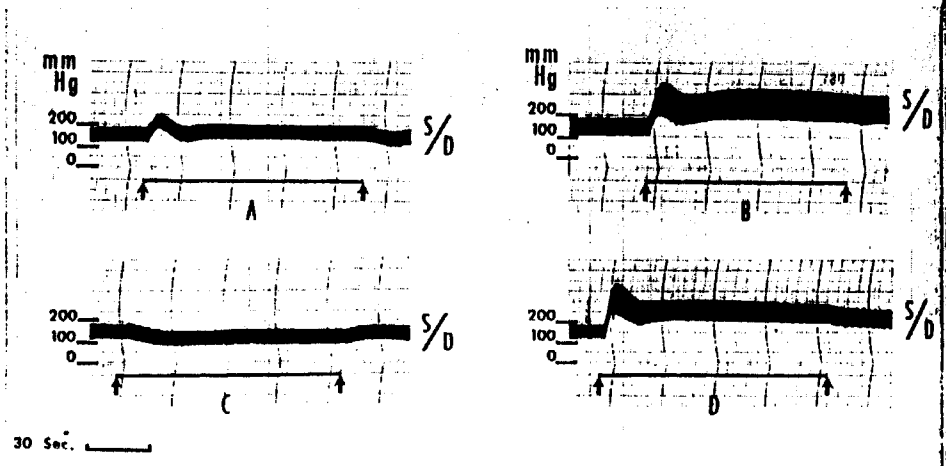

Fig. 2. Blood pressure responses evoked by passage of a 100-ma., 18-volt current through brain with electrodes placed successively in the four positions indicated in figure 1. Dog, male, 15.3 kg., pentobarbital sodium (30 mg. per kg.). Atropine sulfate (100 micrograms per kg.) and succinylcholine chloride (400 micrograms per kg.) were given intravenously prior to first application of electric current. Artificial respiration maintained throughout experiment. S/D = systolic/diastolic arterial pressure. Arrows, with intervening horizontal bars, indicate period during which current was on. Letters A, B, C, and D, signify electrode position during application of corresponding current.

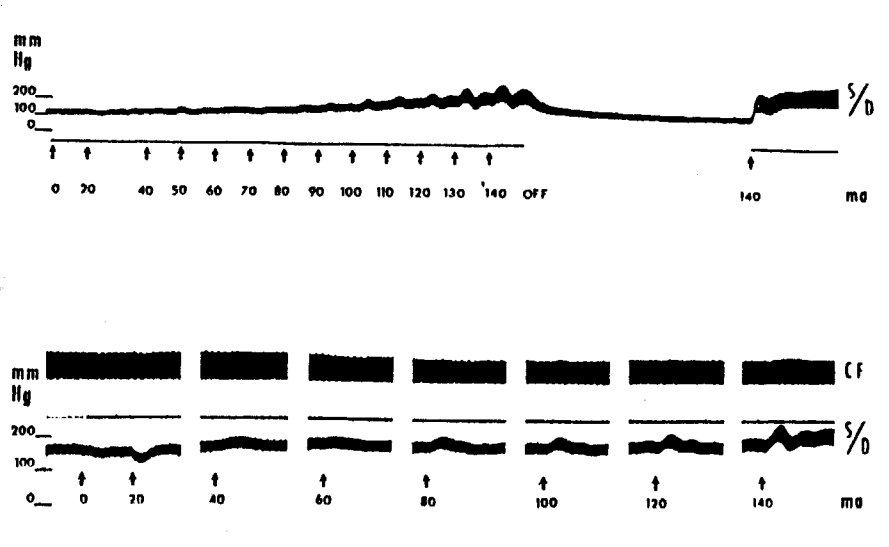

Fig. 3. Blood pressure and cardiac contractile force responses to a cranially-impressed current which was increased in stepwise increments from 0 to 140 ma. (0 to 22 volts). Letters S/D and CF at right represent systolic/diastolic arterial pressure and cardiac contractile force, respectively; ma. — milliamperes of current as specified by the numbers and arrows at the bottom of tracings A and B. Tracing A (upper half of figure): dog, male, 19 kg., pentobarbital sodium (30 mg. per kg.) Atropine sulfate (100 micrograms per kg.) and succinylcholine chloride (400 micrograms per kg.) given intravenously prior to application of current (electrode position B). Artificial respiration throughout. Uninterrupted current, raised in small increments from 0 to 140 ma., each increment maintained for 10 seconds. At right, for comparison, blood pressure response to an instantaneously applied current of 140 ma. is shown. Tracing B (lower half of figure): dog, male, 17 kg., pentobarbital sodium (30 mg. per kg.) Electrode position B. Atropine sulfate (100 micrograms per kg.) and succinylcholine chloride (400 micrograms per kg.) given intravenously before application of current. Artificial respiration maintained. Same procedure as in tracing A except that each increment of current was maintained for 60 seconds rather than 10 seconds.

