MANAGEMENT OF CERTAIN COMPLICATIONS THAT OCCUR DURING ANESTHESIA

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I approach with much diffidence the subject that has been assigned me; from the literature, I am fully aware of the considerable number of complications that arise during anesthesia in even the foremost institutions of this country, as well as in the most advanced in foreign lands. While a complete coverage of the subject would be far beyond the scope of any one brief article, a résumé of my own and of others' experience in preventing or correcting certain complications that, from the literature, obviously occur many times under varying circumstances may prove of interest.

CONVULSIONS

Among the most commonly met complications are the so-called convulsions that occur during anesthesia. While these were described as "ether convulsions" in the literature of some sixteen years ago, when the anesthetic in general use was ether, experience with later technics of anesthesia has revealed the fact that these convulsive seizures occur when other anesthetic agents as well are used.

To provide a background for evaluating the complication, I will briefly review some of the early literature on the subject. In 1932 Daly1 of England reported the case of a patient in whom, after thirty minutes of anesthesia, during closure of the peritoneum, convulsions developed in the face and jaw and spread to the rest of the body. Administration of the anesthetic was discontinued, and carbon dioxide and oxygen were administered to hasten the elimination of ether; but the convulsions continued, and closure of the abdomen was effected with great difficulty between the spasms. Because the patient's face was flushed and congested, the head of the table was raised with the object of relieving the congestion. The convulsions ceased at once and did not recur.

That same year, Blomfield2 of England published an appeal for information concerning the cause and correction of "generalized" convulsions during ether anesthesia, which he described thus: "The attack generally begins with twitchings of the face muscles, when the patient is deeply under the anesthetic. The twitchings are followed by spasmodic contractions of the limbs and of most of the muscles of the body—leading to cessation of the operation, and in many instances ending fatally either on the operating table or later. While some of

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the patients have been suffering from acute infection, others have been in normal health. These attacks must not be confused with the 'fine ether tremors' that are a familiar phenomenon in light ether anesthesia, nor with the jactitations that may occur under ether when there is improper limitation of oxygen."

In his published request Blomfield\textsuperscript{2} wondered if anesthetists in the United States had observed similar convulsive attacks during ether anesthesia, which, he said, seemed not to have occurred in England until recent times (i.e., in 1932) and for which, he said, no satisfactory explanation had been forthcoming. He reported that repeated clinical examination of the ether used yielded negative results, and he commented that convulsions form such a puzzling as well as a dangerous phenomenon that his committee was anxious to collect all information that might lead to an explanation for their occurrence.

The following year (1933) Kemp\textsuperscript{3} of Canada pointed out the lack of a satisfactory explanation for the vigorous clonic muscular contractions that sometimes occur during ether anesthesia and that start with twitchings about the face and often extend to the muscles of the arms and even to those of the abdomen. He cited a case in which, after forty minutes of intratracheal ether-oxygen anesthesia, twitching of the face developed during closure of the abdomen. This seizure he assumed to be due to hyperventilation. He therefore practiced rebreathing to collect the patient's own carbon dioxide and administered oxygen through the anesthesia machine. These procedures checked the twitching of the facial muscles. When the same patient was operated upon again at a later date, twitching of the facial muscles developed fifteen minutes after anesthesia was started; these twitchings were again controlled by rebreathing (collecting the patient's own carbon dioxide) and by covering the face with an ether mask and damp cloths to effect a semi-open system. When the rebreathing was discontinued, twitchings recurred. Kemp postulated that the clonic muscular movements, sometimes seen in nonepileptic individuals under open intratracheal anesthesia, are due to general alkalosis, induced by the elimination of carbon dioxide with a consequent severe upset of the normal respiratory quotient. Noting that, if tetany developed in a patient during operation under ether anesthesia, tetany would develop in that patient if operated upon again at a later date, he concluded that the onset of tetany under ether anesthesia is due to factors inherent in the patient himself.

In 1936 Raab\textsuperscript{4} of New York described convulsions occurring during ether anesthesia. That same year Payne\textsuperscript{5} of London commented on the lack of medical literature on the subject of generalized convulsions that may occur in the course of ether anesthesia; he considered this phenomenon of serious importance, since it had occurred in no less than 200 cases during a ten year period, 1926 to 1936, and for which he stated a mortality rate of 22.2 per cent.

