SYNOPTIC INTERPRETATION OF CONTINUOUS ECG AND EEG IN ANESTHETIC EMERGENCIES

KURT O. LEONHARDT, M.D.

THERE is an increasing number of publications in the literature concerning the usefulness of electrocardiographic and electroencephalographic monitoring during anesthesia. Electrocardiography is recognized as a dependable means of diagnosing cardiac arrhythmias, conduction defects and cardiac arrest. It also offers an opportunity to evaluate the effects of therapeutic measures on the heart such as the administration of drugs, the removal of excessive amounts of anesthetic agents and the correction of inadequate ventilation. However, the limitations of the method have to be realized.[1, 2] The ECG is only moderately helpful in the evaluation of metabolic disturbances of the myocardium since it can only produce circumstantial, nonspecific evidence of metabolic changes. It is even less dependable in the judgment of hemodynamic derangements. Adequacy of cardiac output, arterial blood pressure and organ blood flows cannot be safely inferred from the electrocardiogram.[2]

The encephalograph has gained acceptance as a tool of the anesthesiologist.[3] A one-channel instrument yields adequate information and is compact for the crowded operating room. The EEG is valuable for the determination and maintenance of a certain anesthetic depth, in the evaluation of new anesthetic drugs and procedures and for prognostic purposes after catastrophes.[4] In addition, it has proved helpful as an indicator of the sum of depressing and exciting influences on the cerebral cortex. The former include metabolic derangements such as hypoglycemia and anoxemia and respiratory acidosis as well as localized or generalized hemodynamic deficiencies. The main value of the method from a clinical standpoint may be to have available an objective method from which the state of cerebral blood flow and metabolism may be inferred.

These two accepted methods of physiologic monitoring have been discussed separately in the medical literature. This can be understood on the basis of their different origins and historical development. It remains for the anesthesiologist to emphasize the relationships between the ECG and the EEG.

In stressing a synoptic interpretation of these two tracings obtained from a patient presenting a special pathophysiological problem and subjected to a specific combination of drugs and trauma, we may draw support from the following consideration: Whereas the ECG is a poor indicator of the hemodynamic efficiency of cardiac action and the metabolic integrity of the myocardium, it is the EEG's virtue to present evidence of blood flow and metabolism in a vital part of the organism, the cerebrum. We may conclude that these two parameters will complement each other in the evaluation of complex emergency situations during anesthesia and operation.

This paper will substantiate this conclusion and document its clinical validity. From data obtained in 48 patients in physical status 3, 4 and 6, continuously monitored with an Epsco Anesthograph during major operation, a few examples have been selected for demonstration. The equipment is relatively simple and dependable and can be applied and controlled by one person without requiring constant attendance of a qualified team. The routine employment of needle electrodes further reduces technical problems. The evaluation of the tracing can be combined with the usual anesthetic management. The method has failed in only two instances because of technical difficulties.

Accepted for publication June 23, 1961. Dr. Leonhardt's address: The Memorial Hospital, Danville, Virginia.

Case Reports and Interpretations

Case 1. A 27 year old woman was undergoing a pneumonectomy for bronchiectasis recurring after a previous lobectomy. Anesthesia was induced with 200 mg. thiopental and maintained with halothane, using dimethyltubocurarine as a relaxant. The second line of figure 1 shows the unipolar apical precordial lead and the limb leads 1, 2 and 3 immediately before the incision. Except for sinus tachycardia these tracings were within normal limits. The patient tolerated positioning and thoracotomy well, but developed hypotension associated with bradycardia when the pleural cavity was entered. This was considered to be due to a combination of factors such as vagal reflex, diminished venous return and pre-existing reduction of blood volume. Without delay, 0.4 mg. atropine was administered intravenously and a phenylephrine drip was started. This raised the systolic arterial pressure from 60 to 90 mm. of mercury, but simultaneously caused a bigeminal pulse. As shown in the left part of the third line, the bigeminy consist of a nodal beat coupled with a ventricular premature contraction. The pressor response was short-lived and, therefore, a norepinephrine drip was substituted. This resulted in a prompt rise of the arterial pressure to 130/100 but was associated with ventricular tachycardia. At this time the surgeon had difficulties of exposure causing direct mechanical irritation of the myocardium by retractors and packs. As seen in the right tracing of the third line, the tachycardia of 160 beats per minute was caused by regularly alternating left and right ventricular foci. Immediately the norepinephrine was replaced by phenylephrine, the anesthetic temporarily discontinued, the patient moderately hyperventilated and a blood transfusion started. However, the condition was resistant to all these measures including interruption of the surgical procedure and persisted for 28 minutes. Procaine amide was given intravenously in 100-mg. doses every minute. A total of 900 mg. had been injected when abruptly a supraventricular rhythm reappeared. The left part of the fifth line reveals an activity of 70 beats per minute, originating most likely in the upper