RESULTS

The Effect of Cranially-Impressed Currents on the Blood Pressure of the Dog — By carefully positioning the electrodes, variable blood pressure responses could be evoked consistently in dogs having received atropine and succinylcholine. With the electrodes in position A (fig. 1), a current of 100 ma. produced no marked change in blood pressure (fig. 2) except for an initial moderate pressor response of 15 seconds duration. A caudal movement of the electrodes to position B and a reapplication of the electric current (same parameters) produced a dramatic change in the character of the blood pressure response, and a dangerously high hypertension persisted throughout the period that the current was applied. When electrode position C was employed, the same current parameters evoked no pressor activity, but rather a moderate fall in blood pressure which also endured as long as the current was on. Finally, placement of the electrodes in position D and application of the current elicited a blood pressure response similar in character to that obtained at position B but the maintained component of the hypertensive response was somewhat less in amplitude.

Figure 3 shows the effect on blood pressure produced by a gradual, stepwise increase of the current with the electrodes fixed in position B. In the upper half (A) of this record, the current was uninterrupted, but was increased by a 10-ma. increment every 10 seconds. In this fashion the current was raised from 0 to 140 ma. At the extreme right of this record, for purposes of comparison, the blood pressure to an instantaneously applied current of 140 ma. is shown. The bottom half (B) of

figure 3 shows a typical record from another series of experiments in which the same procedure was followed except that the current was increased by an increment of 10 ma. every 60 seconds instead of every 10 seconds. In order to compress this record, only every second increment of current was included in this figure. Note that when the milliamperage was increased relatively slowly (B), and adequate time was permitted to allow cardiovascular compensation, the base line blood pressure did not increase significantly until the current reached 140 ma. Moreover, as seen in this record, the slow increase in current elicited no appreciable alteration in myocardial contractile force.

Activation of the Vagi by Cranially-Impressed Currents — Under cranially-impressed currents, vagal influence upon the heart can be increased to the point of transitory cardiac arrest. Figure 4 is typical of this action. The sudden application of 100 ma. of current across the electrodes (position B) produced an immediate cardiac arrest. Despite the maintenance of this current, however, "vagal escape" occurred and the heart resumed a very slow but regular beat. This bradycardia persisted until the current was shut off, at which time the heart rate markedly increased but the rhythm became quite irregular. It is interesting to note that during the period of heightened vagal tone, nonetheless, a marked hypertension developed and myocardial contractile force doubled. Both vagotomy and atropinization (100 micrograms per kg.) dramatically eliminate the cardiac arrest and/or bradycardia coincident with the sudden application of these electric currents.

Figure 5 demonstrates that bilateral cervical vagotomy promptly eliminates the bradycardia attendant upon the delivery of the electric current. During the application of 100 ma. of current (position B), first the left and then the right vagus was severed. The immediate onset of tachycardia and hypertension, which follows the release from vagal inhibition, is clearly evident in this figure. As expected, in the nonvagotomized animal the intravenous administration of atropine sulfate (100 micrograms per kg.) prevents the onset of both the transitory cardiac arrest and the subsequent

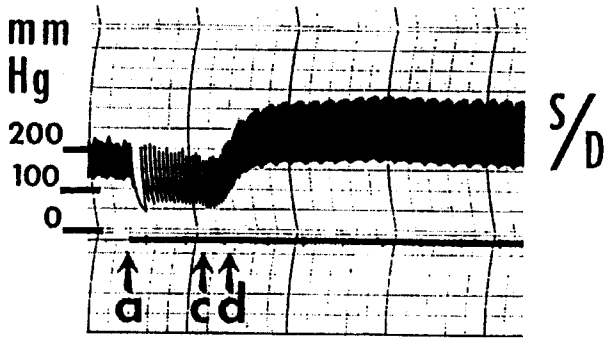

Fig. 5. Effect of bilateral vagotomy on bradycardia induced by passage of a current (100 ma., 17 volts) through brain of unatropinized dog. Electrode position B. Dog, female, 18 kg., pentobarbital sodium (30 mg. per kg.). Succinylcholine chloride (400 micrograms per kg.) given intravenously before application of electric current. Artificial respiration maintained throughout experiment. S/D — systolic/diastolic arterial pressure, a — onset of current, c — transection of left cervical vagus, d — transection of right cervical vagus. Horizontal signal bar represents time current was on, 100 seconds.

