THE ELECTRO-ENCEPHALOGRAM IN THE EVALUATION OF THE EFFECTS OF ANESTHETIC AGENTS AND CARBON DIOXIDE ACCUMULATION DURING SURGERY*

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INTRODUCTION

THE RECENT EMPHASIS on cardiac arrest and unexplained hypotension during surgery makes it desirable to understand something of the physiologic mechanisms responsible for these untoward reactions. One important factor may well be the respiratory acidosis which Beecher,[1] and others[17, 18, 25] have demonstrated to occur, particularly during thoracic surgical procedures. The appearance in the blood of abnormal tensions of carbon dioxide, probably the result of impaired respiratory gas exchange within the lungs, can lead to serious physiologic disturbances. In view of the vital cardiovascular control high within the central nervous system, it appears reasonable to examine the relationship of carbon dioxide and ether on the activity of the central nervous system. Depression of this activity may well be considered in terms of the degree of narcosis present. That carbon dioxide in high concentration within the blood stream becomes a narcotizing agent is well known.[21] This effect is potentiated by the presence of barbiturates,[2] and presumably by other anesthetic agents. Although some success has attended efforts to follow carbon dioxide accumulation in the blood by changes in the carbon dioxide tension of the alveolar air,[8, 9] this method fails to give a direct indication of the overall status of the central nervous system.

The electro-encephalogram offers an index of organized activity within the brain. These fluctuating potentials, measurable at the surface, are influenced by many chemical agents, the most potent being those which affect nerve cell metabolism.[3] Following the early work of Gibbs, Gibbs and Lennox,[11] little attention was given to the brain waves under anesthesia in man until Brazier and Finesinger[4] investigated the barbiturates and Courtin, Bickford, and Faulconer[5] classified the electro-encephalographic patterns found during ether anesthesia. This work and that to be reported indicate that the electro-encephalographic patterns observed under these circumstances give a valid indication of the degree of narcosis present.

This paper is presented to demonstrate that with the electro-encephalogram as the indicator, it is possible and convenient to follow the depression of brain activity during surgery. This depression carried far enough, may be the cause of untoward events. Furthermore, it is shown by experiments in animals and man that the depressant action of certain anesthetic agents and

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that of excess carbon dioxide are summated in their effect on nerve cell function as reflected by the changes exhibited in the brain waves.

METHODS AND MATERIALS

Electro - encephalographic recordings were made with a Grass ink-writing oscillograph, Model IIIB.[10] Stainless steel or silver needles, soldered to wire connectors, were placed in the scalp to serve as electrodes. Amplification was adjusted to produce a pen deflection of 6 mm. for each 50 microvolts potential. A simultaneous electrocardiographic tracing (Lead II) was made on a separate channel of the same oscillograph.

To obviate the necessity of continuous recording, a cathode ray oscillograph was connected to an amplifier of the oscillograph. This permits the observer to monitor the brain waves constantly and to record only those of interest.

Arterial blood samples were analyzed to determine the p CO_2, oxygen saturation, and ether concentration. The p CO_2 was calculated from the nomogram of Singer and Hastings.[23] The CO_2 and oxygen contents of each blood sample were determined by the method of Neill and Van Slyke;[26] the pH was found by means of the glass electrode and corrected to 37° C. In the presence of ether, the modification of the Van Slyke technic described by Shaw and Downing[22] was employed. Under these circumstances oxygen content was established by the electrophotometric method of Hickam and Frayser.[15] A Wood oximeter and cuvette[27] were employed to follow oxygen content of the blood in certain of the experiments. The blood ether concentration was arrived at by the technic developed by Harger,[14] which was found to be accurate to within 9 per cent. In one series pentobarbital anesthesia was used, and the blood levels of this drug were determined by the method of Goldbaum.[13] Otherwise all experiments on animals and patients were conducted in the presence of ether administered at a steady rate from an anesthesia machine.