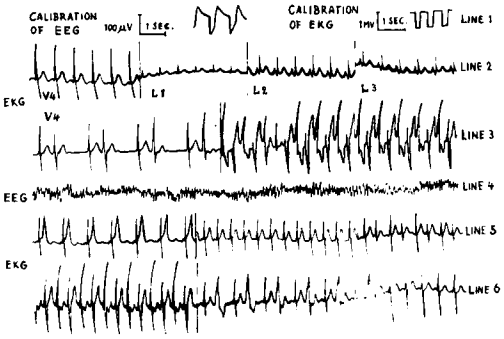

Fig. 1. *Line 1* shows calibration of instrument with standard input for EEG and ECG. *Line 2:* Various leads of normal preoperative ECG. *Line 3:* Bigeminal beats after atropine and phenylephrine. Ventricular tachycardia from alternating foci. *Line 4:* EEG compatible with Artusio's stage 1, plane 3 anesthesia. *Line 5:* ECG after disappearance of tachycardia. Note high, peaked T waves, normalizing in middle part. (Electrical alternation?) *Line 6:* Recurrent ventricular irritability. Tracing in recovery room.

AV node, with a peaked T wave of 16 mm. amplitude. This unusual feature was considered as an early sign of anoxemia or ischemia perhaps caused by a local increase in extra-cellular potassium ions released by the damaged cells.[5]

Moreover, during the one-half hour period of tachycardia the patient also developed bronchoconstriction, associated with the release of large amounts of purulent mucus, wheezing and high resistance to air flow. This condition was treated with 500 mg. theophylline ethylenediamine intravenously and repeated tracheobronchial aspirations. At this point the following problem presented itself: Can the sequence of events be explained by generalized anoxemia and hypercapnia caused by the loss of function of the collapsed right lung being removed? Would we have to revise our opinion, based on bronchospirometry, that it was functionless and only a source of infection, dead space and shunt effects? In this dilemma it was helpful to observe the EEG shown in line 4 of figure 1. It revealed an amplitude of 30 to 40 microvolts and a frequency of 16 to 17 cycles per second without signs of depression such as found in anoxia and respiratory acidosis. The tracing was compatible with Artusio's description of the

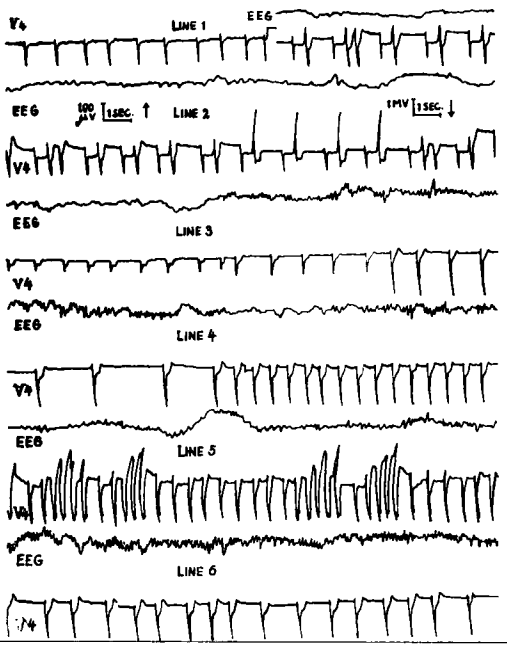

FIG. 2. *Line 1* recorded immediately after disappearance of blood pressure and intravenous injection of norepinephrine. *Line 2:* ECG shows multifocal coupled beats with marked changes of ST and T. EEG is flat with periods of silence. *Line 3:* ECG pattern of posterior wall infarction—abruptly changing to high voltage with emerging R and T waves. *Line 4:* Reflex bradycardia treated with atropine. EEG shows increasing amplitude and frequency. *Line 5:* Frequent bouts of ventricular tachycardia. *Line 6:* Tracings in recovery room.