In 1937 Lundy\textsuperscript{6} of Rochester, Minnesota, stressed the importance

\textsuperscript{2} Blomfield, J.: loc. cit.
of this problem of convulsions or spasms associated with anesthesia, which, he stated, were occurring with increasing frequency. While citing some thirty-three possible causes or significant factors involved in convulsions, he pointed out that, whether the etiology of a condition is known or not, the important thing is to know how to combat it when it arises. He suggested more careful choice of preliminary medication and of the anesthetic agent. He also proposed the intravenous injection of a soluble barbiturate for symptomatic treatment to control the convulsions and to reduce the fatalities, which, he stated, were more common than had been realized. He gave a mortality rate of 18.9 per cent at that time (1937).

In the Department of Anesthesia at Barnes Hospital during an eighteen year period, 1930 through 1948, convulsive seizures developed in fifteen patients during anesthesia. The details of some of these cases may be interesting as a background for the corrective procedures that I will outline later in this article.

**Case 1.**—In 1930, during the course of an appendectomy being performed with the patient under nitrous oxide-oxygen-ether anesthesia, administered by the semi-open (valvular mask) method, progressive convulsive seizures developed during closure of the peritoneum. These started on the left side of the mouth and progressed to the left eyelid, left arm, and left leg. They then became generalized and violent; they were clonic in character and lasted for several seconds. The patient’s temperature increased to 104 F. Fluids were administered, alcohol sponge baths were given, and oxygen was administered by the mask method. The seizures lasted for twenty-four hours, after which there were no recurrences. The patient was discharged from the hospital in two weeks.

**Case 2.**—The following year (1931), during the course of a hysterectomy and associated bilateral salpingo-oophorectomy being performed on a woman, aged 40, the second case of convulsions occurred. Two hours after the anesthesia had been started—nitrous oxide-oxygen-ether, administered by semi-open (valvular mask) method—facial twitchings developed in the same sequence that had been noted in the patient of the year before. The administration of the anesthetic was immediately discontinued, and oxygen was administered with 5 per cent carbon dioxide at intervals. Caffeine sodium benzoate, glucose, and blood transfusion were given, but the convulsive seizures continued. The patient died one hour and ten minutes after administration of the anesthetic had been discontinued or three hours and ten minutes after the anesthesia had been started.

**Case 3.**—In 1933 the third case of convulsions occurred in an adult male patient who had been admitted to the hospital with a gunshot wound. An abdominal exploration was being performed under spinal anesthesia when rhythmic jerking of the head developed. Inhalation anesthesia with nitrous oxide, oxygen, and ether was administered for twenty minutes until completion of the operation. The muscular twitching progressed to involve the entire body until the patient’s death one hour and forty minutes after the spinal anesthetic had been given and fifty-five minutes after inhalation anesthesia had been started.

**Case 4.**—During the year 1937 we had the fourth patient in whom convulsions developed during anesthesia. The patient, aged 27, was undergoing an appendectomy with drainage; the anesthetic agents were cyclopropane-oxygen-ether, given by the carbon dioxide-absorption method. After anesthesia had been under way one hour (forty minutes after the incision was made), convulsive seizures developed and followed the same course noted in the previous cases. Temperature increased to 41.8 C (axillary). Ice packs were applied, and calcium gluconate, saline solution, luminal, and oxygen were administered. The patient died two hours and twenty-five minutes after the induction of anesthesia was started.

With these unhappy experiences before us, we realized the desperate necessity for discovering some method of preventing the development of convulsions. The literature did not contain adequate information. We had tried the procedures that had been proposed but had
found them ineffective in the cases at hand.

