THE ELECTROCARDIOGRAM DURING ANESTHESIA AND SURGERY

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THE ELECTROCARDIOGRAPH is being used with increasing frequency as a monitoring instrument during anesthesia and operation. Its value is unquestioned but it requires careful interpretation on the part of anesthetist, surgeon or internist. We hope this paper will increase the usefulness of the electrocardiograph for those unaware of its limitations, and will point out situations peculiar to the operating room and the anesthetized surgical patient which might be unfamiliar to the clinical cardiologist. This paper is based upon studies made in the operating room and post anesthetic recovery room during the past five years, with observations on approximately 2,500 patients. During much of this time the same cardiologist has been in attendance in the operating room and continuous observation of the electrocardiogram has been possible through the use of operating room oscilloscopes.

Basically the electrocardiograph is a sensitive galvanometer intended to record the differences in electrical potential arising in association with the heart beat. When recorded, free from artifact, this electrical activity gives precise information concerning the heart rate and its mechanism or rhythm. Various patterns have been recognized to occur in association with abnormal states of the myocardium. Under certain circumstances one can infer, from the electrocardiogram, alterations in myocardial oxygenation and can make intelligent guesses concerning effects induced by altered electrolyte balance. However, these are nonspecific changes which can sometimes be misinterpreted. One can infer that a pain is of anginal type because the typical pain is accompanied by abnormalities of the RST segment and T wave that return to normal on cessation of the attack. Similarly, one can suspect the presence of coronary artery disease when these same electrocardiographic changes are produced by a standard exercise test. At best, such applications of the electrocardiograph are subject to errors producing both falsely positive and falsely negative results.

The potentials generated within the heart are associated with the transport of sodium and potassium ions across cellular membranes.[1] Any drug, disease state or condition which affects this ionic exchange can alter the electrocardiogram. Two or more drugs or conditions may affect this ionic exchange in a similar fashion and therefore produce a similar pattern in the electrocardiogram. Because of the nonspecific nature of these ionic changes, the electrocardiogram must be interpreted in the light of known stimuli or conditions capable of producing such changes. Interpretation is empiric and judgment must be exercised in assigning a cause and effect relationship to an effect that may be produced by many different stimuli.

Under no circumstances does the electrocardiogram measure hemodynamic events such as the efficiency or force of myocardial contraction. Reasonably normal electrical activity can be present in the heart when there is no measurable arterial blood pressure;[13] we have observed this situation repeatedly. Hemodynamic events must be measured in terms of cardiac output and venous and arterial pressure; they cannot safely be inferred from the electrocardiogram. Valuable time may be lost before performing thoracotomy and cardiac massage if electrocardiographic criteria alone are used to indicate when the heart needs such support. On the other hand, we have observed most frightening electrocardiographic patterns in patients with adequate blood pressure. Severe degrees of intraventricular block

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may also be present, with very distorted electrical complexes but without hemodynamic alteration.

Within the operating room the electrocardiogram differs in two important aspects from one recorded as a routine diagnostic tracing. The first of these is the greater opportunity for the introduction of artifact. The electrocardiograph is a galvanometer intended to record only the electrical potentials generated within the heart. But it cannot discriminate between cardiac potentials and those created elsewhere and conducted into the instrument. The reader is referred to a standard text on electrocardiography [2] for a detailed general discussion of artifact. Some of the most troublesome sources of artifact in the operating room deserve special discussion.