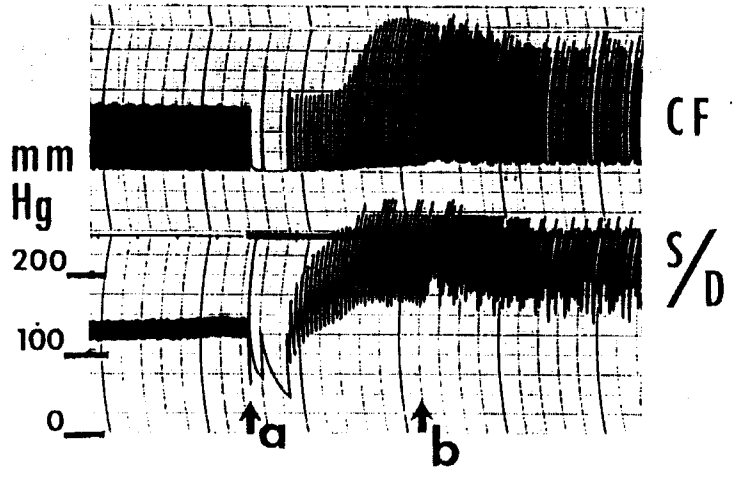

Fig. 4. Vagally-mediated cardiac arrest and subsequent bradycardia resulting from passage of a current (100 ma., 18 volts) through brain of unatropinized dog. Electrodes in position B. Dog, male, 14.8 kg., pentobarbital sodium (30 mg. per kg.) Succinylcholine chloride (40 micrograms per kg.) given intravenously prior to application of electric current. Artificial respiration maintained throughout experiment. CF — cardiac contractile force, S/D — systolic/diastolic arterial pressure, a — onset of electric current, b — withdrawal of current. Horizontal signal bar indicates time current was on, approximately 45 seconds.

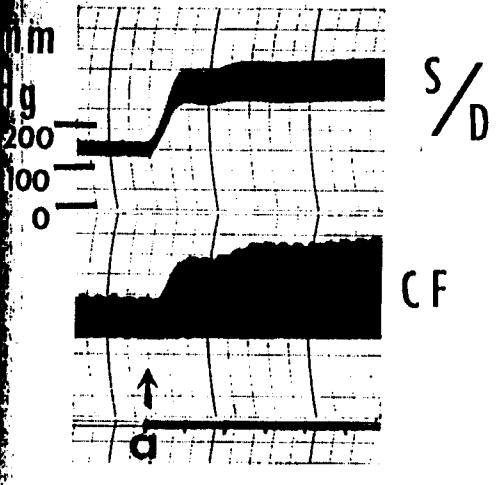

Fig. 6. Prevention of current induced cardiac arrest and bradycardia by atropine. Current of 100 ma., 17 volts. Electrode position B. Dog, male, 13.8 kg., pentobarbital sodium (30 mg. per kg.). Atropine sulfate (100 micrograms per kg.) and succinylcholine chloride (500 micrograms per kg.) given intravenously before application of electric current. Artificial respiration throughout experiment. S/D — systolic/diastolic arterial pressure, CF — cardiac contractile force, a — onset of current. Horizontal signal bar represents time current was applied, 60 seconds.

bradycardia normally evoked by the passage of the electric current (fig. 6).

The Effects of Cranially-Impressed Currents on Drug Responsiveness — During electrical anesthesia, the cardiovascular system retained the capability for vigorous drug responses. Figure 7 is representative of a series of responses to l-epinephrine (2 micrograms per kg. intravenously) administered while dogs were receiving a current of 60 ma. (position B). Note that a significant increase in cardiac contractile force and blood pressure was evoked by epinephrine while the current was still on. Heart rate, which rose from a control of 126 to 138 beats per minute under the influence of the electric current, increased no further upon administration of the epinephrine.

