

A REVIEW OF ANESTHETIC PROCEDURES EMPLOYED IN 1,016 MAJOR THORACIC OPERATIONS FOR PULMONARY TUBERCULOSIS: COMPLICATIONS AND SEQUELAE *†‡

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MANY articles have been written regarding the advantages of some particular anesthetic agent for patients with active tuberculosis in preventing immediate postoperative spread and reactivation. In these communications much emphasis is placed on the technics of anesthesia and the importance of intubation and bronchoscopy in major chest surgery. In this paper we wish to describe our methods in a series of 1,016 cases in which major thoracic procedures were employed and in which the anesthetic agent could have been a factor in the immediate postoperative complications. These cases are classified according to the classification of the National Tuberculosis Association (1).

CHOICE OF ANESTHETIC

Twenty-one thoracoplasties were done under paravertebral and field block, the anesthetic of choice for cases in which general debility, distribution of the disease, the low vital capacity—any one of these factors or a combination of them—caused us to feel that regional anesthesia would produce less shock than general anesthesia. In all but 2 cases regional anesthesia was successful and no secondary agent was required. In these 2 cases an additional amount of 2.5 per cent of sodium pentothal intravenously, 50 per cent of nitrous oxide and 50 per cent of oxygen was sufficient to carry them through the operation. Inhalation anesthesia was used for all other thoracoplasties, cavernostomies, lucite packs (extrapleural pneumonolysis with lucite plumbage) and excisions (lobectomies and pneumonectomies). The major anesthetic agent in the first 580 cases was ether and in 415 it was cyclopropane (table 1).

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During the period when ether was used as the primary agent, 180 to 250 mg. of sodium pentothal was administered for induction. In the last 415 cases cyclopropane-ethylene-ether or cyclopropane-nitrous oxide-ether was used for induction according to the choice of the anesthetist; 1 to 2 drams of ether was added slowly, after the patient had lost consciousness to avoid as much as possible any struggling or coughing. The reasons for adding the ether are as follows: (1) to avoid conduction disturbance; (2) to increase relaxation for intubation; (3) to counteract the parasympathetic effect of the C_3H_6 since ether is a sympathetic drug, and (4) to secure bronchial dilatation.

TABLE I

Anesthetic and Major Agent	Operations	Intubations
Ether Closed Circle or Waters Canister	580	202 34.80%
Cyclopropane Closed Circle or Waters Canister	415	350 84.30%
Regional and Field		—
Metycaine 2% and 1%	21	—
Total	1,016	552 54.30%

Between October 1943 and July 1945, during which period the first 475 patients were treated, only those having excisions were intubated. Of 520 patients treated from July 1945 on, all patients having first anterior or posterior stage thoracoplasties, all Schede-type (2) thoracoplasties, all bronchopleural fistulas and those second and third stage thoracoplasties in which the disease and history indicated that the patient might "spill" during the operation were also intubated. It was decided to give the patient the benefit of the doubt and employ intubation in the majority of the second and third stage thoracoplasties.

The procedure of cavernostomy presented a problem in the choice of an anesthetic agent. By cavernostomy an attempt is made to convert the patient's sputum from positive to negative after various stages of thoracoplasty have failed to effect cavity closure. The procedure includes three steps:

1. The skin flap is prepared, the regenerated rib above the area of cavitation is removed and an oxidized gauze pack is inserted in this area to assure fusion of the pleura for the second stage procedure.

2. The cavity is located by needling and after accurate determina-

tions of the site, the wall is cauterized to effect opening and thus establish the maximum amount of drainage to the outside with the aid of the skin flap.

3. Upon conversion of the sputum to negative and cessation of drainage with the cavity presumably closed, the muscle flap is inserted into the opening of the cavity to secure closure of the open area.

In performing the first and third stages, an inhalation anesthetic agent is administered. The second stage, however, presents a problem inasmuch as the procedure necessitates the use of a cautery and any agent of an explosive nature is contraindicated. This leaves the choice between an intravenous rectal or a local anesthesia. In 2 instances intravenous anesthesia was employed. These 2 patients happened to have large bronchial connections with the trachea. Since the cough reflex is absent and there is very little manipulation of the lung itself, only a minimal amount of the cavitory contents spills into the main bronchus. When the cavity has been opened with the cautery, however, all the purulent material and blood are aspirated and some blood, cautery debris, and cavitory contents may enter the main bronchus. This is easily obviated, however, by bronchial toilet through the intratracheal tube. The difficulty arises when the smoke formed on cauterization enters the bronchial tree filling all the dead space. Ventilation and replacement with oxygen entail some time. Meanwhile the patient becomes cyanotic and a marked degree of cyanosis may persist to the danger point.

Soon after the cavity has been opened and aspirated, a skin flap is inserted in the area, a pack applied, and the patient returned to his bed. It was noted that although oxygenation with pressure and escape oxygen was performed, these patients made a much slower recovery from the anesthesia than was usually the case with the same amount of anesthetic agent and the same length of time of administration. It was concluded that the smoke, whatever its composition and nature may be, not only gradually permeated through the bronchial tree but may also have been partially absorbed into the system, thus deepening anesthesia and causing cyanosis by mechanical blocking of the airway. This hypothesis would explain the insidious onset and the delayed recovery. Roentgenologic examination revealed no evidence of bronchial occlusion.

Although both of these patients recovered with arrest of their disease, the above procedure was abandoned for obvious reasons. At this point it is recommended that if the same surgical procedure is performed under a general anesthetic, at the first sign of cyanosis it should be assumed that there is a direct cavitory connection with the bronchial tree, and the anesthetic should be discontinued. Oxygen under pressure and escape should be instituted and maintained until the patient has regained his cough reflex and is able to cough up any foreign material which may have become lodged in the bronchial tree. By this time the active surgical procedure has been completed and the

pain of cauterizing the lung is relatively mild and well tolerated by the patient.