Artifact

Extraneous repetitive electrical discharges can simulate QRS complexes and may be misinterpreted as the electrocardiogram of the patient. These have occurred from loose patient electrodes which have produced a condenser discharge effect and simulated or distorted the electrocardiogram. It is not difficult to displace an electrode beneath the drapes and record such artifacts. Patterns simulating the large Q-waves and inverted T-waves of myocardial infarction have been observed in patients when one electrode was disconnected beneath the drapes or a wire of the instrument cable was broken within the insulating sheath. Cardiac potentials of a person manually restraining a patient may be recorded simultaneously with the electrocardiogram of the patient. We have observed a sine wave arising in the pump oxygenator and conducted to the patient through the arterial and venous lines; this stimulated ventricular tachycardia and was present only when the arterial pump was turning. This artifact continued even when all electrical connections to the pump oxygenator were disconnected and the pumps turned by hand. It apparently arose in electrochemical potentials generated within the oxygenator. The sine wave was eliminated by measuring the points of potential difference and interconnecting them with a conductor (fig. 1).

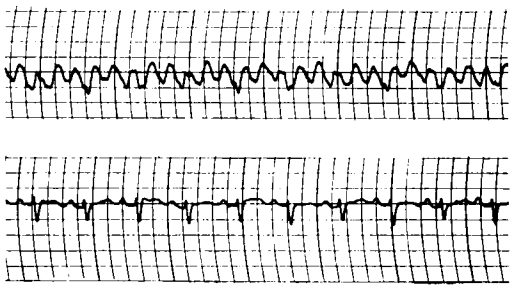

Fig. 1. Artifact from the pump-oxygenator (top tracing) conducted to the patient through the arterial and venous lines and the true electrocardiogram (bottom tracing) it obscured.

Another source of artifact is the interference picked up by the patient, the patient cable or the electrocardiograph from the power line and the strong electrical fields generated by other equipment such as X-ray, diathermy, or electrocautery machines, air conditioners, vacuum and pressure pumps, and fluorescent lights. Most of this can be eliminated by proper grounding. The reader is again referred to a standard text on electrocardiography.[2] There is no single technique by which artifact can be recognized and eliminated. Very low or very high voltage in the recorded signal and abnormal wave forms different from the patient's preoperative tracing should cause one to suspect artifact. Daily experience in watching the electrocardiogram on an oscilloscope teaches one to sense when artifact is present even when the cause is not immediately apparent.

Rapid Alterations in Patterns

Assuming a record free from artifact, the second great difference between routine diagnostic tracings and electrocardiograms recorded in the operating room is the rapidly changing configuration of the recorded signal. Because the electrocardiogram is a graphic presentation of the electrical potentials produced within the heart and conducted through its tissues, any agent or condition which affects the genesis of the electrical impulses within its pacemakers, or conduction through its tissues will alter the tracing. During anesthesia and operation the possibilities of such effects are legion and include preanesthetic medication, anesthetic agents, pressor drugs, cardiac glycosides, electrolyte imbalance, and the mechanical and

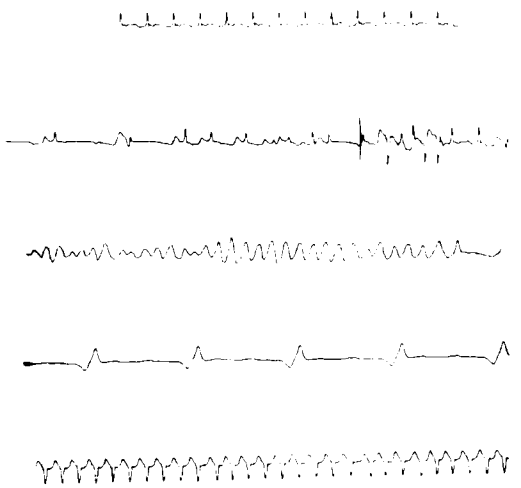

FIG. 2. The changing electrocardiogram. Top line, normal sinus rhythm; second line, ventricular irritability during digital fracture of the mitral valve; third line, ventricular fibrillation a short time later; fourth line, idio-ventricular rhythm following a single electric shock; bottom line, nodal rhythm at end of operation. In recovery room normal sinus rhythm returned.

physiologic changes associated with the operative procedure itself (fig. 2).