By pharmacologically blocking the cardiac responses, it was possible to determine the degree of vasomotor participation in the hypertension induced by electrical anesthesia. Such a blockade was achieved by the intravenous administration of 5 mg. per kg. of dichloroisoproterenol (DCI), a drug capable of blocking the positive cardiac inotropic and chronotropic actions of epinephrine and norepinephrine.[6]

Following pretreatment with DCI, 120 ma. of current (position B) elicited a significant hypertensive response in the absence of an increased cardiac contractility or rate (fig. 8). It is notable that cardiac contractile force was even decreased during the hypertensive period. Moreover, as seen in this figure, the intravenous administration of hexamethonium bromide (1 mg. per kg.), promptly reduced the blood pressure elevation induced in this series of animals by electrical anesthesia. Since cardiac contribution to the hypertensive response was presumably eliminated in these animals, the hypertension obviously was sustained by peripheral vasoconstriction, doubtless caused by a combination of preganglionic sympathetic activation and consequent adrenal medullary discharge. As previously reported by Hardy and associates,[1] phentolamine was also found to be effective in reducing, or blocking, the hypertension elicited.

Effects of Cranially-Impressed Currents on Blood Flow — In order to determine if cerebral blood flow was adversely effected by the passage of these electric currents through the brain, flow measurements were conducted on the common carotid arteries of dogs. Figure 9 is representative of the effect of applying a 100-ma. current through the electrodes at position B. In this anima

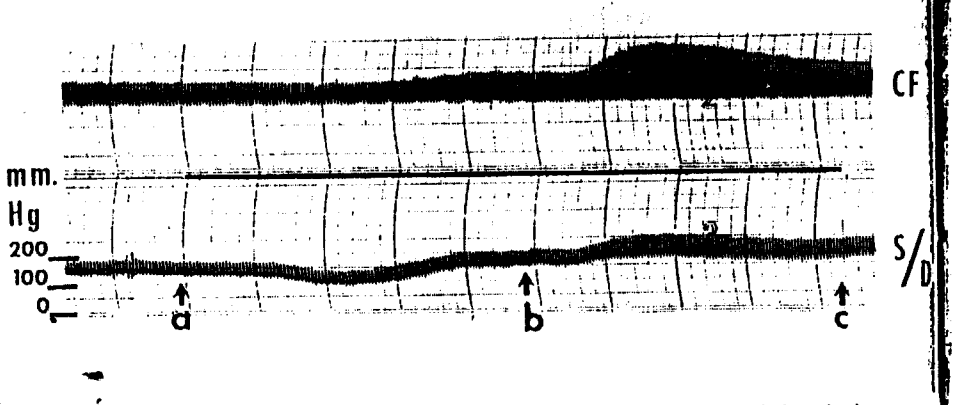

Fig. 7. Blood pressure and cardiac contractile force responses to epinephrine during passage of a current (60 ma., 14 volts) through brain. Electrode position B. Dog, female, 14.4 kg., pentobarbital sodium (30 mg. per kg.). Atropine sulfate (100 micrograms per kg.) and succinylcholine chloride (400 micrograms per kg.) given intravenously prior to application of electric current. Artificial respiration throughout. CF — cardiac contractile force, S/D — systolic/diastolic arterial pressure, a — onset of electric current, b — l-epinephrine (2 micrograms per kg. intravenously), c — withdrawal of current. The horizontal signal bar represents a period of 90 seconds during which current was applied.