pattern obtained in stage 1, plane 3 analgesia.[6] On this basis it was decided that the ECG changes must represent localized myocardial ischemia caused by the prolonged tachycardia. Since almost the entire cardiac cycle during this time consisted of systole with only minimal diastolic relaxation, the coronary blood flow must have been impaired.

The operation was completed, maintaining anesthesia at the same light level and taking great care not to irritate the heart in any way. After the administration of 1,000 ml. whole blood, the vasopressor was discontinued. Twenty-five minutes afterwards, the T wave became normal as shown in the middle tracing of the fifth line. The right tracing of the same line, however, was alarming again. This pattern persisted for 12 minutes and was considered as electrical alternation. More likely this represented bigeminal beats with premature beats late in diastole.[5] The EEG remained unchanged as described above throughout the operation. The lowest line depicts several more episodes of ventricular irritability, the first one being observed when a small defect in the pericardium was closed. The last part shows V_4 after completion of the procedure.

On the third postoperative day an ECG was repeated and found to be normal. The patient had a mild postoperative pneumonitis in the remaining lung and was discharged 26 days after operation in an improved condition.

Case 2. A 61 year old man with obstructive pulmonary emphysema, cor pulmonale, auricular fibrillation and several episodes of congestive failure was scheduled for a transurethral prostatic resection. He refused spinal anesthesia and general anesthesia was induced with 150 mg. thiopental followed by 15 mg. succinylcholine chloride for tracheal intubation. Anesthesia was maintained with small concentrations of halothane using a Fluotec vaporizer and a semiclosed system with a total flow of 4 liters oxygen. After 8 minutes severe hypotension developed resulting in absence of blood pressure and peripheral pulses. Clinically, there was a suspicion of cardiac arrest. Immediately he was given 0.1 mg. of norepinephrine intravenously which after one minute was followed by detection of a blood pressure of 120/100 with pulse of 70. At this time a direct writing electrocardiograph combined with a one-channel electroencephalograph was attached to the patient and the left part of the first line in figure 2 was recorded. All the tracings shown in this figure are V_4 leads and left fronto-occipital EEG's. The first tracing suggested right axis deviation and right ventricular strain. A few minutes later, as seen in the right half of the first line, coupled beats appeared composed of a sinus and a ventricular premature contraction. The ST segment following the former was depressed 2 mm. whereas the one following the latter was 6 mm. elevated. The EEG gave evidence of cerebral depression as judged by the low amplitude and slow frequency.

The second line continued the same pattern of ECG and EEG, the former showing ventricular prematures from multiple foci. In the

third line, left part, a distinct change of the ECG pattern became obvious: QRS complexes were of lower voltage, R wave disappeared and ST segments were depressed followed by negative T waves. This sequence of changes offered evidence of acute myocardial infarction, most likely of the posterior wall. This was considered to be responsible for the entire picture. The right tracing of the third line, taken 25 minutes later, indicated further changes: QRS complexes were gaining in amplitude and a short R reappeared. Then suddenly, the sixth beat of this tracing showed a high voltage of QRS and an emerging T. Following this the connections were checked and the recorder recalibrated showing no change as compared with the first calibration. Drugs were not given to the patient for ten minutes prior to this change. We believed this last alteration represented an improvement in the patient's condition reflecting increased strength of myocardial contraction and improved coronary blood flow. The EEG for the first time also showed increased amplitude and frequency reflecting improved cerebral circulation. This development was explained by the prompt initial treatment and the continuous prevention of cardiogenic shock, by partial removal of a thrombus to a smaller branch or perhaps by the establishment of a substantial collateral circulation.