At present our method of anesthesia for this procedure is paravertebral block and field infiltration. In view of the fact that all landmarks have disappeared because of antecedent thoracoplasties, in some instances the surgeon has to reinforce the paravertebral block.

PREMEDICATION

In the majority of our cases premedication presents a problem. Many patients have been in the hospital for months or several years receiving codeine, $\frac{1}{2}$ grain every three hours, for long periods because of cough, hemorrhage or pain. Many have had hypnotics, any one of the barbiturates daily for varying periods. Thus most patients who have had $1\frac{1}{2}$ grains of seconal at bedtime, $1\frac{1}{2}$ grains of nembutal one and a half hours preoperatively and $\frac{1}{4}$ grain of morphine sulfate and $1/100$ grain scopolamine forty-five minutes to one hour preoperatively will arrive in the operating room fully awake and without much evidence of sedation.

Since all of our patients have an impaired vital capacity, some coming to surgery with a vital capacity as low as 30 per cent, we hesitated to give them a large premedication dose lest respiration be further depressed. We varied the dosage of morphine between $\frac{1}{8}$ and $\frac{1}{4}$ grain with atropine or scopolamine in the ratio of 25:1. Experience has demonstrated, however, that the larger dose does not affect their respiration too seriously. If respiratory depression occurs in patients with a low vital capacity who have not been in the institution long enough to get used to narcotics, it can be easily corrected by the present-day method of intubation with controlled supplementary respiration until the maximum effect of the drug has worn off.

MAINTENANCE

Maintenance is accomplished with cyclopropane and ethylene or cyclopropane and nitrous oxide, employing the carbon dioxide absorption technic with closed circle or Waters canister. (See table 1.) During the conduct of all anesthetics, each patient receives 1,000 cc. of a 5 per cent solution of glucose in saline solution intravenously. The addition of plasma or whole blood depends on the amount of bleeding and oozing which may be present and also on the patient's preoperative condition. During operation the patient's respiration is not only watched but also listened to by lifting the inspiratory tube to the ear; thus even minimal amounts of secretion in the bronchi can be detected and aspirated with a catheter.

When closure of the wound following the operation is begun, cyclopropane is discontinued and anesthesia maintained with ethylene or nitrous oxide. While the skin incision is being sutured the patient

rebreathes oxygen and atmospheric air. Thus, before he is removed from the operating table he has regained his cough reflex, and on irritation from the endotracheal tube, he attempts to cough. Owing to an open glottis, his cough attempts are unsuccessful. A suction catheter is immediately inserted; this partially or temporarily blocks the airway and the patient can thus build up a sufficient amount of positive pressure to expel most of the secretions from the secondary bronchi into the main bronchi and trachea. As the catheter is being agitated up and down rapidly for suction, the tube is slowly withdrawn on expiration.

Soon after being returned to bed, the patient regains consciousness. Shock blocks are placed at the foot of the bed to promote bronchial drainage. The patient lies on the operative side and is turned every two hours, lying for twenty minutes on the contralateral side. If he has much secretion, but suppresses his cough because of pain, the trained attendant or special nurse holds his operative side with both hands anteriorly and posteriorly simultaneously, a procedure which in most instances facilitates coughing up the secretions with less effort and less pain.

Narcotics are withheld after operation until the patient has fully reacted and has coughed up any secretions that may have formed since removal from the operating table. This precautionary measure acts to prevent depression of the cough reflex. By faithful adherence to this routine only a very few of our patients have had to be bronchoscoped either immediately after operation or within the first three days after operation.

BRONCHOSCOPY

Formerly it was thought that if a patient had a slight amount of bleeding into the trachea or an excessive amount of secretion bronchoscopy should be performed immediately after operation even though a thorough bronchial toilet had been done during the operation and no evidence of moisture could be detected on listening through the inspiratory tube of the gas machine. Inasmuch as no gross bronchial secretions or foreign matter could be found in all these cases, bronchoscopy immediately after operation was abandoned and reserved for those patients treated by excision in whom there was an excessive amount of bleeding into the main bronchus. In these cases bronchoscopy is done in order to make certain that no coagulated blood blocks a bronchus which might have escaped auditory detection with the inspiratory tube.

The reasons for discontinuance of this procedure are the following:

1. With individual ligation, which is the current method of excision, the probability of blood entering the bronchus is very small.
2. This probability is further decreased by the fact that in the majority of cases the blood vessels are tied off before the bronchus is opened.

3. Upon closure of the bronchus, positive pressure is applied in the presence of normal saline solution in the thoracic cage; bubbling will reveal incomplete closure.

4. Accumulation of secretions in the lower bronchus is also prevented by employing the Overholt position in excision cases. In a recent paper (3) one of us, B. E. S., and J. D. Murphy have described an adaptation of the Albee-Comper orthopedic table which keeps the patient face downward at a 10-degree Trendelenburg position.

5. We believe that postoperative recovery is enhanced by having the patient awake soon after operation, the sooner the better. This applies mainly to inhalation anesthetics for thoracoplasties and excisions for pulmonary tuberculosis.

In order to bronchoscope the patient, fairly deep anesthesia must be maintained until the incision is closed and dressings are applied before he is turned on his back. This prolongs the anesthesia and delays return to consciousness. Bronchoscopy at best can remove only the secretions from the main bronchi and the lower lobe bronchi. Secretions in the secondary bronchi cannot be removed until the patient can cough voluntarily. This he cannot do until he has fully reacted and even then many patients are afraid to cough owing to postoperative discomfort. By this time a sizeable amount of secretion may have collected, necessitating another bronchoscopy. On the other hand, with no bronchoscopy, 90 per cent of these patients regain the cough reflex before they are removed from the operating table. Both the endotracheal and the suction catheters stimulate cough and enable the patient to expel most of the secretions from the secondary bronchi during the time they have no inhibition, for they still have no pain. Upon withdrawal of the catheters they will continue to cough, and will cough up any secretions which remain after nasal and oral toilet.

