

ANESTHETIC MANAGEMENT OF THE PATIENT WITH THYROID DISEASE * †

L. H. MOUSEL, M.D., AND CHARLES S. COAKLEY, M.D.

Washington, D. C.

Received for publication March 8, 1948

IF recorded historical data are correct, the first thyroidectomy in man was performed by Theodore Kocher, Professor of Surgery, at Byrne early in the 1870's (1). In 1874, Kocher published a case report in which he described psychic and physical disturbances following total extirpation of the thyroid gland (2). Kocher eventually reported 2000 thyroidectomies with a mortality rate of less than 5 per cent. These figures are most interesting and one must be impressed with the thoroughness and understanding of a man who was able to operate upon 2000 patients for removal of the thyroid gland and maintain such a low mortality figure without the aid of modern preoperative and post-operative therapeutic agents.

Very little mention is made in the literature of the anesthetic agents used for thyroidectomy during the last part of the nineteenth century. The anesthetic properties of cocaine were not demonstrated until 1873 (3). Although Koller demonstrated the value of cocaine for local anesthesia in 1884, its widespread use did not come into vogue until many years thereafter. Nitrous oxide had been used for anesthesia many years prior to the first thyroidectomy, but a gas machine which would apportion nitrous oxide and oxygen was not invented until Hewitt and White brought out their respective machines in 1898 and 1899.

Ether and chloroform had, of course, been used as anesthetic agents since the 1840's. The belief that Kocher and others, who were pioneers in thyroid surgery, used one or the other of these agents for anesthesia is probably justified.

After the discovery of procaine by Einhorn in 1904, its use for local anesthesia spread rapidly. Very little was written on the subject of anesthesia for thyroid surgery, however, until Crile combined the use of procaine infiltration and nitrous oxide inhalation anesthesia. Crile's observation, as published in 1923 (4), seems to be classic in that his principles were sound. Crile stated, "In cases of colloid goiter, espe-

* Presented before the Philadelphia Society of Anesthesiologists, Phila., Penna., Mar. 5, 1948.

† From the Department of Anesthesia, George Washington University Hospital, Washington, D. C.

cially if the gland is large, the anesthetist must bear in mind the possibility of compression or collapse of the trachea with consequent interference with respiratory exchange." He stressed the need for the anesthetist to be gentle and to secure the cooperation of the patient by suggestion rather than command. Everything that was to be done for the patient was explained to him in detail. Every comfort for the patient was seen to by the anesthetist, even to the sponging of the eyes and forehead with iced compresses during the operative procedure. Crile's patients, who were being operated upon for exophthalmic goiter, had local infiltration combined with nitrous oxide-oxygen analgesia. Crile stated that all forms of inhalation anesthesia interfered with respiration. He said that in patients with exophthalmic goiter, the internal respiration is near the point of failure and that consequently deep anesthesia should not be used although analgesia does not interfere with internal respiration. In this statement, Crile recognized the effects of anoxia in patients with thyrotoxicosis, although he apparently failed to recognize that oxygen insufficiency and not the anesthetic agent *per se* was responsible for the condition.

During the past fifteen years, anesthetists and surgeons alike have experimented with anesthetic methods and techniques in an endeavor to lessen operative and postoperative complications. Nitrous oxide-oxygen has been used alone (7); combinations of local infiltration, nitrous oxide and oxygen have been used (4). Superficial cervical block with local infiltration, sometimes combined with nitrous oxide and oxygen, has long been accepted as the anesthetic of choice in some institutions. Combinations such as ether with oxygen, ethylene-oxygen, cyclopropane-oxygen, and nitrous oxide-oxygen-ether have enjoyed widespread use although some writers are of the opinion that ether should not be used routinely for thyroidectomy. Rea stated that ether is a bad anesthetic for thyrotoxic patients as it tends to produce pulmonary edema (8).

Following the introduction of cyclopropane as an anesthetic agent by Waters in 1933, this agent gained widespread use.