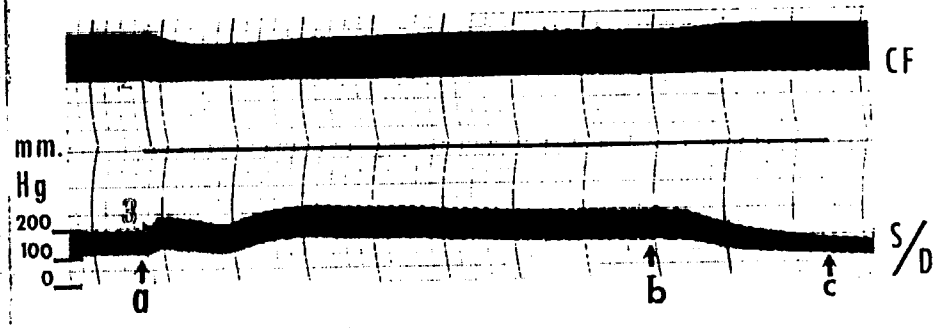

Fig. 8. Modification by DCI (5 mg. per kg.) of cardiac contractile force and blood pressure responses to a cranially-impressed electric current (120 ma., 24 volts). Electrode position B. Dog, female, 17.3 kg., pentobarbital sodium (30 mg. per kg.). Atropine sulfate (100 micrograms per kg.) and succinylcholine chloride (400 micrograms per kg.) given intravenously before application of current. Artificial respiration maintained throughout experiment. CF — cardiac contractile force, S/D — systolic/diastolic arterial blood pressure. At point "a", electric current applied. At point "b", hexamethonium bromide (1 mg. per kg. intravenously) given. Electric current flow terminated at point "c". Time lapse between "a" and "c" equals 4 minutes.

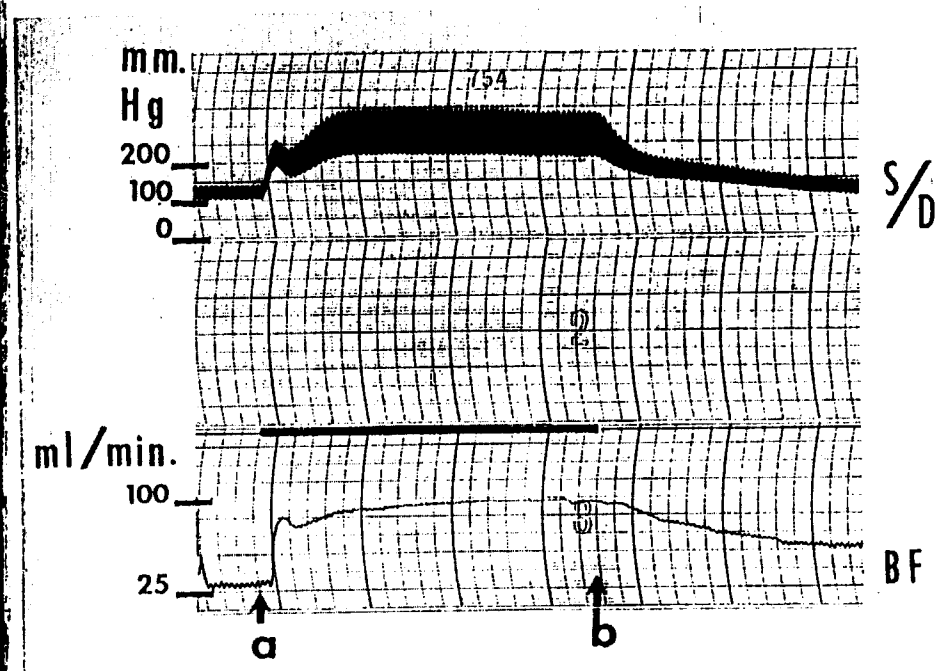

Fig. 9. Effects of a cranially-impressed current (100 ma., 19 volts) on internal carotid blood flow. Electrode position B. Dog, female, 16.4 kg., pentobarbital sodium (30 mg. per kg.). Atropine sulfate (100 micrograms per kg.) and succinylcholine chloride (400 micrograms per kg.) given intravenously just preceding response shown. Artificial respiration maintained. S/D — systolic/diastolic arterial blood pressure, BF — left internal carotid blood flow. Current applied between points "a" and "b", a period of approximately 3 minutes.

the left external carotid branch was ligated, and flow through the patent left internal carotid artery was measured. All other vessels supplying or draining the brain were intact. The increase in internal carotid blood flow induced by passage of the electric current through the brain is seen to mirror the simultaneous increase in systemic blood pressure.

Measurements of femoral artery blood flow during electrical anesthesia, likewise, showed increases, again presumably attributable to the marked increases in systemic pressure. Alterations in skin temperature were used as an indirect index of changes in cutaneous blood flow. Since skin temperature in the hind limbs, fore limbs, and torso fell approximately ½ to 1° C. during the application of a 100-ma. current across the brain, it was inferred that cutaneous vasoconstriction and reduced cutaneous blood flow occurred. Further studies concerning temperature changes are currently under investigation.