Hypercapnia was produced by introducing mixtures of CO_2 and O_2, ranging from 5 per cent to 40 per cent of CO_2, into the bag of the anesthesia machine. To assure the absence of anoxia, respiration was assisted. In some instances, while using nembutal anesthesia, the animal was made to breathe directly from a Douglas bag containing the desired gas mixture. Similarly, anoxia was produced in animals with mixtures of nitrogen and oxygen, containing from 2.5 to 15 per cent oxygen. In no instance was a patient intentionally subjected to anoxia.

PROCEDURE

Animal Experiments

The electro-encephalograms and electrocardiograms of eight dogs, anesthetized with ether, were followed as the anesthesia was carried down to the fourth plane of Stage III. When respiration became depressed, it was assisted, to be sure that anoxia or hypercapnia did not occur. Ether administration was then discontinued until the clinical upper second plane was reached. At this point ether was again given at a rate to maintain as nearly as possible the given anesthetic level. When a steady state appeared to have been attained both clinically and by E. E. G., hypercapnia was induced. A comparison between the E. E. G. of deep anesthesia, and light anesthesia with hypercapnia was made.

A second series of seven dogs were given pentobarbital intravenously 60 mg. per Kg. of body weight. After a lapse of at least 60 minutes, permitting a relatively steady level of anesthesia to develop,[24] an arterial blood sample was drawn to determine the p CO_2, O_2, and pentobarbital levels. This was repeated at the termination of the experiment to be sure that no marked change in the blood concentration of pentobarbital

had occurred during the experiment. Progressive degrees of hypercapnia were induced while oxygenation of the blood was maintained at a normal level. Arterial blood samples were obtained at intervals to compare the changes in p CO_2 with those of the electro-encephalogram. Upon completion of these observations, the animal was again allowed to breathe room air and to return to his basal state of anesthesia. Anoxia of various degrees was then induced, keeping arterial CO_2 tension normal. The degree of anoxia was followed on the oximeter and checked by three blood samples obtained as the electro-encephalographic changes appeared.

Experiments on Man

Eight suitable patients who were to have pelvic or other relatively uncomplicated operations were selected for examination of electro-encephalographic patterns under controlled conditions of anesthesia. All were carried on ether anesthesia except for small intravenous doses of thiopentobarbital sodium (up to 250 mg.) at the outset to aid induction. A Cournand needle was placed in the radiol artery. Electro-encephalographic electrodes were placed at the apex and occiput. When induction was complete and a steady state of light anesthesia had been attained, an arterial blood sample was drawn to determine p CO_2, oxygen saturation, and ether concentration. This was repeated throughout the experiment, six to 10 samples being obtained for each patient. Using assisted respiration to avoid anoxia or hypercapnia, ether was administered until lower plane II of the third stage was reached. Ether was shut off and the patient allowed to return to upper plane II. An attempt was made to determine the rate of ether administration for maintenance of anesthesia at this level. Mixtures of oxygen containing 10 per cent, 20 per cent, and 40 per cent carbon dioxide were then added to the ether. As first hyperpnea and then depression developed, the encephalogram was carefully followed until there appeared an E. E. G. type of pattern similar to that of deep ether anesthesia. The carbon dioxide absorber was then turned on and the patient was allowed to return to a state of light anesthesia.

A second group of 36 patients undergoing major intrathoracic surgical procedures were simply followed by the electroencephalogram throughout the period of anesthesia. No attempt was made to alter the condition of the blood beyond what took place as a result of the procedure. Arterial blood samples were obtained from 16 of these at intervals as the E. E. G. pattern changed, or at other specific times during operation.

RESULTS

Effect of Ether on the Electro-encephalogram. Six distinct types of electro-encephalographic patterns were distinguished as the anesthesia was made to progress from induction down to very deep levels. The patterns illustrated in Fig. 1, which were taken from a 36-year-old woman undergoing hysterectomy, are quite typical of what may be expected. The patterns are classified according to Courtin, Bickford and Faulconer,[5] with minor modifications. The electro-encephalograms obtained from animals under the experimental conditions were similar in all respects except that, in general, somewhat higher frequencies were found.