The type of anesthesia used in many clinics is not always to the best interest of the patient. Many prefer to work under local anesthesia, even when the patient is extremely toxic and uncooperative, so that they will be able to hear the patient's speaking voice during surgery and thus determine whether or not the recurrent laryngeal nerve has been damaged (5). Others prefer to have the patient only lightly anesthetized with nitrous oxide or ethylene and oxygen. Here again, only light anesthesia is desired in order that the patient may be awakened from time to time so that the speaking voice may be heard. Some object to the routine use of tracheal intubation because they feel that laryngitis or tracheitis may result. Tracheitis, however, is a frequent complication following thyroidectomy in cases in which endotracheal tubes have not been used (6).

In spite of the improvements made in anesthetic management and technics during the last two decades, a great amount of improvement can be obtained by closer observation of patients before operation. An effort should be made to evaluate not only their physical but their mental status as well, so that anesthetic methods may be individualized.

The modern methods of preoperative preparation of patients who are to undergo thyroidectomy, by the use of Lugol's solution, thiouracil and more recently, propylthiouracil, have improved the condition of the patient with thyrotoxicosis preoperatively to the extent that most of these patients are fairly good operative risks at the time they reach the operating room. Thyroidectomy is not an emergency procedure and there is no excuse for these patients being brought to surgery without adequate preoperative therapy. Patients coming to the operating room with marked palpitation, sweating, tachycardia and mental confusion should be refused for operation until more adequate preparation has been made.

Many experimental efforts have been carried out in an attempt to prevent medullary adrenal releases during and after operation. Crile, Bartells, Stuart and Johnson used spinal anesthesia in the management of postoperative thyroidectomy storm (7). More recently Charles E. Rea (8) reported the use of spinal anesthesia given preoperatively to 20 patients to forestall recurrence of postoperative reactions. Eighty to one hundred twenty milligrams of procaine was injected low into the subarachnoid space. The patient was tilted on the table into a slight Trendelenburg position, until sensory anesthesia reached the level of the fourth dorsal vertebra. This amount of anesthetic agent, under ordinary circumstances, does not interfere with activity of the intercostal muscles, although the concentration probably is sufficient to inhibit medullary adrenal releases during operation.

In discussing anesthesia for the surgical removal of the thyroid gland we must first classify diseases of the thyroid according to pathologic manifestations instead of using the structural or clinical features. Summers' classification based upon clinical pathologic findings seems to be a satisfactory one. His classification is as follows:

1. Diffuse colloid goiter.
2. Adenomatous goiter without hyperthyroidism.
3. Adenomatous goiter with hyperthyroidism.
4. Exophthalmic goiter.
5. Thyroiditis.
6. Myxedema.
7. Cretinism.
8. Malignant diseases.
9. Congenital abnormalities (9).

Constitutional symptoms are not present with colloid goiter. The basal metabolic rate is usually normal or slightly below normal. These

patients usually do not require surgical removal for cure. Surgical resection, however, may be indicated in those cases in which marked obstructive dyspnea is present, or in cases of associated adenomas. The anesthetic management of these patients is simple in comparison with patients who have toxic thyroids in that most any of the anesthetic agents or procedures may be used since the patient usually presents no systemic manifestations of the disease. In cases of marked obstruction and dyspnea the maintenance of an adequate airway will be the chief problem. Adenomatous goiter without hyperthyroidism may be classed with colloid goiter in the selection of anesthetic techniques and agents. Here again the patients usually do not have systemic manifestations of disease.

Most patients with diffuse colloid goiter and adenomatous goiter without hyperthyroidism are excellent subjects for local anesthesia, or the combination of local anesthesia with one of the analgesic gases, such as nitrous oxide or ethylene. Adequate amounts of preoperative sedation should be given to every patient who is to be operated on under local anesthesia. The preoperative sedation, however, should not be carried to the point where the patient will become uncooperative and frequently unmanageable on the operating table. These patients require close attention by an understanding anesthetist throughout the time of operation. The traction on the thyroid gland sometimes gives patients the feeling of suffocation. The anesthetist who is in charge of the patient should place himself in such a position that he will be able to watch most of the operative procedure. This is done so that he can anticipate the surgical maneuvers and reassure the patient during discomforting moments. Many good local anesthetic procedures for operation upon the thyroid gland are defeated because the discomforts have not been explained in detail to the patient. Tracheal compression or perhaps tracheal collapse must be watched for very closely during operations for colloid goiter. If the airway should be so obstructed, the obstruction must be relieved at once.