After they are back in bed and fully awake, the patients are encouraged to cough, and they expectorate very easily any secretions which may have accumulated in that short interval between bronchial toilet and full consciousness.

Some patients are bronchoscoped during their recovery period. The indications include: (1) those patients who are unable to cough up and expel secretions because of general debility or a low vital capacity; (2) elevation of temperature from unknown cause, and (3) presence of a shadow in the roentgenogram which might indicate fluid, atelectasis or pneumonitis. Bronchoscopy is done in this instance to determine whether a plug or other factors may be causing atelectasis.

COMPLICATIONS

In reviewing the 1,016 cases we have considered only those complications in which anesthesia may have been a factor. Thus we have not discussed such complications as bronchopleural fistula, empyema, traumatic pneumothorax, wound infections and the like.

In table 2 the patients treated are listed by sex and age. We note that about 45 per cent fall into the age group of 20 to 30 years; 17 per cent, in the 30 to 40 age group; and a total of about 37 per cent in the next three periods—namely, those 40 to 50, 50 to 55, and 55 to 60 years of age. Although the 40 to 55 age group comprises only about 34 per cent of the total number of patients, 61 per cent of all deaths occurred in this group; whereas only 27 per cent of the deaths occurred in the 20 to 30 age group, which is to be expected, since this is the youngest age group and therefore the most robust, with shorter duration of disease.

TABLE 2

Age, Years	Sex	Operations	Percentage
20-30	M	454	44.60
	F	4	.39
30-40	M	172	16.90
	F	3	.29
40-50	M	171	16.80
	F	5	.49
50-55	M	163	16.10
	F	2	.19
55-60	M	42	4.13
	F	—	—
Total		1,016	

Table 3 lists the various surgical procedures employed and the number of patients treated by each procedure.

In table 4 we have tabulated the distribution of disease. Of the 414 patients operated on, 249 or 60.1 per cent had so-called unilateral disease; 153, or 37 per cent, had bilateral disease; and 12, or 3 per cent, had contralateral pneumothorax at the time of operation. In reviewing the roentgenograms and classifying the patients, we placed some who might have been included in the bilateral group with the unilateral cases. These were the patients we knew had infiltrations on the contralateral side but healing had taken place leaving a few fibrotic strands. In the bilateral group were placed those who had disease in varying degrees on the contralateral side, but in which healing occurred leaving fibrotic strands, calcification, dense fibrosis and some apparently healed cavities. A number also had minimal infiltrations.

The question may arise, why do we operate upon patients with active disease on the contralateral side? Time and experience have shown that with most patients who have considerable bilateral disease—but who with the aid of bed rest, phreniclasia, pneumothorax or pneumoperitoneum (any one or a combination of these therapeutic measures) have become sufficiently healed on one side and with disease cleared more or less on both sides, leaving one or more cavities on one side and a small infiltration and fibrosis on the other—if operation is successful and the cavities close, the other side will continue to heal in spite of, or perhaps with the help of, the operation, since the cavity or cavities were the source of cross infection.

TABLE 3

Type of Operations	Number
1st Stage	304
2d Stage	256
3d Stage	137
4th Stage	5
Anterior Stage	164
Revision Thoracoplasty	46
Cavernostomy	25
Schede	9
Scapulectomy	2
Pneumonectomy	18
Lobectomy	22
Segmental Lobectomy	2
Lucite Pack	26
Total	1,016

TABLE 4

Distribution of Disease	No. of Patients
Unilateral Disease	249
Bilateral Disease	153
Contralateral Pneumothorax	12
Total Number of Patients	414

In going over the literature on complications resulting from anesthesia in tuberculous patients, we read of "spreads." A spread may be defined as the appearance of the tuberculous process in a portion of the lung in which there was no disease before operation. If after operation a patient develops a reactivation of an old, apparently healed process, an extension of still active disease, or has a cavity reopen which seemed to be closed, one can hardly term these "spreads." Therefore, we have classified them rather as reactivations or extensions of old disease. In the group of 153 patients with bilateral disease (see table 4)

comprising 37 per cent of the total, there was reactivation or extension of the old disease in 4.35 per cent of patients, or 1.77 per cent of operations. In the group of 249 patients with unilateral disease comprising 63 per cent of the total number of patients, in only 1.2 per cent of patients or 0.49 per cent of operations was there any spread of disease to apparently healthy lung tissue. In table 5 seven spreads are listed. However, 2 of these 7 patients who presumably developed new disease, had preexisting bilateral disease. But even if we include these 2, there was spread in only 1.69 per cent of patients and in 0.69 per cent of operations. Combining these two groups, we have a total of 25 reactivations, extensions and spreads, or in 6.03 per cent of patients and 2.46 per cent of operations.

TABLE 5

	Complications			Anesthesia			
	No. of Patients	Percentage of Patients	Percentage of Operations	Ether	Cyclopropane	Regional	Tube
Reactivations and Extensions	18	4.35	1.77	10	8	—	12
Spreads	7	1.69	0.69	5	2	—	5
Reactivations and Spreads Combined	25	6.02	2.46	—	—	—	—
Pneumonitis	1	0.24	0.09	1	—	—	—
Atelectasis	5	1.45	.49	4	1	—	1
Early and Late Deaths (Not including Excision Deaths)	15	4.03	1.63	10	7	1	9
Deaths in Excisions	3	7.14	7.14	1	2	—	3

Most of the articles written on the subject of anesthesia in procedures for tuberculosis contain arguments *pro* and *con* regarding ether as a causative factor in the development of postoperative spread. This has already been disproved by several authors (4-8). From the figures compiled in table 5 we have further proof that ether is not a factor. Of the 25 reactivations and spreads, 15 patients were given closed ether, or 2.58 per cent of all those who had ether as a major agent, and 10 had cyclopropane, or 2.41 per cent of all those who received cyclopropane as a major agent.