The T-wave is the most easily influenced complex in the electrocardiogram; smoking a cigarette or drinking cold water is sometimes enough to change its configuration.[3] Elevation or depression of the RST-segment is somewhat more difficult to produce, but can be present along with T-wave changes even when myocardial hypoxia is not apparent. To be meaningful these changes must be interpreted in the light of the stimulus producing them. Exposure of the surface of the heart to the drying action of the air and operating room lights, irritation of the surface of the myocardium by blood or irrigating fluids, the presence of retractors on the heart, and the administration of drugs such as digitalis and procaine amide have all been observed to produce these changes. During the course of a single operation the electrical complex may shift through varying RST- and T-wave patterns without obvious alteration in the patient's condition. Most changes disappear shortly after the chest is closed. Sometimes they may persist for as long as a year postoperatively. These nonspecific RST and T-wave patterns occurring during operation may simulate those associated with myocardial hypoxia or infarction and may cause unwarranted alarm in the minds of those unfamiliar with their benign nature. Their changing and nonspecific nature makes them a less reliable guide to myocardial oxygenation in the operating room than in the cardiologist's office. Despite the problems in the recognition of cyanosis, a more useful evaluation of myocardial oxygenation can be made by visually assessing the pinkness of the myocardium and the arteries and veins on its surface, or even the color of the mucous membranes, than can be made from the electrocardiogram (fig. 3).

When RST-segment and T-wave changes are associated with myocardial hypoxia, they are very meaningful and should be heeded as a warning that oxygenation must be improved. Such situations arise during the inhalation of gas mixtures low in oxygen, during respiratory obstruction, or hypotension. They may also be produced by interference with the blood supply to a portion of the myocardium as during digital fracture of the posterior commissure of the mitral valve when the posterior descending coronary artery may be occluded by pressure. If they appear just after placement of an intracardiac suture, a coronary artery branch may have been occluded and the suture should be removed. This is particularly likely to occur when plicating the mitral valve to correct mitral regurgitation or when suturing near the aortic valve.

During anesthesia and operation, the electrocardiogram is most helpful and precise in detecting disorders of cardiac rhythm. These include extrasystoles, paroxysmal tachycardia, atrial flutter and fibrillation, and ventricular arrhythmias.

EXTRASYSTOLES

The most frequent disorder of rhythm observed in the operating room is the extrasystole. It may be atrial, nodal, or ventricular in origin. Extrasystoles are for the most part insignificant and become important only when

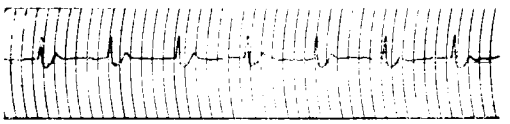

FIG. 3. Nonspecific RST-segment and T-wave changes seen during anesthesia and operation.

frequent enough to disturb cardiac efficiency to the point of producing hypotension. They may also be a warning of cardiac irritability produced by drugs, reduction of body temperature, inadequate myocardial tissue perfusion, or metabolic derangement. They are most significant when of multifocal ventricular origin. In these instances they may presage ventricular fibrillation. Their importance lies in the recognition and elimination of the stimulus producing them.

In contradistinction to most extrasystoles noted medically, those seen during anesthesia and operation can more readily be eliminated by recognizing and removing the cause. The most common cause is mechanical stimulation of the epicardium or endocardium. Hypothermia may produce irritable foci anywhere within the heart causing extrasystoles. When ventricular extrasystoles occur with increasing frequency, ventricular tachycardia or fibrillation may follow. Multifocal ventricular extrasystoles or ventricular tachycardias are consistently observed following removal of the tapes used for inflow occlusion during hypothermia for pulmonary valvulotomy and repair of atrioseptal defects. The heart is extremely irritable immediately following restoration of the circulation and remains so for several minutes.[4] All mechanical stimulation must be avoided following release of the occluding tapes until the electrocardiogram shows less myocardial irritability as indicated by disappearance of the extrasystoles and return to the control pattern (fig. 4).