Patients who have adenomatous goiter with hyperthyroidism and patients with exophthalmic goiter require the benefit of a complete medical survey and intelligent preoperative treatment. Most patients with hyperthyroidism or exophthalmic goiter can be operated on successfully under local anesthesia if the entire surgical team cooperates completely in reassuring the patient and in handling tissue gently.

Preoperative sedation is individualized in order to take care of the patient's needs without oversedation. If the patient, after being given sedatives, is brought to the operating room showing signs of extreme nervousness and emotional turmoil, additional sedative drugs are used. A 2.5 per cent solution of nembutal given very slowly by the intravenous route frequently transforms a very nervous individual into one of almost complete tranquillity. The local anesthetic procedure is carried out with as much gentleness as possible. The anesthetic solution

should be injected from fifteen to twenty minutes before an operative procedure is begun. In occasional patients whose response to pre-operative treatment has not been sufficient to make him a good risk, very careful management must be planned and carried out if operation is to be performed. These patients may be operated on with greater safety if they are spared the excitement of the trip to the operating room.

There may be occasion for 'stealing' of the goiter by giving small doses of one of the basal anesthetic agents such as avertin in amylene hydrate rectally or pentothal sodium intravenously in the patient's room. If this method of management is attempted, preoperative hypodermic infusions are avoided until the patient has lost consciousness. If the anesthetist goes into the patient's room, attired in street clothes or in a laboratory or hospital coat, it will usually be possible for him to place a needle into the patient's vein and inject enough pentothal sodium to produce anesthesia without arousing suspicion. If avertin is given rectally, it is administered by the nurse who commonly attends the patient. When basal narcosis has been established, the patient is taken to the operating room and a close watch is maintained during the trip. After proper placement of the patient on the operating table, a general anesthetic agent is administered. Combinations of avertin and local anesthetics are not satisfactory in these patients and it is our opinion that combinations of pentothal and local anesthetic agents are not safe.

In patients who have been treated preoperatively with thiouracil or propylthiouracil, the thyroid gland may be extremely large and vascular unless iodine therapy has been carried out to reduce the vascularity and the size of the gland. Severe hemorrhage in patients who have not had iodine therapy must be anticipated. If hemorrhage occurs, vigorous replacement therapy with blood must be carried out on the operating table. Most thyroidectomies can be performed successfully and safely under inhalation anesthesia without endotracheal intubation. Endotracheal anesthesia, however, should not be denied the patient if an adequate airway cannot be established early. The average thyroid patient can be operated on safely under general inhalation anesthesia if the less toxic agents are used and if the technical administration is kept simple and logical. It is our custom to carry out preliminary anesthetic induction with sodium pentothal. After the patient has lost consciousness, a mixture of gases containing 80 per cent nitrous oxide and 20 per cent oxygen or 80 per cent ethylene and 20 per cent oxygen is given. Small amounts of ether are added to the anesthetic mixture in order to decrease reflex activity and to deepen the anesthesia to the first plane of the third stage. Anesthesia may be maintained with a high flow of an 80/20 mixture with the addition of ether, or with a completely closed system with ether and oxygen. Postoperative vomiting following the administration of ether by this method is kept to a mini-

num and we believe is no more frequent than the vomiting which follows the administration of other agents.

Patients who show signs of early obstruction, either preoperatively or during the early induction phase of anesthesia, are given the protection afforded by endotracheal intubation. This is especially true when a substernal or intrathoracic goiter is to be removed. Complete compression of the trachea or tracheal collapse should be anticipated and prevented. It is believed that intubation should be carried out on all patients who are to have substernal or intrathoracic goiters removed, and a slight amount of positive pressure should be maintained throughout the operation to prevent pneumothorax from developing should either one of the pleural spaces be entered. This complication develops occasionally and may result in death.