The first important step forward revolved around recognition of the fact that the convulsive seizures generally developed during closure of the peritoneum and were presumably associated with reflex stimuli consequent to that procedure. We also noted that during, or as a result of, the convulsive seizure, the temperature increased in every case, in one case reaching 104.5° F. In view of this association of temperature increase with the onset of symptoms in each case, it was postulated that this type of convulsion was perhaps related to a corollary identifying disturbance of the heat-regulating mechanism. By then we had identified symptoms that we thought were characteristic and that would give the alert anesthetist adequate warning of the advent of an attack. With such warning the anesthetist could be prepared to prevent its development if corrective procedures were devised and if methods of applying them were at hand. The remedial procedures developed at that time (1937), and used by us since then, have proved quite effective both as preventive and as corrective measures. Since adopting them, we have not had a single fatality from convulsions. These procedures are as follows:

The preanesthesia temperature is carefully noted. If pyrexia, even of ½ degree, is present, an ice bag is placed under the nape of the patient’s neck immediately after he loses consciousness, and administration of fluids is started at once. These precautionary measures usually prevent a further increase in temperature. Oxygen want is strictly avoided (as it should be under any circumstance), and it is verified that the carbon dioxide-absorption power of the soda lime is adequate.

Even if no pyrexia is present before anesthesia, the anesthetist is continually on the alert during anesthesia for the characteristic symptoms of preconvulsive seizures. At frequent intervals, about every ten minutes, the temperature is taken; with the development of the slightest elevation of temperature, the previously described measures are carried out. Since the described procedure has been put into routine effect and rigidly observed at the Barnes Hospital, we have not had a patient in our clinic in whom the violent convulsive seizures, described in the literature as “ether convulsions,” have developed during anesthesia.

If the warning twitching around the mouth and eye (left side) does develop, a 2½ per cent solution of pentothal sodium is administered intravenously immediately, at a rate not faster than 1 cc. per minute. The twitching is usually under control after 2 or 3 cc. has been administered; rarely is it necessary to give more than 5 cc. of a 2½ per cent solution, the dosage range being 5/100 to 12/100 Gm. I repeat, however, that the alert anesthetist will not permit the condition to develop to the point that the convulsive seizure occurs.

The following symptoms are characteristic of approaching convulsive seizures during anesthesia: an elevation of temperature of from ½ to 1 degree, associated with an elevated blood pressure; and tugging or pulling of the muscles of the throat, which can be readily detected by the hand of the anesthetist in supporting the jaw. Cyanosis is not present, nor have we observed any characteristic change in the rate or depth of respiration, nor any appreciable change in the rate or volume of pulse.

These symptoms of approaching
convulsions develop from one half to one hour before convulsions develop. If these early warning symptoms are not recognized, there will be a gradually progressive development of a more ominous nature. The temperature will progressively increase to 102-104 F.; the tugging or pulling of the throat muscles will become more pronounced. It is important to remember that, if this latter symptom were considered only by itself, it could be mistaken for a reaction to an overdose of the anesthetic (ether). On the basis of such a mistaken interpretation of the cause, the ether tension would be lowered and additional oxygen administered; these procedures, while indicated for overdosage of the anesthetic, would not correct the condition at hand if it were in reality a warning of an approaching convulsive seizure and not a symptom of overdosage. In the case of such misinterpretation of the cause and consequent attempts to correct it, further symptoms would develop, namely, twitching of the muscles of the left side of the mouth, progressing to the left eye, the left extremities, and on to generalized convulsive seizures of great intensity, which would interfere with the operative procedure and even possibly terminate fatally.

It is also very important that the symptoms warning of the approach of the convulsive seizures referred to here (those possibly related to a disturbance of the heat-regulating mechanism) should not be confused with the symptoms that warn of approaching convulsive seizures due to oxygen want. These latter are: (1) rapid, shallow respiration, becoming jerky and irregular, followed by gasping, slow respiration; (2) general tonic muscular rigidity, jactitations, and opisthotonus; (3) elevated blood pressure with rapid, weak pulse; (4) markedly dilated pupils without reaction to stimulation by light; (5) cyanosis with arterial blood of venous color.

If such an inexcusable condition should be permitted to develop, it can be corrected by the administration of oxygen and manual compression of the breathing bag and the maintenance of free respiratory exchange.