Extrasystoles have been observed in normal patients and with all anesthetic techniques and agents. They are more likely to be observed in patients with diseased cardiovascular systems and during the administration of inhalational agents such as cyclopropane, halothane, ethyl chloride and chloroform. In the experience of Price and co-workers,[5, 6, 7] they increased in frequency as the blood levels of cyclopropane increased or when the blood P_{CO_2} increased while the cyclopropane blood level was constant. With halothane,[8] increasing depth did not produce extrasystoles but an elevation of P_{CO_2} did. A conducted supraventricular impulse alternating with a ventricular or nodal extrasystole is a common cause of the bigeminal pulse observed during anesthesia. We believe this may occur more frequently with cyclopropane than with other agents. If the extrasystole does not open the aortic valve, or if the pulse it produces is so weak that it cannot be palpated, an erroneously slow pulse rate may seem to be present. This is one possible explanation for the "slow pulse" frequently reported with cyclopropane. Observation of the electrocardiogram in these instances will permit diagnosis of the cardiac rhythm and "true" heart rate.

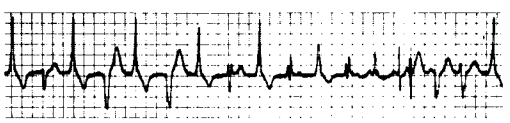

FIG. 4. Ventricular irritability following the release of inflow occlusion during hypothermia.

Extrasystoles should be treated by removing their cause whenever possible. When this cannot be done and they are adversely affecting blood pressure and cardiac compensation, lidocaine (Xylocaine), quinidine, or procaine amide (pronestyl) may be used intravenously under electrocardiographic control.

TACHYCARDIA

Tachycardia in patients scheduled for anesthesia and operation presents a challenge to the judgment of the anesthesiologist. Apprehension and anxiety are common causes of a rapid heart rate at this time. Sympathetic mediators, released through emotion, increase atrioventricular conduction and ventricular irritability. The heart rate may also increase in patients with atrial fibrillation as more effective atrial impulses reach the ventricle. The pacemaker of the heart fires more rapidly with increased sympathetic activity. So-called sensitization of the myocardium to certain inhalational agents, and to mechanical stimuli appears to follow such arousal of the sympathetic nervous system. The sensitization usually is evident in the form of arrhythmias. Preanesthetic drugs, particularly atropine and meperidine, also may produce tachycardia. There is seldom cause for alarm unless the patient has serious cardiovascular disease and it is thought that the tachycardia will strain the cardiovascular system to the point of decompensation. We fear tachycardia when cardiac

output is decreased to the point of hypotension.

When preanesthetic sinus tachycardia exists, we elect to anesthetize the patient lightly, usually with a small dose of thiopental (50–100 mg.), and observe the patient's reaction. The anesthetic must be administered under electrocardiographic control in order that a rapid rate will not be missed because of a pulse deficit. If the tachycardia persists or signs of decompensation develop, the operation is cancelled and the patient returned to the ward for further investigation and preparation. When apprehension and anxiety are the cause of the tachycardia, the heart rate usually becomes slower and blood pressure is maintained, as anesthesia is established. As long as thirty minutes of light thiopental anesthesia may be required before cardiac rate decreases below 100/minute.

A similar plan is followed in patients with atrial fibrillation and a rapid preanesthetic ventricular rate. The heart rate tends to slow under anesthesia when apprehension has been a significant contributing factor. Failure of the heart rate to slow under anesthesia suggests inadequate preparation of the patient with insufficient digitalization, or with persistent congestive failure.

The administration of additional intravenous digitalis should not be resorted to unless it is imperative that the operation proceed in spite of the signs of cardiac decompensation. Instead the patient is returned to the ward for further preparation.