For observation of E. E. G. patterns during anesthesia the vertex-occipital lead was found to be the most useful because it provided the highest potentials. Changes from the basal were therefore more easily apparent.

Type I pattern. Shortly after anesthesia is started, and usually during induction, the normal alpha wave occipital pattern (eyes closed) changes to one characterized by waves of irregular, fast frequency, 20 per

FIG. 1

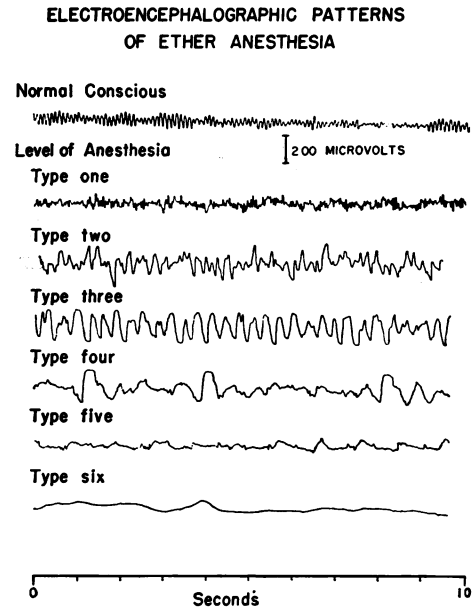
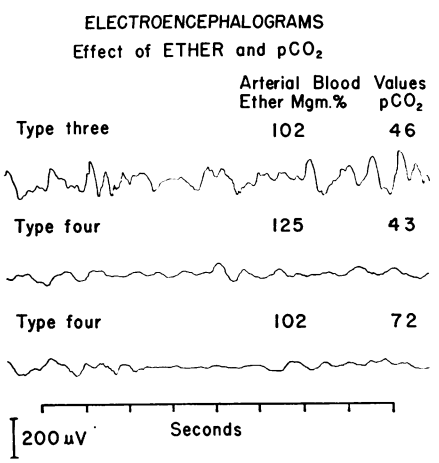


FIG. 2



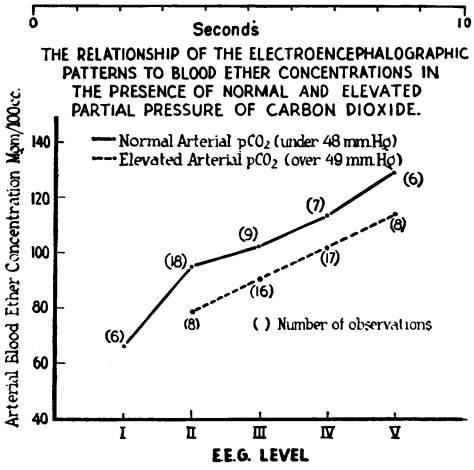


FIG. 3

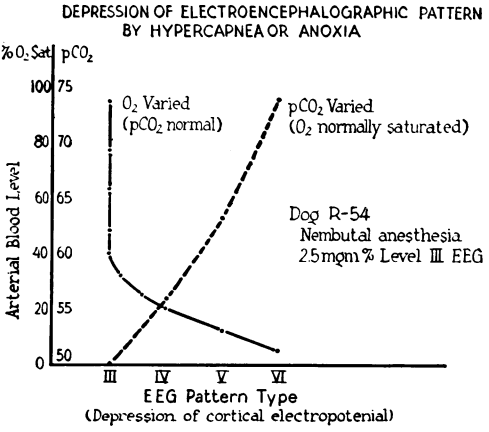


FIG. 4

FIG. 1. The typical occipital electro-encephalograms at the various levels of ether anesthesia in a patient undergoing a surgical operation.

FIG. 2. The electro-encephalographic patterns of man obtained during a representative experiment which indicate that the changes in pattern are affected similarly by an increase of blood ether concentration or by a rise in the arterial carbon dioxide tension.

FIG. 3. A comparison of the blood ether concentrations required to produce the various electro-encephalographic patterns in the presence of normal and elevated carbon dioxide tensions, showing the greater degree of narcosis produced at a given ether level when arterial p CO_2 is elevated.