In referring to spreads, etc., in papers on anesthesia, the authors simply stated the percentage of spreads which resulted without giving any explanations or qualifications. It must, therefore, be assumed by the reader that the spreads were the result of the anesthesia.

We disagree with this interpretation of the case. We have already shown the large percentage of patients coming to thoracic surgery with bilateral disease. It has also been demonstrated that most of the reactivations and spreads occurred in this group. These patients showed considerable resistance to the process by clearing much of their disease or by achieving stabilization of the process. Such patients undergo an

operation which causes extensive trauma. Who can say that this is not a factor in lowering resistance, thus aiding the bacilli to gain the upper hand and cause reactivation or extension? The fact that extension of the disease occurred in only 2.46 per cent of operations shows that most of the patients had enough resistance to withstand the operative procedure without reactivation or spread in spite of the trauma incurred.

There were 69 colored patients in the series comprising 16.6 per cent of the operative patients, among whom there were eight spreads, or 32 per cent of all spreads. Given the same anesthetic agents, the same method of conducting the anesthesia, the same anesthetists and surgeons, there should have been only 4 spreads instead of 8. It is an accepted fact, however, that the colored race has a much lower resistance to tuberculosis than the white race. Thus we have further evidence that the amount of resistance of the individual plus the lowered resistance owing to surgical trauma comprise the major factors in the etiology of spreads.

Can we absolve the anesthesia altogether as a causative factor in this complication? The anesthetist must supply the answer himself, and the answer is that if the anesthesia is conducted in such a manner as to be as little debilitating to the patient as possible—that is, by carrying the anesthesia very light and with the earliest possible termination to avoid protracted recovery, also caring for the patient by replacing fluids and preventing the accumulation of bronchial secretions by frequent bronchial toilet—then the part anesthesia plays in the development of spreads is almost negligible. For example, it would seem that in spinal anesthesia in which the patient is kept awake throughout the operation and is able actively to rid himself of all secretions, the number of spreads and reactivations should be less than with inhalation anesthesia. However, Schaffner and Found (9), employing spinal anesthesia in 335 thoracic operations, reported a total of 3.0 per cent of postoperative spreads and reactivations. In our series of 1,016 operations in which inhalation anesthesia dominated the picture, there were postoperative spreads and reactivations in 2.46 per cent.

The statistics regarding extension and spread per operation should be taken, not those per patient. If extension of disease occurs after an operation, further surgical procedures are usually postponed until the spread clears more or less and there is evidence of definite healing of the tuberculous process. Then surgery is resumed with more caution and in several stages. Some patients have undergone as many as seven surgical interventions to convert sputum and to bring about arrest of the disease. Thus, each procedure represents a separate risk, and any given procedure may be the cause of a spread or a reactivation, a fact clearly demonstrated in the descriptions of the cases.

PNEUMONITIS

There was only one case of a clear-cut pneumonitis—a small patchy density which cleared within three days. Several of the spreads which cleared within four to six weeks could have been put in the classification of pneumonitis, but since opinion was divided as to whether it was an infection other than tuberculosis or a tuberculous spread, these were classified as spreads.

ATELECTASIS

Of the 5 cases of atelectasis or 1.45 per cent of patients and 0.49 per cent of operations, 4 had ether anesthesia and had not been intubated, and one had cyclopropane and was intubated. Although the total percentage is very small, it can be argued that had the 4 been intubated, perhaps the atelectasis in those patients might have been avoided. All of these patients were subsequently bronchoscoped and the atelectatic lobe or lobule eventually reexpanded. In our earlier cases intubation was not done because it was thought that it might cause laryngeal tuberculosis; however, even though many patients came to thoracoplasty with bronchial tuberculosis, no case of laryngeal tuberculosis following intubation was reported. Until we analyzed our cases we were under the impression that intubation played an important rôle in preventing spreads and reactivations. After careful analysis, however, we found that spreads occurred in almost equal proportion with or without intubation. Moreover, all poor-risk patients were intubated and when their poor general condition is considered, it is difficult to determine just how important the intubation was. At present we are routinely carrying out intubations on all patients for thoracic surgery who receive inhalation anesthesia for the following reasons: (1) to secure adequate bronchial and tracheal toilet during anesthesia and immediately after operation; (2) to secure better control of the patient; (3) to lessen the amount of anesthetic agent required, and (4) to facilitate reexpansion of the lung after traumatic pneumothorax.

All cases except 3 were classified as far advanced when the patients came to surgery. Three cases were classified as moderately advanced. Since many months must elapse after conversion before the case can be considered arrested, our statistics of final results go back one year to March 1947. At that time there were 325 patients on whom 625 operations had been performed. There were 62 unsuccessful thoracoplasties in which the sputum failed to be converted to negative, with spreads and reactivations, representing 19 per cent of the total number of cases. The remaining cases, 81 per cent, represent successful thoracoplasties in which arrest of the tuberculous process (10) was finally achieved.

The deaths as described are self-explanatory. There were 15 (both early and late deaths are included), or 4.03 per cent of patients and

1.63 per cent of operations. No death in this group was attributed to anesthesia.

SUMMARY

This critical survey of 1,016 operations for pulmonary tuberculosis reveals the following:

1. The anesthetic agent was not a factor in causing spreads or reactivations.
2. Lowered resistance of the patient is the vital causative factor in the development of spreads or reactivations.
3. Profound and prolonged anesthesia superimposed upon surgical trauma is assumed to play a major rôle in lowering the patient's resistance.
4. Endotracheal anesthesia affords the anesthetist a better opportunity of approaching the ideal in anesthesia for tuberculosis, namely, light anesthesia; bronchial and tracheal toilet and better control.