Clinical judgment concerning the degree of digitalization is sometimes difficult. In patients with atrial fibrillation we have used an exercise test. With adequate digitalization, mild exercise should not increase the heart rate more than 10–15 beats from the resting rate of 80 or 90 beats per minute. The test can be several sit-ups in bed, walking the length of a ward, or climbing a flight of stairs. Atropine may be used instead of exercise. The first action of digitalis is a vagal effect which can be abolished by atropine. As much as 2 mg. intravenously may be required to block completely the cardiac vagus. Atropine, however, does not effect the control digitalis exerts on ventricular muscle. In atrial fibrillation a fully digitalized heart will not significantly increase its rate on the administration of atropine or with exercise. In patients with sinus rhythm, the cardiac rate is a less valid criterion of the state of digitalization.

We have never seen preoperative tachycardia from too much digitalis, although others describe its occurrence.[9] If digitalis toxicity is suspected, and if operation is judged imperative, 40 mEq. of potassium chloride should be infused under electrocardiographic control over a thirty minute period. Over-digitalized patients may show improvement of the electrocardiographic pattern, and infused potassium is generally harmless, provided the patient's kidneys are not seriously diseased.

Differential Diagnosis of Tachycardia. The distinction between supraventricular and ventricular tachycardia is important, since the treatment and prognosis of each is different. The diagnosis of supraventricular tachycardia (SVT) rests upon evidence that the ventricle is responding to an electrical impulse arising above the bifurcation of the bundle of His. Ventricular response to P-waves or to nodal impulses as shown by retrograde conduction of P-waves, constitutes such evidence. An esophageal electrode inserted to the level of the atrium, where P-waves of maximum voltage are recorded, may be helpful in identifying the mechanism. The slower the tachycardia, the easier it is to identify disorders of rhythm, since the electrical impulses are better separated. P-waves can often be identified by caliper measurements even though some are hidden in QRS and T-waves. At rapid heart rates, the P-waves becomes hidden in the T-waves and identification is difficult. If significant degrees of atrioventricular block or intraventricular block are present the complex may take on a saw-tooth or sine-wave appearance making the identification of P-waves impossible (figure 5).

Other criteria are helpful in differential diagnosis. Most SVT will respond to vagal stimulation. Pressure over the carotid sinus or eyeball may mechanically increase vagal tone while methoxamine, phenylephrine (neosynephrine), and prostigmine may produce the same result pharmacologically. The degree of tachycardia is sometimes of differential value. Ventricular tachycardia seldom exceeds 180

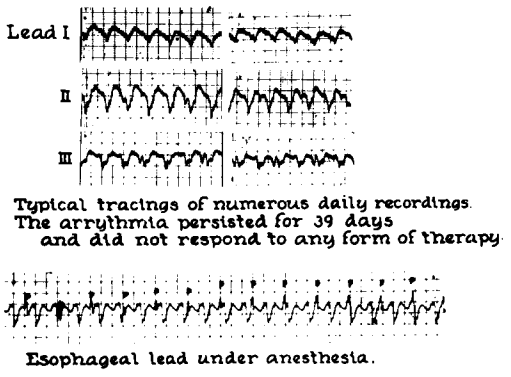

FIG. 5. Use of the esophageal electrode. Saw tooth appearance in leads I, II, and III of ventricular tachycardia. Lower tracing is esophageal lead showing P-waves which bear no relationship to QRS complexes.

beats per minute while SVT may range from 140 to 300 per minute. The most rapid rates are seen in atrial flutter with a 1:1 ventricular response.

Tachycardia is generally well tolerated in patients with normal hearts. It is more likely to cause trouble when underlying heart disease is present. Therapy is indicated for a tachycardia when it results in cardiac failure or dangerously low blood pressure. Ventricular arrhythmias are more dangerous during anesthesia and operation, since they tend to deteriorate into ventricular fibrillation, particularly in hypotensive or hypoxic patients.