FIG. 4. A representative animal experiment to illustrate the severe degree of anoxia required to affect the electro-encephalogram during anesthesia compared to the progressive additive depressant action of rising arterial carbon dioxide tensions.

second or more, and of lower potential. This corresponds to the second stage of anesthesia.

Type II pattern. As the first plane of Stage III is approached, the pattern changes to one showing waves of an irregular, slower frequency (4 to 12 cycles per second) and of higher than normal potential (up to 200 microvolts).

Type III pattern. In the lower level of plane one or upper level of plane two there appear large delta waves of very low frequency (one to three per second). These are of large potential, over 200 microvolts.

Type IV pattern. With further deepening of anesthesia, the potential begins to decrease (under 200 microvolts) and the large delta wave complexes of Type III become less frequent. These complexes may occur at intervals of 3 to 5 seconds, with isopotential periods between. This pattern is usually seen in middle or lower plane two of surgical anesthesia.

Type V pattern. In the upper third plane of anesthesia as intercostal breathing begins to disappear the Type V pattern appears. It is characterized by a loss of organized frequency and markedly diminished potential. At times fast waves of very low potential (5 microvolts) are seen superimposed on the slow waves, which seldom have a potential over 20 microvolts.

Type VI pattern. If anesthesia is pushed to the fourth plane in which respiratory arrest occurs, only extremely slow waves of less than 10 microvolts remain. These waves may last as long as three seconds with periods of as much as 10 seconds between, with no activity. In dogs which were maintained for periods up to 5 minutes at this level, minute waves (5 microvolts) of 10 to 20 per second were seen superimposed on the long, slow waves. The fine waves were not observed in the three patients which reached this level.

In four dogs which were allowed to progress to death from overdosage of anesthetic during artificial respiration, the Type VI pattern disappeared and no waves were visible after periods up to nine minutes of observation.

The same progression of pattern type was found in all the patients studied. The clinical signs upon which are based the classification of the plane of anesthesia were not as clear-cut, and did not all follow exactly the average levels of anesthesia mentioned in describing the types of patterns. Furthermore, between Types III, IV, V and VI there may be a gradual transition from one type to the next, as depression of activity takes place.

Forty-six determinations of blood ether concentration in 17 patients are compared with the electro-encephalographic pattern types in the presence of normal p CO_2 values. The average blood ether levels required to produce the patterns I to V are found in the first column of Table I. Because of the wide range of tolerance to ether exhibited by the patients, the standard deviations and standard errors of the mean values are quite large.

Effect of Hypercapnia on the Electroencephalographic Patterns of Anesthesia. The preliminary animal experiments indicated that when respiratory acidosis was made to occur by the administration of carbon dioxide, a transient stimulation of respiration and rise of blood pressure occurred. This was followed by depression, and in 75 per cent of the dogs, a moderate hypotension. These changes were seen to be reflected in the electro-encephalogram. Animals at a given anesthetic level, whether ether or nembutal, showing a given type of E. E. G. pattern, were found to exhibit an increase in the cortical depression when given CO_2. This was manifested by a decrease in potential from Type III to Types IV and V in every respect similar to that produced by an increase in anesthetic agent.

In eight patients, in whom CO_2 was purposely made to accumulate, exactly the same events occurred. The addition of CO_2 with a concomitant rise of p CO_2 resulted in a depression of cortical electrical activity similar to that produced by the addition of ether. This was particularly true if the patient's E. E. G. was already of Type III or higher. The E. E. G. patterns obtained in a typical experiment are illustrated in Fig. 2.

The Effect of Anoxia on the Electro-encephalographic Patterns of Anesthesia. At a given pentobarbital anesthetic level and in the presence of normal arterial p CO_2 values, little effect on the E. E. G. pattern was produced until a level of 40 per cent oxygen saturation was reached in the arterial blood. From that level downward, the cortical depression progressed rapidly. The contrast between the effect of anoxia and elevated p CO_2 is illustrated by the result of a typical experiment in Figure 4.