**Cocaine Poisoning**

Let us now turn to a different type of complication that not infrequently faces the anesthetist for correction, although she is usually not present when the unfavorable reaction begins. I refer to those eventualities that develop following the use of cocaine or of one of its derivatives, such as pontocaine, procaine, nupercaine, and butyn. The use of cocaine or its derivatives is advocated by some authorities to prevent reflex closure of the glottis during the introduction of an intratracheal catheter. Some of the champions of the drug even narcotize the larynx and throat with cocaine to the extent that an intratracheal catheter may be introduced without administering any inhalation anesthetic whatever.

Procaine or other cocaine derivatives are used by some anesthetists as a throat spray in preparation for a bronchogram or bronchoscopy. The drug also is used locally in nasal surgery. The injection of procaine into the heart has been advocated for cardiac arrest, as has the intravenous use of procaine for the prevention and treatment of cardiac arrhythmias during anesthesia and as a protection against ventricular fibrillation. Reports concerning these intravenous uses of procaine are very conflicting.
Cardiac arrhythmias and cardiac arrest, occurring during thoracic operations, remain a dreaded and occasionally fatal catastrophe, and many clinical observations have been made during these episodes. However, despite the considerable amount of investigative work done on the problem, there is even now no unanimity of opinion as to the cause or the treatment. According to one group, manipulation of the lung root or any segment of the heart or great vessel is likely to set up vago-vagal reflexes that touch off an arrhythmia or cause cessation of the heartbeat. It is this group that believes that great benefits derive from the use of procaine intravenously, as well as from the topical use of cocaine or pontocaine, both as a prophylactic and as a curative measure.

At Barnes Hospital there is considerable skepticism in regard to the practical importance of vago-vagal reflexes. Graham, on the basis of an extensive experience in thoracic surgery and dog experiments, doubts the existence of true vago-vagal stimuli as a modus operandi of cardiac arrhythmias and arrest. Burford believes that myocardial ischemia is a more likely mechanism and doubts that arrest or serious irregularities occur in hearts previously undamaged by disease. In the few cases of arrhythmia and/or arrest that have occurred on the Chest Service at the Barnes Hospital, Graham and Burford have contended that cardiac massage is the one dependable method for restoring the beats; and that, if this measure fails, no adjunctal expedient, including electrical stimulation, is likely to be of benefit. They place small reliance on the administration of pontocaine locally or of procaine intravenously. Instead, they stress smooth anesthesia with maximal oxygenation and gentle operative technics.

While there is no question that cocainization prior to intratracheal intubation facilitates the intubation procedure, the practice is considered by some to be accompanied by considerable danger. It is therefore strongly urged that, instead of using cocainization as a preparation for intubation, a general anesthetic be administered to secure a plane of anesthesia that will abolish the afferent impulses, which under too light a plane of anesthesia are active and interfere with introduction of the intratracheal catheter.

In our institution the spraying of the throat of a patient scheduled for a bronchogram or bronchoscopy was from time to time attended by a high incidence of convulsions and other symptoms before we adopted the practice of routinely administering nembutal prior to the cocainization. However, even after these precautionary measures had been inaugurated, untoward symptoms developed all too frequently.

Many years ago, two deaths occurred in our clinic in patients cocainized for nasal surgery, and one death occurred in a patient prepared for a bronchogram. In all three cases, the patients suddenly stopped breathing, were pulseless upon the arrival of the anesthetist, and failed to respond to resuscitative measures. A recent fatality occurred in a patient who had been prepared by local infiltration block for an abdominal operation. Immediately after the injection of 1 per cent novocain, the patient's head began to jerk, and a generalized convulsive seizure developed of such great violence that pentothal
sodium could not be administered intravenously, and, although an intratracheal catheter was introduced and oxygen given through it by manual compression of the breathing bag, pulse and blood pressure became imperceptible, and the patient was pronounced dead twenty minutes after the novocain injection. Several cases of convulsions occurred in patients whose throats had been sprayed with procaine prior to bronchoscopy; typical symptoms of cocaine poisoning developed, namely, shortness of breath, resistance, restlessness, fast pulse, rigidity, anxiety, muscular twitchings, elevated blood pressure, and pallor, followed by cyanosis. In each of these cases, however, the symptoms were controlled by administration of pentothal sodium plus oxygen, and in each case bronchoscopy was performed without further incident. All of these patients had previously been prepared with 0.09 Gm. of nembutal administered an hour before bronchoscopy. Waters and Gillespie\(^9\) reported “several deaths after topical application of a drug to the pharynx,” but as no member of the department of anesthesia was present when the deaths occurred, no records were available.