Logical treatment of a tachycardia rests upon its origin and the stimulus producing it. When it begins during anesthesia and operation its cause can usually be found in the events immediately preceding its origin. The offending stimulus should be sought and removed; this may be a catheter in the heart, surgical manipulation of the heart, anoxia, hemorrhage, deep anesthesia, or the effect of drugs such as methedrine, gallamine, or atropine. Often a tachycardia will disappear spontaneously with improved oxygenation and elimination of the anesthetic.

The anesthesiologist has the two least harmful methods for the treatment of SVT at his fingertips. These are carotid sinus or eyeball pressure, and vagal stimulation by vasopressor drug therapy. Eyeball or carotid sinus pressure should be tried first on one side and then on the other. Care should be exercised to avoid injury to the anesthetized eye. If unilateral pressure is not effective, bilateral pressure should be used. Death from vagal arrest has been reported following this maneuver, but this must be extremely rare. On the other hand, we have terminated persistent supraventricular tachycardia with bilateral carotid sinus pressure when other measures have failed (fig. 6). If mechanical vagal stimulation is ineffective, either methoxamine or phenylephrine (Neosynephrine) should be injected intravenously. We have successfully used as little as 2 mg. of methoxamine in one injection in a child of five years, and as much as 20 mg. in an adult. Small doses should first be tried in adults to avoid an excessive pressor response. Methoxamine may be effective in terminating SVT without the production of a significant pressor response.

When these simple and reasonably safe therapeutic measures are ineffective, digitalis may

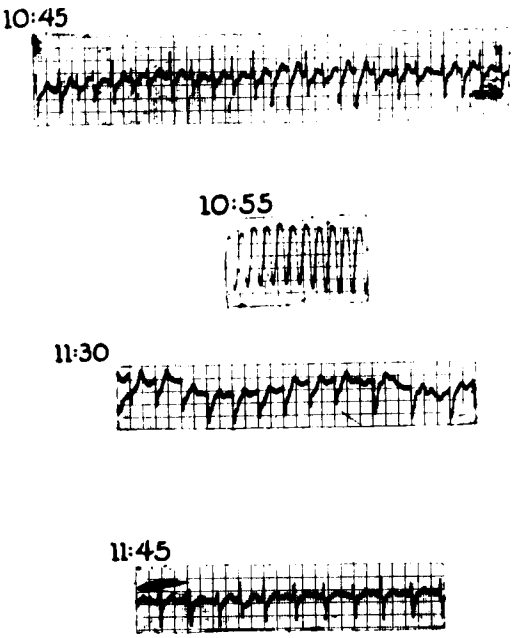

FIG. 6. Conversion of supra-ventricular tachycardia to normal sinus rhythm with bilateral carotid sinus pressure: 10:45 auricular tachycardia with block, 10:55 auricular tachycardia with rate of 300 per minute, 11:30 conversion to normal sinus rhythm with bilateral carotid sinus massage, 11:45 normal sinus rhythm after additional intravenous digitalis.

be used to terminate a SVT, if it is certain that the tachycardia is not due to digitalis toxicity. Intravenous administration of digitalis is unquestionably associated with greater hazard than oral administration. One should not attempt to terminate a tachycardia by rapid digitalization with rapidly administered increments of the total digitalizing dose, unless the tachycardia is considered a threat to the patient's life. The amount of digitalis required for digitalization should be estimated by the most experienced person available.

When doubt exists concerning the origin of the tachycardia it is reasonable to use a drug that may be effective in both supraventricular tachycardia and ventricular tachycardia. Procaine amide, quinidine and lidocaine (Xylocaine) are such drugs. They should be administered intravenously under electrocardiographic control. Widening of the QRS complex and ventricular extrasystoles are danger signs indicating the drug selected should be discontinued. These drugs are generally used to depress foci of ventricular irritability which give rise to a ventricular tachycardia.