TABLE I. *The Effect of Ether and Carbon Dioxide Concentrations in Arterial Blood on the Electro-encephalographic Patterns of Anesthesia.*

E. E. G. Type	Blood-Ether Concentration Mg./100 cc.							
	p CO_2 Normal Range				p CO_2 Elevated (over 49 mm. Hg.)			
	Mean	No. of Determ.	Std. Dev.	Std. Error	Mean	No. of Determ.	Std. Dev.	Std. Error
I	66	6						
II	95	18	22.4	5.27	79	8	19.9	7.3
III	102	9	44	14.6	90	16	13.4	3.4
IV	113	7	17	7.5	102	17	23.5	5.7
V	129	6			114	8	22	7.8

It will be seen that, while the blood ether level remained the same (102 mg. per cent), an increase of CO_2 tension to 24 mm.Hg. above normal caused the E. E. G. pattern to progress from Type III to lower Type IV. Previously, the same E. E. G. pattern had been produced by an increase in blood ether level to 125 mg. per cent while p CO_2 remained normal.

At a lighter stage of anesthesia in which Type I or Type II patterns were present, the induction of hypercapnia often acted as a stimulant, and progression of cortical depression did not occur. Instead the frequency of the brain waves increased, with a moderate reduction of potential. This was found in both the dog and man.

The data on blood ether levels obtained from 17 patients in the presence of abnormally high partial pressures of carbon dioxide in arterial blood are set forth in the second set of columns of Table I. Although the standard errors are high, the ether values producing Types IV and V are sufficiently different from those of the first column to be significant. This suggests that less ether is required in the presence of abnormally large CO_2 blood levels to produce a given degree of cortical depression (Fig. 3).

The Electrocardiogram. Changes from the normal electrocardiographic pattern were not seen in patients or animals with intact chests until the p CO_2 had increased to the point where a Type V electro-encephalogram was produced. At this stage, only two of eight patients with experimentally produced hypercapnia exhibited slight depression of the P and T waves. Fifty per cent of the experimental animals exhibited similar changes during severe cortical depression by CO_2.

On the other hand, in animals, a decrease in electrocardiographic potential was found in all complexes with accompanying ventricular extrasystoles when the oxygen saturation in the blood fell to less than 20 per cent.

Clinical Experience. Of the 79 operations in which the electro-encephalogram has been used to follow the patient's progress through the procedure, it has proven of value in several instances by warning of dangerous depression. Two such cases are illustrated in Figures 5 and 6, which are records of the changes of the E. E. G. patterns, blood p CO_2, and ether levels.

The operation course shown in Figure 5 is that of an upper lobectomy performed to eradicate a tuberculous focus in a 42-year-old white man. During the major portion of the operation, the anesthetist maintained a fairly steady level of ether in the blood. The patient was in the prone position, and the maintenance of a normal blood CO_2 tension by assisted respiration was not difficult. The E. E. G. pattern remained between Type II and IV throughout with but two exceptions. The only severe depression of cortical activity occurred near the end of the operation when bleeding, which proved difficult to control, developed in the apex of the thoracic cavity. The lower lobe was packed down with a large taped sponge, and the patient was temporarily allowed to breathe without assistance. The partial pressure of CO_2 rose to 74 mm.Hg. Shortly thereafter the E. E. G. pattern showed severe depression, and respiration ceased. When the lower lobe was re-expanded, and adequate respiratory exchange was instituted, the p CO_2 fell. Concomitantly, the cortical depression disappeared, as the E. E. G. returned to Type IV pattern. The operation was successfully completed without further incident.

Figure 6 illustrates the course of a pneumonectomy in a 40-year-old Negro man, rendered difficult by the presence of tuberculous lymph nodes about the hilus. This patient exhibited a considerable tolerance to ether in that his E. E. G. pattern was of type IV with an ether blood level of 150 mg. per cent, while the arterial CO_2 tension was nearly normal. At point two the bronchus was clamped, leaving the vessels intact. The blood CO_2 tension started to rise, and despite a slight fall of the ether concentration, the E. E. G. pattern progressed from Type IV to Type V, and nearly reached Type VI. Shortly after the pulmonary artery had been ligated, the arterial p CO_2 had fallen from 94 to 68 mm.Hg. This was accompanied by a gradual return of cortical electrical activity as the E. E. G. pattern progressed from Type V to Type III. The operation was successfully finished, with a gradual return of the E. E. G. to a normal pattern.