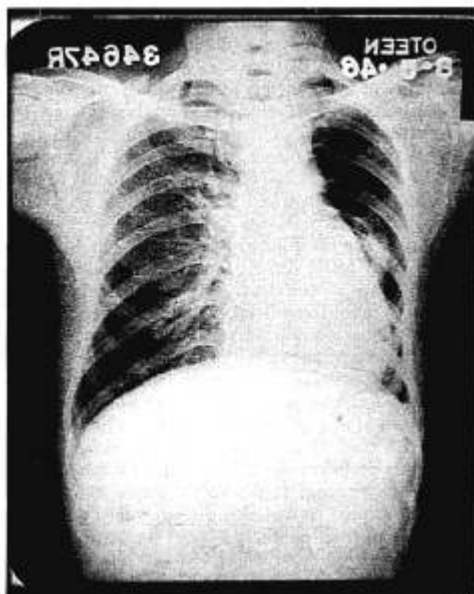


FIG. 1. Plate I: September 5, 1946, hydro-pneumothorax on the left and a fibroexudative lesion in the upper half on the right; in the second costochondral junction there is an area suggestive of minimal cavitation.

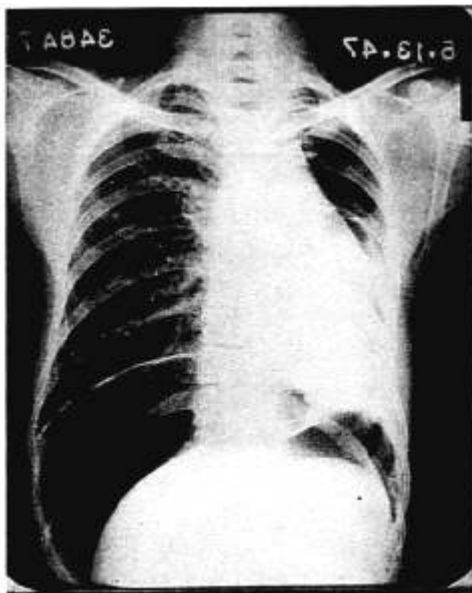


FIG. 2. Plate II: May 13, 1947, existing pneumoperitoneum. Left pneumothorax with an unexpandable lung. On the right most of the exudative disease has disappeared leaving fibrotic strands and some areas of dense fibrosis at the second costochondral junction.

5. No case of laryngeal tuberculosis has been observed following endotracheal anesthesia.

6. Bronchoscopy performed routinely immediately after operation has proved an unnecessary procedure, in our opinion, when adequate bronchial and tracheal toilet is carried out during anesthesia.

REVIEW OF CASES

Reactivations and Extensions—18 Cases

1. L. S. Male, age 40. Vital capacity: 1,950/4,750. Hydropneumothorax, left. Productive lesion, right. Reactivation and spread on right side after first-stage thorocoplasty, left, 6/30/47. Anesthesia: cyclopropane (tube). Roentgenograms (figs. 1, 2, 3, 4) demonstrate extension of an active lesion which was present before surgical intervention.

2. E. H. Male, age 37. Vital capacity: 2,700/4,550. Large multiple cavities, upper half, left. Minimal infiltration, second interspace. Reactivation

and spread of disease in upper third, right, after first-stage thoracoplasty 11/20/46. Anesthesia: cyclopropane (tube).

3. L. N. P. Female, age 28. Vital capacity: 1,400/3,450. Cavity, left, under fifth rib. Thoracoplasty, left. Fibroid deposit, upper third, right. Re-activation of old disease after revision thoracoplasty, 10/14/46. Anesthesia: cyclopropane (tube).

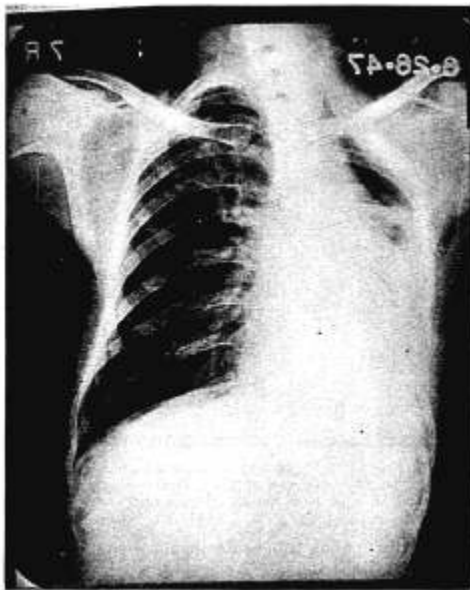


FIG. 3. Plate III: June 26, 1947, first-stage thoracoplasty, left, to obliterate the hydro-pneumothorax space. There is extension and reactivation of the disease with a small cavity showing in the second interspace near the hilum.

4. A. E. Male, age 32. Vital capacity: 2,400/5,100. Infiltration and cavitation, upper half, left. Fibrosis with apparently stationary lesion, right. Re-activation of old lesion, right, after first-stage thoracoplasty 10/18/46. Anesthesia: ether (tube).

5. E. C. K. Male, age 28. Vital capacity: 2,000/4,550. Cavity, 4 cm., upper left, scattered infiltration, right. Increased disease to cavitation on right after third-stage thoracoplasty 9/18/46. Anesthesia: cyclopropane (tube).

6. J. W. Male, age 34. Vital capacity: 2,000/4,350. Cavity, 8 cm., right. General fibrosis, left. Reactivation of old disease with opening of old cavity, left, after second-stage thoracoplasty 8/21/46. Anesthesia: cyclopropane (tube).