Heart Block

Atrio-ventricular block or intra-ventricular block may develop during anesthesia and surgery. Generally, the cause is immediately apparent as in its occurrence with the placement of a suture in the heart which has interrupted impulse conduction within the bundle of His or one of its branches. Removal of the offending suture usually results in the return of a normal conduction pattern. Transient intra-ventricular block or atrio-ventricular block frequently follows digital fracture of the mitral valve and the normal conduction pattern returns soon after the finger is removed from the heart.[11]

The Dying Heart

Patients near death from any cause frequently show electrocardiograms with widely distorted complexes and slow idioventricular rhythm either of single or multifocal origin. This electrocardiographic pattern has been sufficiently characteristic to have been termed "the dying heart." A similar pattern is seen after cardiac standstill or ventricular fibrillation, when resuscitative efforts have succeeded in restoring a heart beat. One must therefore view the so-called dying heart pattern as a reflection of grave disorder which will progress to death if untreated or unsuccessfully treated.

As myocardial function deteriorates, intraventricular conduction becomes progressively delayed and the QRS complex becomes increasingly widened. The heart rate slows and various disorders of rhythm may occur including sinus bradycardia, atrio-ventricular block and sinus arrest with a nodal or idioventricular pacemaker. The last recognizable electrical activity is usually a broad sine-wave which may continue for minutes after death is apparent. The ventricle may fibrillate at any time during this period of deterioration. Hypotension usually accompanies the dying heart pattern and contributes to inadequate coronary artery perfusion which in turn leads to further myocardial deterioration.

It is often possible to reverse this pattern and resuscitate the heart. The first step in resuscitation is oxygenation of the myocardium. When ventricular contraction fails to produce sufficient pressure in the aorta, blood flow must be maintained by manual compression of the ventricles. Cross clamping of the aorta distal to the arch to raise coronary artery pressure is particularly useful when hemorrhage is the primary cause of ventricular failure. Next to myocardial oxygenation the most useful therapy in our hands has been the infusion of sodium lactate which is often specific in reducing intraventricular block.[12] When a brisk drip of molar sodium lactate does not produce results we have infused 2.5 molar lactate rapidly enough to produce the desired narrowing of the QRS complexes. This may require as much as 250 ml. of 2.5 molar sodium lactate within 5 to 10 minutes.

The simultaneous infusion of norepinephrine has been found to be helpful. The highest dilution effective in raising blood pressure and increasing myocardial tone should be used beginning with 4 mg. in 500 ml. of 5 per cent glucose in water. The rate of infusion is that required to maintain blood pressure at satisfactory levels, usually about 80–100 mm. of mercury, systolic. Care must be exercised to be certain that excessive peripheral vasocon-

striction has not obscured normal or even dangerously high blood pressures. Norepinephrine is more effective in producing a pressor effect in an alkaline medium such as sodium lactate than in an acid medium such as occurs with anoxia.[16]

We have been successful in resuscitating more "dying hearts" with the combination of norepinephrine and sodium lactate, together with manual cardiac compression and adequate myocardial oxygenation, than with any other therapy. Electric counter shock is used to terminate ventricular fibrillation when it occurs. A heart has been successfully resuscitated after as many as 52 shocks. Calcium gluconate, epinephrine, isopropylarterenol and magnesium salts have been used by others but have not proved as successful as the above treatment in our hands.

Cardiac failure with pulmonary edema may occur in patients undergoing resuscitation, particularly those with hypotension requiring prolonged intravenous pressor therapy. To avoid this complication, it is important to restrict fluid administration; this can be done by increasing the concentrations of norepinephrine and sodium lactate so that a lesser volume is infused at a slower rate. Such infusions may be required for days or even weeks, before they can be reduced and discontinued. On occasion, hydrocortisone has apparently improved the response to pressor therapy. Digitalis may also be required in patients with failure.