In approximately 15 per cent of the operations in which the brain waves have been followed, the electro-encephalograph has not been satisfactory because of outside electrical interference or the failure to reproduce the waves. Ordinarily, the cause of these difficulties was found to be faulty grounding of the patient or the electroencephalograph. Another cause of trouble was lack of good contact to the needle electrodes, or that a wet drape was touching one of them. Of course, the use of electrical devices such as the electrocautery in the operating room, will completely abolish the E. E. G. pattern while it is in use.

DISCUSSION

The data presented in this report confirm the work of others regarding the electroencephalographic changes which occur in man at different depths of anesthesia. Gibbs, Gibbs and Lennox[11] showed that the brain waves under ether anesthesia resembled those of normal sleep. They evidently did not administer ether beyond that necessary to produce light surgical anesthesia. Rubin and Freeman[20] described distinct reproducible E. E. G. patterns found in cyclopropane anesthesia. Courtin, Bickford and Faulconer[5] classified the types of waves present at various degrees of ether anesthesia. Kiersey *et al.*[16] followed up the work of Brazier and Finesinger[4] on the en-

cephalograms under barbiturate anesthesia and showed a correlation with the clinical signs denoting anesthetic depth. The data in Table I concerning the blood ether concentrations required to produce the various types of E. E. G. patterns are in close agreement with those found by Faulconer.[8] The similarity of these results suggests that the electro-encephalogram may be used as a valid index of the extent of narcosis.

of anesthesia indicated by the E. E. G. may be increased by the presence of an abnormally high arterial blood CO_2 tension. If this be carried to an extreme degree, a dangerous cortical depression may result, as in the operative course of the patient illustrated in Figure 5. This patient was spared serious complications by prompt reexpansion of his lower lobe and adequate respiratory exchange. The relief of the respira-

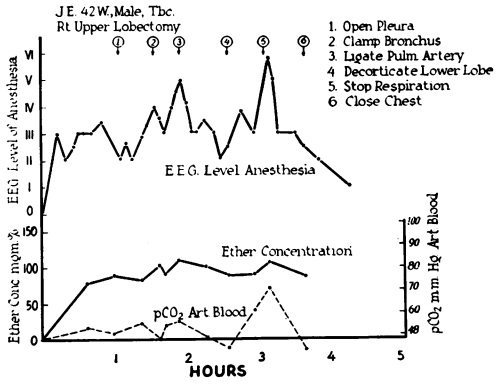


FIG. 5

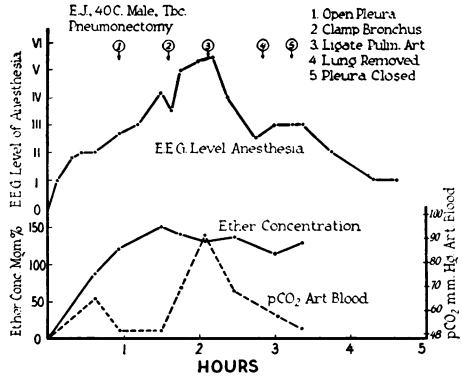


FIG. 6

FIG. 5. The progress of the brain wave pattern types during the operative course of a patient undergoing an upper lobectomy, which shows the depressant action of carbon dioxide when superimposed on a given ether level at point 5.

FIG. 6. The depression of brain activity, as reflected by the electro-encephalogram, when severe respiratory acidosis occurred during a pneumonectomy.