DISCUSSION

The important potential advantages of electrical anesthesia represent a constant enticement to those who do research in the area of general anesthetics. As pointed out by Hardy and associates,[4] the immediacy of induction and recovery could reduce both preanesthetic and postanesthetic hazards and spare the patient considerable discomfort. Moreover, since the brain is primarily affected by the electric current, the liver, kidneys, and similar organs need not be directly exposed to the potentially noxious actions of some of the drugs used for chemical anesthesia. But if the advantages are noteworthy, so are the disadvantages.

Formidable dangers attendant upon the use of electrical anesthesia still exist. The massive autonomic discharge occasioned by the electric current must be acknowledged. Furthermore, such features as marked hypertension, arrhythmias, salivation, muscle twitchings, and laryngospasm are troublesome. The difficulty in assessing accurately the degree of analgesia and anesthesia after administration of the necessary neuromuscular blocking agents also has contributed to the unpopularity of electrical anesthesia. Pharmacologic advances, however, reflected in the ever-growing armamentarium of the modern physician, have made readily accessible the means for overcoming most of these objections.

Any excessive or dangerous degree of hypertension occasioned by electrical anesthesia can be abolished by careful administration of such adrenergic blocking agents as phentolamine. Perhaps more important, there exists the possibility of eliminating this hypertensive response in patients by a more discreet positioning of the electrodes (fig. 2, electrode position C). We are currently investigating this prospect clinically.

Arrhythmias, bradycardia, and excessive salivation can be largely controlled by atropine sulfate. Convulsions and laryngospasm are prevented by the administration of suitable neuromuscular blocking agents, and intubation, with artificial respiration, insures adequate ventilation. Moreover, Price and Dornette[3] have indicated that careful monitoring of blood pressure, heart rate, and eye signs is clinically useful in evaluating the degree of electrical anesthesia.

The authors do not wish to imply that electrical anesthesia stands at the threshold of general clinical acceptance. It is by no means perfected, and conceivably it may never supplant conventional chemical anesthesia; but it would appear that many of the major problems inherent in its use may be resolved by continued research.

SUMMARY

The effects of cranially-impressed currents (electrical anesthesia) on cardiovascular function were studied in 45 dogs. Instead of the conventional bitemporal placement of the anesthesia-inducing electrodes, a modified electrode position was employed, utilizing the roof of the mouth (palatal vault) and the top of the head (vertex of skull). Precise positioning of the electrodes is important inasmuch as a hypertensive response to the application of an electric current can be reversed to a hypotensive response (or vice versa) by a slight rostral or caudal movement of the head electrodes. Moreover, by slowly increasing the current and thereby permitting a degree of cardiovascular compensation, the intensity of the evoked cardiovascular response appears to be sharply reduced.

Cranially-impressed currents of anesthetic intensity strongly activate the autonomic nervous system. The consequent vagal and sympathetic effects on the heart are shown to be blocked by atropine and dichloroisoproterenol (DCI), respectively.

Internal carotid and femoral artery blood flows were significantly increased by electric currents which produced marked hypertension. Cutaneous blood flow, conversely, appeared to be reduced.

During electrical anesthesia, the cardiovascular system remains vigorously responsive to drugs such as certain adrenergic blockers, ganglionic blockers, and sympathomimetics.

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Cave Men of the Future. We are the cave men of the world of the future, fumbling our way amid dangers of our own creation and destruction. *James T. Shotwell, Lesson on Security and Disarmament.*

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Tell me thy company, and I'll tell thee what thou art. — *Cervantes*

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It is a good thing to be rich, it is a good thing to be strong, but it is a better thing thing to be beloved of many friends. —Euripides

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Our problem is within ourselves. We have found the means to blow the world physically apart. Spiritually, we have yet to find the means to put together the world's broken pieces. —Thomas E. Dewey

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Creative thinking is today's most prized, profit-producing possession for an individual, corporation or country. It has the capacity to change you, your business and the world. —Robert P. Crawford

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How can great minds be produced in a country where the test of great minds is agreeing with the opinion of small minds? John Stuart Mill

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The ass will carry his load, but not a double load; ride not the free horse to death. —Cervantes

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Better a little chiding than a great deal of heartbreak. —Shakespeare

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Those who play with cats must expect to be scratched. — Cervantes