Since the anesthetist is called upon to assist in emergencies that arise after cocaine administration, she should familiarize herself with the symptoms of cocaine poisoning and the method of treating them, as well as with precautionary measures that may be inaugurated prior to cocainization. The symptoms of cocaine poisoning and poisoning from its substitutes are similar: dizziness, marked pallor, dilated pupils, nausea, vomiting and abdominal pain, finally delirium, dyspnea, slow weak pulse, and convulsions. Fatal cases run a very rapid course, with anxiety, sudden falling, extreme pallor, dyspnea, sometimes brief convulsions, arrest of respiration, and death. The prior administration of a barbiturate diminishes the risk by minimizing the cocaine convulsions and their effects when they develop.

In our clinic 0.09 Gm. pentobarbital sodium (nembutal) is administered two hours before the operative procedure. Alternative recommendations that have been made are: 0.2 Gm. phenobarbital or 0.6 Gm. sodium barbital one hour before operation.

If symptoms of cocaine poisoning develop, the patient’s head is lowered, and the convulsions are arrested by the intravenous injection of pentothal sodium. Artificial respiration is administered (usually an intratracheal catheter is introduced), and cardiac massage is instituted if the circulation fails.

**Complications during Thoracic Operations**

I shall refer briefly now to procedures associated with the conduct of thoracic operations. It is axiomatic that, first, adequate pulmonary ventilation must unfailingly be maintained at all times and, second, that the possibility is always present that, as a result of a manipulative procedure, lung exudate may be released into the tracheobronchial tree.

Accordingly, at Barnes Hospital, where there is a major clinic in thoracic surgery, anesthesia procedures have been set up, the details of which may be interesting.

Immediately after introduction of the intratracheal catheter, suction is effected through it. This suction is again thoroughly effected.

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just prior to turning the patient from the supine to the lateral position. Suction is again promptly effected through the catheter immediately after the patient is turned, if indicated.

It is to be remembered that, after the incision has been made and during rib resection, there is likely to be a release of purulent material into the bronchial tree. Accordingly, it is a routine procedure with us to use suction at that time, as well as during the opening of the pleura. Again, during actual manipulation of the lung, there are periods when excessive material may be forced into the respiratory tract. At such times the tracheal catheter is routinely suctioned.

Throughout the operation, the anesthetist is on the alert for moist sounds; should they develop, she does not hesitate to ask the surgeon to interrupt the operation to permit needed suction. In cases of suppuration it is especially necessary that suctioning be done at frequent intervals. While these suctionings may interfere with and delay the operative procedure, it must be realized that any secretion permitted to be present in the air passages will interfere with the gaseous exchange in the lung and, if not removed, may lead to severe anoxia, with its accompanying detrimental effect on cardiocirculatory functions.

Upon completion of the operation and after deanesthetization, the patient’s mouth is thoroughly suctioned, and the suction catheter is introduced into the trachea to aspirate secretions from the lung, as well as normal secretions or mucus that may be present in the trachea. After this procedure the normal cough reflex is usually present and aids in the avoidance of postoperative pulmonary complications.

From the foregoing, it will be apparent that we consider it imperative for the anesthetist to have at hand at all times during anesthesia a properly functioning suction apparatus that may be instantly used if and when needed. I repeat for emphasis that, if secretory or mechanical obstruction is permitted to exist, various degrees of anoxia will supervene, with consequent depression of the central nervous system and resultant hypertension and tachycardia during anesthesia, as well as postanesthesia pulmonary complications, such as atelectasis and bronchopneumonia.

Complications Caused by Reflexes

I cannot leave the subject of complications of anesthesia without briefly referring to those that revolve around anoxia caused by laryngeal and other reflexes, which interfere with untrammelled respiratory exchange and which, if permitted to continue, eventuate in myocardial exhaustion, cerebral damage, and even death.