The decision had to be made whether the operation should be discontinued. On the basis of our findings we decided to complete the operation. We believed that with the present monitoring facilities, supportive treatment, assisted respiration with high oxygen concentrations and a minimum of anesthesia he would have the best possible care.

Line 4 shows a reflex bradycardia developing during vasectomy. Since the patient had auricular fibrillation, this could be explained either by the presence of a nodal rhythm or by a vagal effect on atrioventricular conduction. As shown in the right tracing of line 4, the intravenous administration of 0.2 mg. atropine was effective.

Line 5 illustrates a well-known difficulty in the management of thse patients. There is controversy whether pure vasoconstrictors or drugs with a positive inotropic and chronotropic effect on the myocardium are best tolerated in cardiogenic shock.[7] In this case a mixture of 3 mg. methoxamine plus 3 mg. desoxyephedrine intravenously and intramuscularly was used to treat another hypotensive episode. The frequent paroxysms of ventricular tachycardia shown in this tracing represented a dangerous effect of the ephedrine-like drugs. Without depressive antiarrhythmic treatment the paroxysms became less frequent and ceased after 12 minutes.

The lowest tracing shows the record obtained in the recovery room immediately after operation. There were no specific or conclusive changes present in this lead. The patient had a satisfactory recovery and was discharged 24 days after operation. On the third postoperative day the ECG was repeated and revealed essentially nonspecific changes, possibly suggestive of a posterolateral infarction.

Case 3. A 6 year old boy was admitted with bronchopneumonia accompanied by severe asthma. He was given the usual treatment but showed little response. A chest roentgenogram revealed a large tumor in the anterior mediastinum which was found later to be lymphosarcoma. He was scheduled for a thoracotomy and anesthesia was induced with halothane. Intubation of the trachea was with the help of 10 mg. succinylcholine chloride and anesthesia was maintained with small concentrations of halothane and dimethyltubocurarine as a muscle relaxant. A Jefferson ventilator was used for controlled respiration.

So far the patient had been in a semi-sitting position which he preferred because of his dyspnea. He was placed supine with a large roll under the lower thoracic spine. At this time severe obstructive hypoventilation developed which could only be improved slightly by high inflation pressure and 125 mg. theophylline ethylenediamine intravenously. Auscultation confirmed our impression of lower tracheal and bilateral bronchial compression. There was cyanosis of the face, chest and upper extremities which was attributed to partial obstruction of the superior vena cava. The position was corrected as much as compatible with the surgical procedure.

Ten minutes after start of the thoracotomy

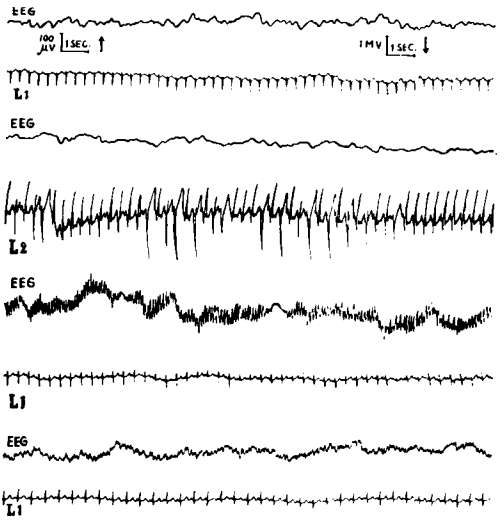

FIG. 3. *Line 1* tracings obtained during prolonged episode of obstructive hypoventilation. EEG of low frequency and changing amplitude. *Line 2:* Increasing anoxia and hypercapnia evidenced by periods of silence in the EEG and marked ventricular irritability in ECG. *Line 3:* Postanoxic "grand mal" discharge. Normalizing ECG. *Line 4:* Normal EEG and ECG at end of operation.

the first line tracing of figure 3 was obtained. The ECG showed pronounced right axis deviation and sinus tachycardia. The EEG revealed sluggish waves of low amplitude and a frequency of 7 cps. Occasionally higher and wider excursions were present without superimposed high frequency activity. This was considered anoxic depression of a halothane pattern 2 tracing.