In view of the well known representation of the cardiovascular control high within the central nervous system, the degree of narcosis may well have a direct bearing on certain of the untoward events which occur during surgery, such as hypotension or cardiac arrest. Therefore, all the factors which enter into the sum total of narcosis become important. That high tensions of CO_2 potentiate the effect of barbiturates was demonstrated by Beecher and Moyer[2], and there is abundant other evidence of the narcotic properties of excess carbon dioxide.[19, 21] It becomes necessary, then, to consider the effects of this agent as additive to those of the anesthetic employed. The example presented in Figure 2 and the data in Table I and Figure 3 indicate that for a given blood ether concentration the depth

tory acidosis restored the level of narcosis to that expected with the quantity of ether present in his blood stream and tissues.

Normally carbon dioxide is a respiratory stimulant in mixtures up to 9 per cent. When breathed in greater concentrations, it becomes a depressant. This reversal may take place at levels of 5 per cent or less in the presence of narcotics.[7] These observations explain to a certain extent the behavior of brain waves when carbon dioxide is administered. Gibbs, Williams and Gibbs[12] demonstrated in normal men that CO_2 inhalation increased the frequency and depressed the amplitude of the brain waves. This effect tended to disappear with high CO_2 tensions. Often during light anesthesia in the experiments reported here the administration of CO_2 simply increased the brain

wave frequency. Only with difficulty could the CO_2 tension be pushed up to the point that the E. E. G. patterns resembled the types found at deeper levels of anesthesia. On the other hand, at deeper levels of anesthesia, relatively smaller elevations of p CO_2 increased the depth of narcosis, as indicated by the E. E. G. pattern. This is a reflection in the E. E. G. of the reversal of the stimulant action of even low tensions of CO_2 to a depressant one in the presence of a narcotic agent.

In animals, anoxia does not alter the E. E. G. pattern for a given level of anesthesia until the blood is severely unsaturated, 40 per cent or less (Fig. 4). This is in keeping with the findings of Davis, Davis and Thompson,[6] who found marked alterations of the E. E. G. of three normal subjects only after they lost consciousness while breathing a mixture low in oxygen. This makes it clear that cyanosis can not be considered as an adequate guide to early failure of respiratory gas exchange in the lungs.

In both animals and man the electrocardiogram was found to be a very insensitive indicator of carbon dioxide accumulation or oxygen depletion compared with the electro-encephalogram. Only when stimuli about the pericardium or hilus of the lung were present did it show irregularities in rhythm. Marked rises in CO_2 tensions were necessary to cause even slight changes of potential in the electrocardiograms of the patients observed.

This experience with the electro-encephalogram leads us to suggest that it is a practical means of indicating the depth of narcosis from any cause, and that the electroencephalograph is a useful instrument in the operating room. At present it can not supplant the anesthetist's careful observation of the clinical signs exhibited by the patient. But in difficult or protracted operations such as thoracic procedures in which respiratory difficulty is anticipated, it may serve to good purpose by giving warning of severe brain depression and collapse. In addition, it may shed further light on what parts of a given operation may be expected to give trouble from increased narcosis due to CO_2 accumulation. This is illustrated in the course of the patient shown in Figure 6. The E. E. G. pattern progressed to Type VI when the bronchus was clamped, but returned to Type III after the pulmonary vessels had been ligated.

SUMMARY AND CONCLUSIONS

1. Six reproducible electro-encephalographic patterns observed at progressively deeper levels of ether anesthesia are considered to be a valid index of the degree of narcosis present in animals and man.

2. Type I pattern is found during induction. The E. E. G. pattern then passes through Type II to Type III, which occurs in light surgical anesthesia. As further ether is administered, Types IV, V, and VI appear. The last is seen just before respiratory arrest takes place.

3. The same patterns recur at equivalent levels of anesthesia during recovery.

4. In the series of patients studied, the blood ether concentration required in the presence of normal arterial carbon dioxide tension to produce the various types of E. E. G. patterns ranged from an average of 66 mg. per cent for Type I to 129 mg. per cent for Type V.

5. Significantly less ether in the blood is required to produce a given type of E. E. G. pattern in the presence of an abnormally high arterial carbon dioxide tension than is required in the presence of a normal carbon dioxide tension. This is found to be particularly true at levels of anesthesia at or beyond which the Type III E. E. G. pattern appears. This finding, both in animals and man, suggests that the blood ether concentration and the depressant action of high carbon dioxide tensions summate their ef-

fects in producing the total degree of narcosis present at any moment of anesthesia.