As Brewer and Luckhardt pointed out, laryngeal reflexes are divided into two classes, first, those that originate in the larynx itself and, second, those that result from stimulation of afferent nerves elsewhere in the body. An illustration of this latter reflex action is the cessation of respiration—blocking off of respiration by approximation of the vocal cords—that results from traction on the splanchnic viscera during an abdominal operation, if the plane of anesthesia is permitted to become unnecessarily light for the surgical procedure be-
The vital importance of maintaining anesthesia at sufficient depth to avert the superimposition of such traction-stimulated, reflex closure of the glottis must be recognized, and such complication inviolably avoided. Another illustration of the occurrence of laryngeal reflex because of too light anesthesia is the severe laryngeal spasm, with complete adduction of the vocal cords, that occurs if surgical intervention is undertaken too early, i.e., before the patient has fully reached the plane of anesthesia necessary for the procedure at hand.

Obviously, different surgical procedures call for different planes of anesthesia for their safe execution. Different stages of a given operation also require different planes of anesthesia. In my experience, one of the best ways of verifying that the plane of anesthesia is proper for the procedure at hand is the change in character of the respiration, which clinically manifests the various zones during anesthesia. During the induction period respirations do not vary greatly from normal, but their change in character with loss of consciousness and supervision of the second stage must be studied critically if the characteristic increase in rate and in depth that is typical of entry to that stage is to be identified. This change is easily noted when the anesthetic agent is ether, but it is less noticeable when the anesthetic agent is one of the quicker acting gaseous agents, nitrous oxide-oxygen or ethylene-oxygen. When the anesthetic is cyclopropane-oxygen, this increase in rate and depth of respiration is neither marked nor of long duration, so that the anesthetist must be especially alert to identify and interpret its occurrence. After the increase in rate and depth of respiration that occurs at this point, a characteristic type of respiration develops, which, for lack of any formal description, I describe for my anesthesia students as “superficial and hesitant in character, with a slightly shorter inspiratory phase and a slightly longer expiratory phase and with a slight pause between those inspiratory and expiratory phases.”

The anesthetist must not misinterpret this characteristic “superficial hesitant” respiration as the “unequal inspiratory and expiratory phases” of respiration that indicate approaching overdosage; such misinterpretation would result in the lightening of the plane at this point with activation of the vomiting reflex to produce vomiting, with the danger of aspiration of the stomach contents into the trachea, possibly leading to dire consequences. Nor should surgical intervention be undertaken at this stage of anesthesia, because of the reflex closure of the glottis and laryngeal spasm that would result.

After successful transition through the “superficial hesitant” respiratory phase of anesthesia that I have just described, a type of respiration develops that is indicative of the establishment of the surgical plane of anesthesia. This is characteristically automatic, rhythmic, and machine-like, with the inspiratory and expiratory phase of nearly equal length and with no noticeable pause between these two phases.

A pharyngeal airway and intratracheal catheter of appropriate size should always be at hand and used if needed to insure mechanically unobstructed respiratory exchange. An anesthetist’s boast of seldom using an airway is a confession that that anesthetist is not alert
to the importance of this principle of anesthesiology and of the damage that can result from violating it.

**Complications During Upper Abdominal Operations**

In concluding this article, I shall make some recommendations concerning the avoidance of some crises that are reported to arise during anesthesia for upper abdominal and thoracic surgery. At Barnes Hospital routine procedures for caring for patients undergoing these types of operation have reduced to a minimum certain complications that we know from the literature otherwise occur frequently.

The surgical procedures to which I refer are gastric and intestinal resections and operation for duodenal perforation, intestinal obstruction, etc. One of the untoward circumstances I have in mind is the ejection of the stomach contents into the mouth and respiratory tract during anesthesia. I have also in mind the release of lung exudate into the tracheobronchial tree when manipulative procedures are being performed during thoracic operations; the presence of mucus in the upper respiratory tract as the result of the secretion of saliva during early stages of anesthesia; and the presence of a plug of mucus in the bronchus following anesthesia.

To reduce to a minimum the occurrence of such complications, the Barnes Hospital Department of Anesthesia has set up certain techniques that have proved quite effective.