Line 2 was recorded 20 minutes later and demonstrated ventricular irritability and a marked Q wave. The right part showed return of sinus rhythm after the administration of 75 mg. procaine amide. The EEG represented a flat, shapeless pattern, occasionally interrupted by low, wide and sluggish waves. This was evidence that the cerebral depression caused by the respiratory and circulatory impairment [8] was growing worse. During this development the blood pressure was maintained at a normal level.

Should the operation be continued as planned or should it be abandoned? On the basis of the clinical evidence, substantially aided by the EEG and ECG an impending catastrophe was diagnosed. As an emergency procedure, the incision was extended to a bilateral thoracotomy, in order to remove the tumor from the chest as soon as possible. Within 5 minutes this was accomplished followed by rapid improvement of the patient's condition.

However, 5 minutes after the decompression, when respiration and circulation were fairly normal, line 3 was recorded, showing severe changes of the EEG. A high voltage, high frequency paroxysmal cerebral dysrhythmia was present identical with the rapid spikes of a grand mal discharge. Observation of the patient revealed abortive uncoordinated muscular activity of the arms, neck and shoulders which was abolished by an additional dose of dimethyltuborcurarine. The ECG during this episode appeared normal, showing a slower rate, a larger R and a shorter S wave as compared with the first tracing. These changes were attributed to the alteration in heart position and conductivity of thoracic contents caused by the removal of the tumor.

Since there was no history of epilepsy, the convulsive discharge undoubtedly was related to prolonged cerebral anoxia. It was interesting that this convulsion did not occur during the anoxic episode but only after normal respiration was restored.

After 8 minutes the spikes diminished in amplitude and finally ceased. A flat tracing then persisted a few minutes giving way to the EEG shown in line 4. It presented a fast, low voltage pattern compatible with analgesia. The ECG normalized further by developing a larger R and a smaller S wave and the rate decreased to 120. The operation was completed and the patient had a satisfactory recovery. Convulsions or personality changes were not observed in the postoperative period.

Case 4. This 65 year old man was admitted with a bronchogenic carcinoma of the left lung complicated by pulmonary emphysema. Respiratory function tests showed a moderate to severe restrictive and obstructive hypoventilation associated with disturbances of the ventilation-perfusion ratio. It was hoped that operation

could be limited to a lobectomy. Anesthesia was induced with 200 mg. of thiopental and maintained after tracheal intubation with halothane in a semiclosed system, dimethyltubocurarine for relaxation and a mechanical ventilator was used. The patient tolerated positioning, thoracotomy and exploration fairly well.

The first row of figure 4 shows a normal electrocardiogram and a typical pattern 1 electroencephalogram as seen in halothane anesthesia. At this point it was obvious that the tumor involved a large part of the lung and that lobectomy was not feasible. The preliminary steps for a pneumonectomy were begun when the patient developed an obstructive crisis which was treated with increased inflation pressure, repeated bronchial aspirations and 750 mg. theophylline ethylenediamine intravenously. Because of this complication, it was decided to assess the function of the remaining lung. Therefore, the left main bronchus was clamped and the patient's condition observed closely for 5 minutes.

During this waiting period row 2 was recorded which revealed interesting changes: The ECG gave evidence of ventricular irritability despite complete cessation of the surgical procedure. Vasopressors were not given at this time. The most important information was derived from the EEG: Ninety seconds after the bronchus was occluded the tracing began to flatten. Periods of complete electrical silence lasting up to 10 seconds appeared, occasionally interrupted by a sluggish excursion. This was evidence of a rapidly developing anoxemic depression of the brain. It was realized, however, that occlusion of the bronchus with the left pulmonary artery intact created a shunt effect. The blood flowing through the nonventilated lung returned to the left heart as venous admixture. Therefore, the bronchus was released for several minutes to obtain ventilation of both lungs. Then the test was repeated, this time bronchus plus artery was occluded.