6. The possibility is discussed that this additive narcotic effect of carbon dioxide may be the reason for the untoward cardiovascular reactions which may occur in the presence of respiratory acidosis.

7. Anoxia during anesthesia in animals produced little change in E. E. G. patterns until blood levels of 40 per cent oxygen saturation or less were reached.

8. It is suggested that the electro-encephalograph is a useful and practical instrument for employment in the operating room to give warning of dangerous degrees of cortical depression during operations in which respiratory acidosis may occur.

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DISCUSSION.—DR. WALTMAN WALTERS (Mayo Clinic, Rochester, Minn.): I think this is a very interesting contribution. It applies not alone to accumulation of carbon dioxide resulting from disturbance of the lung and its function, but also to myocardial disturbances.

For the last three years my colleagues—to whom Dr. Simeone referred—have been using a similar type of apparatus combined with the electrocardiogram, in certain cases in which myocardial disturbance, and as a result, accumulation of carbon dioxide seems at all likely.

I would like to emphasize its value in two cases in particular. I believe in both of these cases the results of such observations and their correction made during the course of the operations, prevented a fatal outcome.

In the first case the patient had an inoperable carcinoma of the pancreas. Cholecystogastrostomy was performed. During the closure of the incision the anesthetist reported a decrease in blood pressure, in pulse rate, a cyanotic appearance in the color of the jaundiced patient's skin and irregularity of the pulse.

A portable machine—by which the electro-encephalogram and the electrocardiogram can be seen on a screen, and also recorded on a tape recorder—was immediately attached to the patient, tracings were made, and a diagnosis was made of ventricular fibrillation.

Intravenous administration of procaine amide was immediately started and the ventricular fibrillation disappeared. The excursions of the patient's brain waves improved from the type 4 to a more nearly normal type 2, as Dr. Simeone has shown. Recovery followed.

In the second case the patient had a perforated gastrojejunal ulcer. While the incision was being made the same changes as those in the other case were noted in the patient's clinical responses, namely a drop in blood pressure, a decrease in pulse rate, and a slight degree of cyanosis.

The leads had been attached for electrocardiographic and electro-encephalographic studies before administration of the anesthetic was started. At the time the anesthetist noted the clinical changes mentioned, the young physicist, who was observing the tracings in another room, came in with the word that the electro-encephalogram appeared to show anesthesia of a fifth degree, and the electrocardiogram showed myocardial anoxia.

The patient had a history of coronary infarction six months previously, but his recovery from that and his activities seemed to indicate that he would be able to stand a surgical procedure.

The operation was terminated immediately. During the next week the concentration of hemoglobin which had been 11.5 Gm. per 100 cc. of blood was increased to 15 Gm. by transfusions. It was low in spite of the fact that he had been given two preoperative transfusions. His physical activities were increased during that week, and then we did the operation, under abdominal wall block, with intercostal block by the anesthetist and a transabdominal splanchnic block which I did.

The patient's response to the surgical procedure, which consisted of excision of the perforated gastrojejunal ulcer and gastric resection, was without incident, and recovery was normal.

The point that I want to make is this: The anesthetist is able to assist in determining the patient's progress under anesthesia by observing the electro-encephalographic and electrocardiographic tracings and, whenever necessary, intervening with whatever procedures are necessary to relieve the anoxia. I think it is important to remember that in both of these cases there was clinical evidence of abnormalities, yet the type of the dysfunction was indicated by the electrical apparatus.

DR. FIORINDO A. SIMEONE (Western Reserve University, Cleveland 9, Ohio): Dr. Dinsmore and gentlemen: I know Dr. Clowes joins me in thanking Dr. Walters for his discussion of our paper.

In our paper we did not intend to explain the nature of these changes demonstrated in the electro-encephalogram, because, although electrical activity in the brain has been known now for some three-quarters of a century, the physiologic