7. Q. D. Male, age 51. Vital capacity: 1,900/4,250. Extensive cavitation, right, upper. Stringy infiltration above third rib, left. Extension of disease, left after first-stage thoracoplasty 8/26/46 (tube). Healed before second stage. Anesthesia: cyclopropane. For second and third stage used regional block.

8. A. J. W. Male, age 52. Vital capacity: 3,500/4,300. Cavity, 4 cm., right apex. Dense nodular fibrosis, left apex. Minimal contralateral reactivation, second interspace after third stage thoracoplasty 3/25/46. Anesthesia: ether (tube).

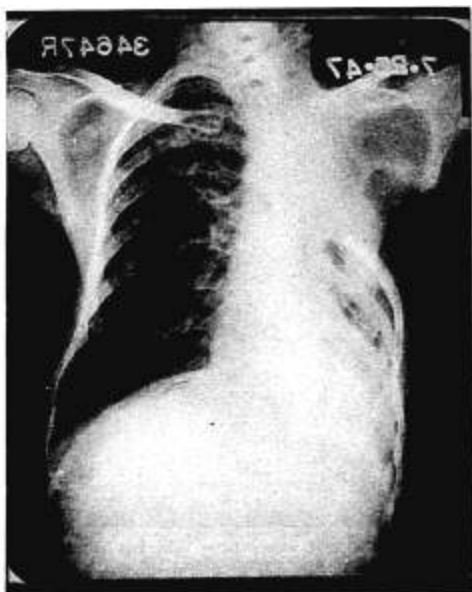


FIG. 4. Plate IV: July 25, 1947, after the second-stage thoracoplasty on the left. The only change on the right is accentuation of the cavitary lesion.

9. C. J. B. Male, age 26. Vital capacity: 3,300/4,900. Cavity, 4 cm., right. Fibronodular disease in hilum. Small cavity apparently healed. Ipsilateral spread to right base after first-stage thoracoplasty 10/17/45. Anesthesia: ether (tube).

10. C. S. Male, age 49. Vital capacity: 3,300/5,100. Fibronodular deposit throughout right lung. Empyema with bronchopleural fistula, left. Flare-up of contralateral disease after second-stage thoracoplasty, left, 3/14/45. Anesthesia: ether.

11. R. F. Male, age 28. Vital capacity: 4,200/4,800. Fibroexudative infiltration, upper third, right. Cavity, 1 cm., left. Reactivation of disease with spread after first-stage thoracoplasty 12/29/44. Anesthesia: ether.

12. C. M. Male, age 28. Vital capacity: 3,100/4,300. Small cavities, right apex. Reactivation in perihilar area, left, after the third-stage thoracoplasty.

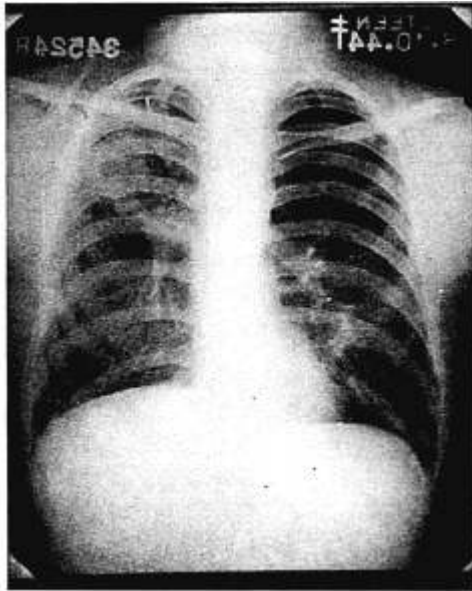


FIG. 5. Plate I: June 10, 1944, fibroexudative lesion and several small cavities in the right upper lobe. On the left there is a pneumothorax and an apparently controlled lesion in the lower lobe.

Cleared in four weeks. Anesthesia: ether. Roentgenograms (figs. 5, 6, 7, 8) show a reactivation of an apparently fibrotic lesion in the contralateral lower lobe with subsequent clearing.

13. H. W. A. See under *deaths*. Considered as a reactivation and extension of old disease, right. Anesthesia: ether.

14. A. C. Male, age 36. Vital capacity: 3,100/4,200. Cavity, 5 cm., with exudate disease, upper half, right. Recent small spread, third interspace, left anterior. Contralateral minimal extension of disease after first-stage thoracoplasty 3/8/44. Anesthesia: ether (tube).

15. C. R. See under *deaths*. Male, age 23. Vital capacity: 2,500/4,300. Anesthesia: ether.

16. A. A. See under *deaths*. Male, age 21. Anesthesia: ether.

17. B. M. Male, age 30. Cavity, 3 cm., left mid-lung. Lobectomy, upper left 11/14/46. Five months after operation patient developed a bronchopleural fistula and contralateral spread. Cured by thoracotomy and streptomycin. Anesthesia: cyclopropane (tube).

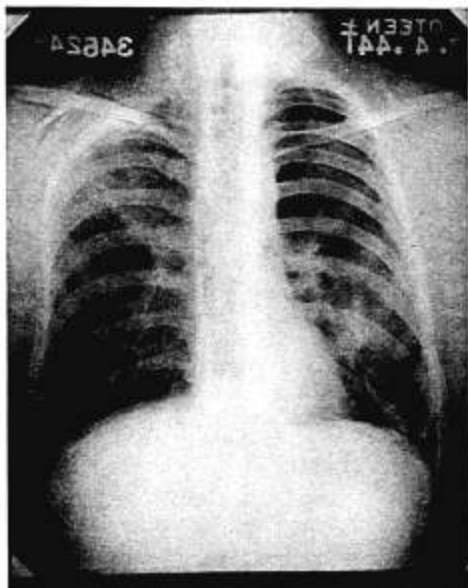


FIG. 6. Plate II: July 4, 1944, first-stage thoracoplasty, right, and no change on the left.