Patients who are being operated on for carcinoma of the thyroid or who are to undergo radical resection deserve the same protection. In these cases, also, it is possible for the surgeon to break through into one pleural space or the other. A large opening into the pleura results in a major pneumothorax with embarrassment of respiration. A small opening into the pleura, unrecognized, may result in tension pneumothorax with possible death. A few years ago one of us (L. H. M.) was called to see a patient who was suffering from marked respiratory embarrassment following total thyroidectomy for carcinoma of the thyroid gland. This patient had bilateral pneumothorax with mediastinal emphysema. The pneumothoraces were relieved by the aspiration of air through needles. The operative incision was reopened by the surgeon. Oxygen given under pressure soon relieved the mediastinal emphysema to the extent that respiratory and cardiovascular embarrassment were no longer apparent. It was originally thought that the respiratory embarrassment resulted from tracheal collapse and a tracheotomy had been done without benefit to the patient about one half hour before the true condition was recognized.

Occasionally a patient comes to the operating room who has severe obstructive symptoms of long standing. These patients may be admitted for thyroidectomy but usually they are brought to surgery for tracheotomy in order to relieve obstructive symptoms. Patients who suffer from prolonged severe obstruction of the trachea may develop sudden pulmonary edema after the obstruction is relieved. This condition can usually be adequately managed by repeated aspirations of the fluid from the trachea and continued positive pressure oxygen therapy.

Cyclopropane has been recommended as the anesthetic of choice for operations upon the thyroid gland. As early as 1935 cyclopropane anesthesia was recommended for the thyrocardiac patient (10, 11). Griffiths, of Canada, gave further support to the use of cyclopropane, and disagreed with cardiologists who believed the agent too dangerous to be used in the presence of cardiac disease (12).

In 1943 Lahey (13) reported three operative deaths probably from ventricular fibrillation in patients with severe thyrotoxicosis. He realized that cyclopropane further insulted a heart already weakened by the action of thyrotoxin. Cyclopropane has excellent anesthetic properties but it affects the parasympathetics and tends to stimulate the vagus. Bradycardia and bronchoconstriction may occur. Myocardial irritability is increased and extra systoles, multiple foci ventricular tachycardia and fibrillations are risked. During the past few years, this agent has lost popularity in patients who have toxic manifestations. It is not our policy to use cyclopropane for patients in this category for we believe that the operative risk is greatly increased.

Procaine, given intravenously, may tend to decrease cardiac sensitivity. If an agent is being used which gives an indication for administering procaine, we believe those indications positively contraindicate the use of the primary drug.

REFERENCES

1. Leonardo, Richard A.: *History of Surgery*, Froben Press, 1943.
2. Crotte, Andre: *Diseases of the Thyroid, Parathyroid and Thymus*, Lea and Febiger, 1933.
3. Keys, Thomas E.: *History of Surgical Anesthesia*, Henry Schumann, 1945.
4. Crile, George W.: *The Thyroid Gland*, W. B. Saunders, 1923.
5. Adams, R. Charles, and Dixon, Claude F.: *Anesthesia in Thyroid Surgery*, *Surgery* 16: 700 (Nov.) 1944.
6. DeCourcy, James L.: *The Aims of Thyroidectomy and Their Achievement; The Need for Close Cooperation between Surgeon and the Anesthetist*, *Anesth. & Analg.* 23: 135-142 (May-June) 1943.
7. Crile, George W.: *Management of Patients with Hyperthyroidism; Preoperative and Postoperative*, *Surg. Clin. North America* 16: 1051, 1936.
8. Rea, Charles E.: *New Plan in Operative Treatment of Patients with Severe Hyperthyroidism*, *Surgery* 16: 731 (Nov.) 1944.
9. Christopher, Frederick: *A Textbook of Surgery by American Authors*, W. B. Saunders, 1942.
10. McArthur, J. W., et al.: *Thyrotoxic Crisis*, *J. A. M. A.* 868 (July 8) 1947.
11. Sise, L. F.: *Anesthesia in the Thyrocardiac Patient*, *J. A. M. A.* 105: 1662 (Nov. 23) 1935.
12. Griffith, Harold R.: *Prevention and Treatment of Complication during Cyclopropane Anesthesia*, *Anesth. & Analg.* 141-144, 1940.
13. Lahey, F. H.; Hurxthal, L. M., and Driscoll, R. E.: *Thyrocardiac Disease*, *Annals of Surg.*, 118: 681 (Oct.) 1943.