In the observation period following this manipulation, the third row of figure 4 was recorded. By now the ECG was normal and the EEG did not show signs of depression. It

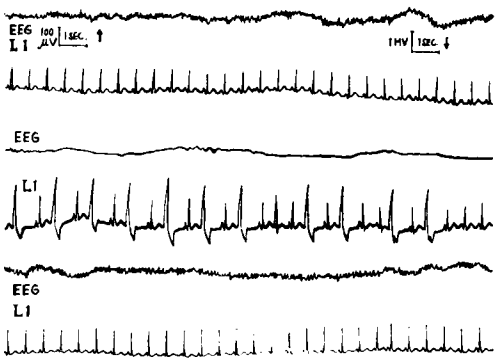

FIG. 4. *Line 1:* EEG pattern 1 as seen in halothane anesthesia. Normal ECG. *Line 2:* Left main bronchus clamped, left pulmonary artery patent. Complete electrical silence developing in EEG, ventricular irritability in ECG. *Line 3:* Left main bronchus and pulmonary artery occluded. Normal EEG and ECG.

presented a normal pattern 1 tracing of halothane anesthesia which persisted throughout the operation.

We were now confronting the problem whether a pneumonectomy should be done in the face of threatened respiratory decompensation. On the basis of the EEG tracings as an intraoperative pulmonary function test, it was decided that the patient should be given the benefit of the only possible procedure. The pneumonectomy was completed and followed by a tracheostomy to facilitate the postoperative management.

The patient required continuous mechanical assistance of breathing for several days postoperatively. Finally, intermittent use of the respirator was possible, increasing the intervals of spontaneous respiration. In the third postoperative week he developed a bleeding gastric ulcer and aspirated some of the vomitus. A pneumonitis of the remaining lung followed and the patient died 4 weeks postoperatively in respiratory failure.

Retrospectively, we concluded that a patient showing cerebral depression after bronchial occlusion in the presence of light anesthesia, high oxygen tension and controlled respirations will not tolerate a pneumonectomy.

DISCUSSION

Although a great variety of methods for the determination of physiological parameters

compete for clinical application, many of these are difficult to perform without a team of experts, require meticulous care and calibration and do not offer a well-defined relationship to the underlying disease. Nevertheless, the recording of cardiac and cerebral electrical activities has passed the test of clinical applicability. Whereas its usefulness is optimal in some problems, it is less specific in certain other conditions. However, there is substantial clinical and experimental evidence that a number of conclusions can be derived safely.[5, 10] The EEG does not produce absolute values in regard to hypoxia, ischemia, changes in the cerebral vascular bed or oxygen uptake, but it may be cautiously interpreted as the sum of all these factors as the expression of the individual's tolerance to combinations of trauma.[10]

In the cases presented the synoptic interpretation of the ECG and EEG has consistently supplied helpful information for the diagnosis, management and prognosis of these emergencies. Provided that the inherent limitations are realized, it appears justified to utilize the EEG as an indicator of blood flow and tissue metabolism in a representative segment of the body.

The interesting observation of a grand mal convulsive discharge after a prolonged episode of obstructive hypoventilation represents a contradiction to the currently accepted viewpoint. It has been stated authoritatively [9] that anoxic convulsions are caused by the normally continuing discharge of the caudal portion of the reticular formation when the latter is no longer controlled by the anoxic higher centers. These convulsions are always tonic and never clonic in nature since the clonic mechanism depends on an intact thalamo-caudate inhibitory system. In our patient, however, the seizure occurred only after a normal cerebral metabolism was restored and was accompanied by a cortical discharge. Therefore, it can be concluded that perhaps under halothane anesthesia the above statement is not valid or that a second category of anoxic convulsions may exist. This should be more correctly classified as an immediate postanoxic convulsion originating from a higher focus which was created or conditioned by the anoxic episode.

Summary

The ECG and EEG as related parameters are considered in regard to their application in the surgical patient. A synoptic interpretation of both tracings is emphasized claiming that they will, in addition to their individual usefulness, complement each other in the management of operative complications. The concept has been of help whenever the conventional signs were inconclusive, inconsistent or contradictory.