18. C. B. Male, age 22. Cavity in right lower lobe. Lobectomy, right lower 4/2/47. Received streptomycin. Left AWOL two weeks postoperatively. Returned one month later with contralateral spread. Cleared with streptomycin. Anesthesia: cyclopropane (tube).

Spreads—7 Cases

1. M. W. D. Male, age 26. Vital capacity: 2,050/4,450. Cavitation, right upper lobe. Bronchogenic disease, lower half of left lung. Contralateral spread to second interspace, left, after first-stage thoracoplasty 11/3/47. Anesthesia: cyclopropane (tube). Figure 9 shows two large cavities, one in upper right, and one in lower lobe, respectively; left side is clear, 10/31/47. Figure

10 is a roentgenogram 11/18/47 showing first-stage thoracoplasty, right side and contralateral spread in left second interspace, anterior.

2. O. P. Male, age 21. Vital capacity: 1,800/4,250. Giant cavitation, upper two-thirds, left. Minimal productive strand in mid-lung field, right. Spread to contralateral side after second-stage thoracoplasty 10/1/47. On streptomycin. Anesthesia: cyclopropane (tube).

3. W. O. F. Male, age 29. Vital capacity: 2,100/4,650. Fibrothorax and a 4 cm. cavity, right. Left side fairly clear. Contralateral pneumonitis after first-stage thoracoplasty 3/11/46. Anesthesia: ether (tube).

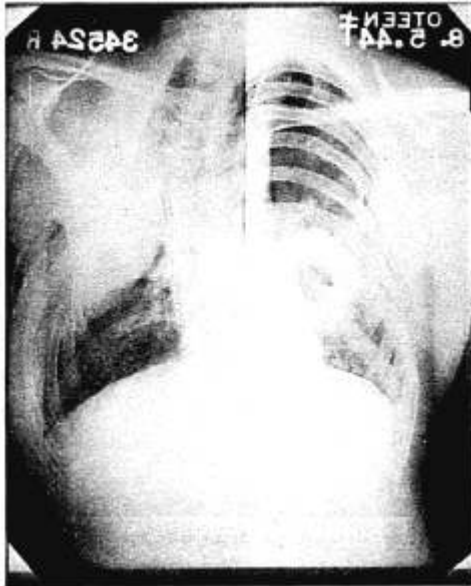


FIG. 7. Plate III: August 5, 1944, third-stage thoracoplasty, right, and dense pneumonic involvement of the left lower lobe.

4. E. J. Male, age 25. Vital capacity: 2,500/4,650. Cavity, 5 cm., left; right side clear. Slight ipsilateral spread after first-stage thoracoplasty 2/11/46. Cleared in two months. Anesthesia: ether (tube).

5. F. G. C. Male, age 20. Vital capacity: 2,500/4,600. Cavity, 3 cm., upper right. Left side clear. Elevation, fifth postoperative day after first-stage thoracoplasty 10/24/45. Roentgenograms showed no increase. Bronchoscopic examination, negative. On sixth day hilar ipsilateral spread which cleared later. Anesthesia: ether (tube).

6. G. L. M. Male, age 51. Vital capacity: 4,300/4,400. Right side clear. Cavity, 2 cm., left upper. Slight contralateral spread after first-stage thoracoplasty 10/25/43. Cleared. Diabetic. Anesthesia: ether.

7. E. T. See under *atelectasis*.



FIG. 8. Plate IV: October 24, 1947, successful third-stage thoracoplasty, right, and a fibrotic healed lesion in the left lower lobe.

Pneumonitis

1. C. P. J. Male, age 51. Vital capacity: 3,750/3,900. General fibrosis, right. Cavity, 4 cm., left apex. Fever, second postoperative day, after a first-stage thoracoplasty, left, 11/13/44. Roentgenogram showed pneumonitis which cleared in three days.

Atelectasis

1. W. B. C. Male, age 52. Cavity, 3 cm., right upper lobe. Clear on left. Atelectasis of right upper lobe after first-stage thoracoplasty 9/19/46. Anesthesia: cyclopropane (tube).

2. C. H. Male, age 24. Slight apical fibrosis, right. Cavity, 3 cm., left. Temperature 103° F. on third postoperative day. Atelectasis, right lobe. Bronchoscopy with suction; subsequent clearing. Anesthesia: ether.

3. J. M. Male, age 56. Slight fibrosis, right. Honeycomb cavitation, upper left lobe. Atelectasis, left base after third-stage thoracoplasty 8/9/44. Bronchoscopy revealed partial stenosis of left main bronchus. Anesthesia: ether.

4. E. T. Male, age 27. Right side clear. Fibroexudative disease with 2-cm. cavity, left upper half. Contralateral minimal apical spread and basal atelectasis, right. Fever. Suction bronchoscopy relieved the patient. Anesthesia: ether.

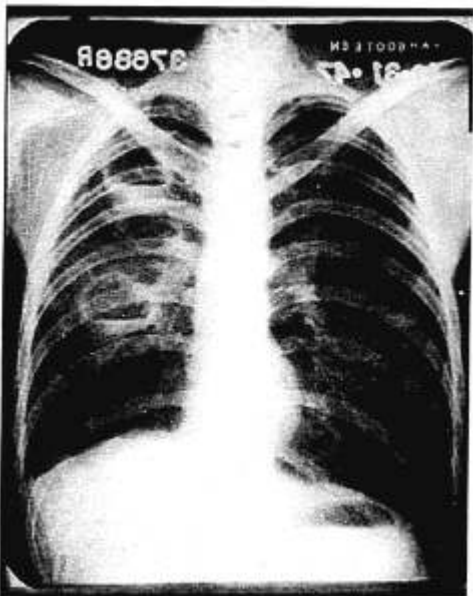


FIG. 9. Plate I: October 31, 1947, two large cavities, one in the upper and one in the lower lobe, right. The left side is clear.