Comment

A new species of electrocardiographer is emerging. He is developing among the anesthesiologists and surgeons who have learned to monitor the heart beat during anesthesia and operation. Many of them have had no formal training in electrocardiography. They rediscover many of the artifacts and pitfalls described in the past with which they were unfamiliar because of inexperience. They are likely to demand more information from this instrument than it can give and to use it in situations where it is not helpful.

On the other hand, the average cardiologist is frequently at a loss in the operating room. He is not familiar with the anesthetic agents used nor with the rapidly changing effects of multiple stimuli upon the heart during anesthesia and operation. His classical training gained by electrocardiographic interpretation under static conditions is not transferable without modification to the dynamic situations of the operating room; but his knowledge is most useful.

These two species of electrocardiographers are meeting daily in the operating room and contributing to each other's development. They both belong on the surgical teams contributing to our knowledge of the diagnosis and treatment of cardiovascular disease. Team work between them is essential as they both learn the significance of electrocardiographic changes during anesthesia and operation.

Summary

Experiences gained in monitoring the electrocardiogram during anesthesia and operation, over the past five years, have been presented. These tracings differ from a routine diagnostic electrocardiogram in two ways: (1) the increased opportunity for artifact recording and (2) the rapidly changing configuration of the wave forms comprising the electrocardiogram. Common forms of artifact and wave form changes have been discussed. The concept that the electrocardiogram cannot measure hemodynamic adequacy has been stressed.

Common changes observed in cardiac rhythm during anesthesia and operation have been presented. Extrasystoles are generally considered benign. The differential diagnosis of supraventricular and ventricular tachycardia have been presented and their treatment discussed. The problem of preanesthetic tachycardia has been related to the adequacy of preoperative preparation and the patient's emotional status. The management of this problem has been presented. Our experience in the treatment of the "dying heart" with molar sodium lactate, norepinephrine and adequate myocardial oxygenation has been presented and discussed.

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NERVE BLOCKING In 1851, Pravaz invented the hypodermic syringe; in 1853, Wood developed the hypodermic needle; in 1884 Koller introduced the local anesthetic cocaine; and within a year Halsted had performed over a thousand operations under regional anesthesia. Therapeutic nerve blocking refers to the injection of appropriate solutions along the course of segmental or autonomic nerves. Blocks of greatest value are those which eliminate reflexes arising as the result of trauma or disease. These may be due to spasm of skeletal muscle or pain involving joints and posttraumatic conditions and pain due to certain vascular conditions. Patients with pain due to carcinoma are difficult to treat as the pain is hard to localize and relieve with nerve block. (*Rovenstine, E. A.: Nerve Blocking, Postgrad. Med. 26: 583 (Nov.) 1959.*)

INTERCOSTAL BLOCK Postoperative incisional neuralgia has prompted the use of intercostal nerve blocks for prophylaxis and therapy. A critical evaluation of this block for the prevention and treatment of this painful syndrome following thoracotomy is long past overdue. The concept of breaking a pain cycle by anesthetic interruption of the nerve pathways is whimsy. The long-acting anesthetic agents add little as their effect is measured in hours or days while the complication seemingly goes on forever. Diligent study of excised intercostal nerve fragments has not established a recognizable nerve disease. The greatest indictment against intercostal block for the treatment of incisional pain is response of the patient. The majority of patients who undergo such block reject the idea of a second or third treatment because the "rebound pain" exceeds the original subjective pain. (*Effler, D. B.: Failure of Nerve Block in Postoperative Intercostal Neuralgia, Surg. Gynec. & Obst. 109: 516 (Oct.) 1959.*)

SPINAL ANESTHESIA Two patients had a subdural hematoma after spinal anesthesia. Diagnosis of subdural hematoma should be considered in patients with persistent post spinal headache who develop confusion, dementia, somnolence and other neurological disturbances. (*Welch, K.: Subdural Hematoma Following Spinal Anesthesia, A. M. A. Arch. Surg. 79: 49 (July) 1959.*)