Four representative cases selected from 48 poor risk patients continuously monitored during operations have been presented. In the first case a differential diagnosis was established between myocardial ischemia due to severe ventricular tachycardia and generalized anoxemia possibly caused by the loss of function of the lung being resected. In the second case the possibility of cardiac arrest was excluded, a diagnosis of acute myocardial infarction secured and the patient's condition optimally managed while the surgical procedure was successfully completed. In the third case, which was complicated by severe bronchial and mediastinal compression, a decision to change the plan of operation was made in order to remove the cause of obstruction as an emergency procedure before serious cerebral damage occurred. In the last case the problem was confronted, whether the contemplated pneumonectomy would be tolerated in the face of long-standing bilateral pulmonary disease. A functional evaluation of the remaining lung was accomplished during the course of operation and the procedure completed accordingly.

REFERENCES

1. Sabawala, P., Gunter, R., and Dillon, J. B.: Persistence of electrical activity in heart after clinical death, Anesthesiology 18: 236, 1957.
2. Cannard, T. H., Dripps, R. D., Helwig, J., and Zinsser, H. F.: Electrocardiogram during anesthesia and surgery, Anesthesiology 21: 194, 1960.
3. Martin, J. T., Faulconer, A., and Bickford, R. G.: Electroencephalography in anesthesiology, Anesthesiology 20: 359, 1959.

4. Faulconer, A., and Bickford, R. G.: Electroencephalography in Anesthesiology. American Lecture Series, Springfield, Illinois, Charles C Thomas, Publishers, 1960.
5. Dressler, W., and Roessler, H.: An Atlas of Electrocardiography. Springfield, Illinois, Charles C Thomas, Publishers, 1949.
6. Bellville, J. W., and Artusio, J. F., Jr.: Electroencephalographic pattern and frequency spectrum analysis during diethyl ether analgesia, ANESTHESIOLOGY 16, 379, 1955.
7. Aviado, D. M.: Cardiovascular effects of some commonly used pressor amines, ANESTHESIOLOGY 20: 71, 1959.
8. Dornett, H. C., and Brechner, L.: Instrumentation in Anesthesiology. Philadelphia, Lea & Febiger, 1959.
9. Gastaut, H., and Williams, M. F.: Handbook of Physiology. Section 1, Vol. 1, American Physiological Society, 1959.
10. Gibbs, F. A., and Gibbs, E. L.: Atlas of Electroencephalography, Vols. 1 and 2. Addison-Wesley Publishing Company, Inc., 1952.

CARDIAC MONITORING Two patients operated upon for closure of atrial or ventricular septal defects had their cardiac action monitored by pacemaker electrodes affixed to the myocardium. In both patients (one during operation and one on the third postoperative day), ventricular fibrillation occurred when an electrocardiograph was recorded. Both were resuscitated and recovered uneventfully. The fibrillation was caused by an electric current arising either from imperfect insulation of the electrocardiograph, from a "standard" leak or from the capacity effect in the instrument. (*Noordijk, J. A., Oey, F. T. I., and Tebra, W.: Myocardial Electrodes and Danger of Ventricular Fibrillation, Lancet 1: 975 (May 6) 1961.*)

ELECTROCARDIOGRAM Results of the study of electrocardiograms of 1,000 patients revealed that 341 patients displayed an arrhythmia or disorder of conduction in the postoperative period. Most of the tachycardias (over 120 per minute) were directly attributable to the physical condition of the patient prior to or during operation. The electrocardiographic changes occurring in patients with hypovolemia were fairly consistent. The postoperative electrocardiogram was instrumental in discovery of 11 cases of acute myocardial infarction and two cases of metastatic disease of the heart unsuspected prior to operation. (*Schweizer, O., and Howland, W. S.: Value of the Electrocardiogram in Immediate Postoperative Period, Surg. Gynec. Obstet. 113: 33 (July) 1961.*)

VAGO-VAGAL REFLEX In a study of the cardiac asystole produced by vagal stimulation in dogs, it was found that a gradual increase in carbon dioxide content of the arterial blood, caused by respiration of 20 per cent carbon dioxide in a closed circuit, resulted in a gradual increase in the duration of asystole. The increase in asystole appeared before any marked decrease in *p*H value of the blood had taken place. It was concluded that carbon dioxide might have a direct effect on the vagal nerve endings or on the activity of the chemical transmitters liberated here. (*Mohamed, A. H., and others: Some Factors Affecting the Vago-Vagal Reflex, Amer. J. Physiol. 200: 936 (May) 1961.*)