Every patient scheduled for a gastrointestinal operation has a duodenal tube introduced prior to the administration of the anesthetic. Usually this tube is in place when the patient arrives in the operating room. If for any reason this procedure has not been executed, the anesthetist requests that she be permitted to introduce such a duodenal tube. Generally the surgeon is most appreciative of the suggestion.

Then, prior to the administration of the anesthetic and while the patient is still conscious, the duodenal tube is suctioned thoroughly. Experience has shown that, even though a duodenal tube is in place and draining, as much as 1,000 cc. of fluid, and usually 500-700 cc., may be obtained by suction. After suctioning, the open end of the duodenal tube is placed in an emesis basin, which has previously been strapped securely to the operating table by means of adhesive, so that the pan will not fall to the floor at an inopportune time during the operation.

The patient is then given pentothal sodium intravenously until he loses consciousness, after which, an inhalation anesthetic is administered. As soon as the eyelid sign is abolished, an intratracheal catheter is introduced orally under direct vision. The balloon on the catheter is inflated to the proper tension, causing the sides of the balloon to press gently against the walls of the trachea, thereby preventing any fluids in the mouth from invading the respiratory tract. The anesthetic of choice is then administered by the carbon dioxide filtration, closed circuit technic.

The duodenal tube is frequently suctioned during the operative procedure, especially if there is considerable fluid flowing from the tube. This is important, because during manipulation of the digestive tract, the amount of fluid forced out through the esophagus is sometimes greater than can be carried away by the duodenal tube. By fre-
quent suctioning, however, the amount of fluid in the digestive tract is kept at a minimum, thus reducing the possibility of digestive fluids being suddenly forced into the mouth.

While a patent airway is assured by the presence of the intratracheal catheter, the mouth must be kept free of any digestive fluid in preparation for the time when the catheter will be removed and when some fluid in the mouth might reach the respiratory tract in spite of the fact that thorough suctioning has been done.

While we have for many years used the intratracheal technic of anesthesia for gastrointestinal surgery, only during the past few years have we taken the detailed precautionary measures described. I cannot stress too emphatically the importance of following these comparatively simple procedures to prevent possible aspiration into the trachea of stomach contents that may be forced into the mouth during surgical manipulation.

**Summary**

By the adoption and meticulous execution of protective technics such as I have outlined in this article, together with appropriate postoperative care, we have reduced postoperative pulmonary complications to an incidence of 0.0028 per cent (4 cases out of a total of 14,157 during the year 1948).

I therefore propose that the procedures that I have set forth in this article be combined with those already in effect in departments of anesthesia of progressive institutions, to reduce to a minimum the incidence of certain complications during anesthesia and to make smoother and less eventful the course of anesthesia generally.

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**ANOXIA**

(Continued from page 86)

regarding the nature of cerebral changes occurring after acute periods of anoxia in human beings. *Permanent damage to the brain as a result of acute cerebral anoxia not only is a function of the duration of the anoxic episode, but also, in the case of sublethal periods of anoxia, must be conditioned by the duration and severity of a period of postanoxic cerebral edema.*

It is suggested that a considerable portion of the permanent cerebral damage reported in these unfortunate cases may be the result not of the initial insult, but rather of a period of untreated cerebral edema subsequent to the initial insult. If this hypothesis is accepted then it must follow that treatment should be directed toward the alleviation of the cerebral edema secondary to acute anoxic accidents. We suggest that the most effective treatment of this kind presently available is the intravenous administration of comparatively large doses of 25 per cent human serum albumin at frequent intervals.

At present animal studies are in progress to determine whether a sound, experimental basis can be established for the hypothesis proposed. If these observations and comments are valid, then the advisability of similar treatment in such cases as acute poisoning with barbiturates and other depressant agents must be considered as adjuncts [sic] to other therapeutic measures.

**Summary**

An attempt has been made to review the literature pertaining to anoxia and its effect on the cerebral cortex.* In this survey it was not intended to draw any positive conclusions. The opinions of the authors as to the importance of anoxia varies. That the subject has a continuing interest is indicated by current reports of investigation and experiment. The whole answer may never be known, but the mass of evidence that is being accumulated should eventually lead to the clarification and elimination of these devastating episodes.

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