5. S. V. Male, age 27. Cavity, 3 cm., right. Chronic pleuritis, left. Temperature 102° F. on fifth postoperative day after second-stage thoracoplasty 11/10/43. Roentgenogram revealed atelectasis, right base. Cleared. Anesthesia: ether.

Postoperative Deaths

1. B. M. Male, age 45. Cavity, 4 cm., right apex; cavity, 2 cm., right mid-lung; cavity, 2 cm., with scattered fibrosis, left. Patient also had chronic laryngeal tuberculosis and extension of disease after first-stage thoracoplasty 1/7/44. Died of progressive disease; pneumonitis 4/18/44. Anesthesia: ether.

2. L. N. O. Male, age 41. Vital capacity 2,900/4,300. Fibrosis, throughout right lung. Cavity, 3 cm., left, with hydropneumothorax. Third-stage thoracoplasty—unsuccessful collapse. Last stage 4/14/44. Patient had a late spread. Died 1/7/46. Anesthesia: ether.

3. W. H. A. Male, age 25. Vital capacity: 2,700/3,900. Minimal fibrosis, right, with recently closed cavity, upper lobe. Left pyopneumothorax. Anterior stage thoracoplasty 2/9/44. Posterior stage 2/28/44. Surgery, left, dis-

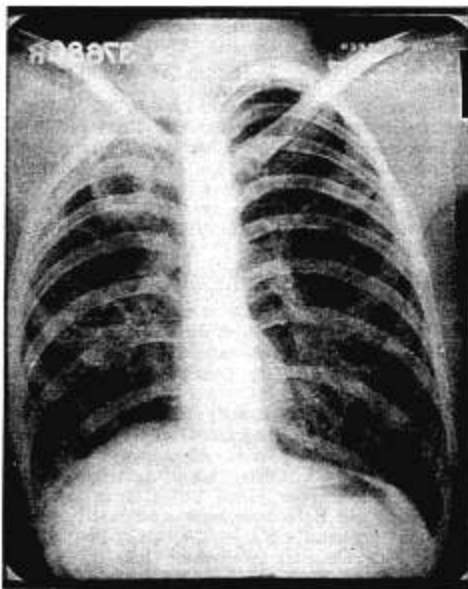


FIG. 10. Plate II: November 18, 1947, first-stage thoracoplasty, right, and a contralateral minimal spread in the second interspace anterior.

continued owing to reactivation, right. Died of asphyxia resulting from hemoptysis 9/16/44. Anesthesia: ether.

4. C. S. M. Male, age 47. Vital capacity: 3,000/4,200. Cavity, 5 cm., upper right. Three stages thoracoplasty 12/13/44. Patient became cyanotic after operation. Blood and plasma given at 3:00 p.m. with slight reaction. Patient in extremis at 5:00 p.m. Bloody drainage from wound. Wound opened and transverse process packed. Patient died at 5:20 p.m. Anesthesia: ether.

5. H. T. Male, age 50. Vital capacity: 1,200/4,300. Ten-rib thoracoplasty, 1936. Two draining sinuses. Anterior revision 2/6/47. Anesthesia: ether. Posterior revision 3/7/47 and Schede 3/12/47. Anesthesia: cyclopropane.

Fever postoperatively, which subsided in one week after drainage was established. Died 5/14/47 (tube, each operation).

6. A. A. Male, age 21. Vital capacity: 2,400/4,000. Cavity, 5 cm., with fibrosis, upper third, right. Hilar infiltration, left. First stage thoracoplasty 6/9/44. Fever postoperatively. Extension of disease, left. Continued to fail. Died 10/26/44. Anesthesia: ether.

7. R. C. Male, age 53. Vital capacity: 3,500/3,700. Died eight days after first-stage thoracoplasty. Patient had a normal afebrile course, but developed severe dyspnea on eighth postoperative day and died in thirty minutes. Probable cause: embolism. Anesthesia: ether.

8. C. R. Male, age 23. Vital capacity: 2,500/4,300. Cavity, 5 cm., left upper, and fibrosis, right upper lobe. Died of extension of disease two months after last operation 2/13/44. Anesthesia: ether.

9. P. M. Male, age 31. Vital capacity: 2,600/5,050. Cavity, 5 cm., with infiltration, right, and considerable fibrosis, left. Previous bilateral pneumothorax, phreniclasia, pneumoperitoneum. Two stage thoracoplasty. Postoperative respiratory depression, rate 8. Died ten days after operation 12/27/45 of cardiorespiratory failure. Anesthesia: ether (tube).

10. F. R. Male, age 35. Vital capacity: 2,200/4,600. Had two stages of thoracoplasty followed by a cavernostomy 8/12/46. Traumatic pneumothorax. Died suddenly three weeks after operation. Anesthesia: regional.

11. R. H. Male, age 53. Vital capacity: 1,900/4,700. Arteriosclerotic hypertension, mild. First-stage thoracoplasty 10/2/44. Died thirty-four hours after operation. Anesthesia: ether. Postmortem examination showed no apparent cause. Symptoms compatible with cardiac or embolic phenomenon.

12. E. R. Male, age 28. Had two stages of thoracoplasty. Vital capacity after second stage: 1,100 on 12/13/46. Continued to fail. Died of cardiovascular failure 4/28/47.

13. C. P. Male, age 54. Vital capacity: 3,550/4,750. Had three stages of thoracoplasty, last stage 4/1/47. Died 5/3/47 of a probable cerebral abscess resulting from otitis media. Anesthesia: cyclopropane (tube).

14. E. G. M. Male, age 53. Cavity, 8 cm., right upper lobe and fibrosis with slight infiltration, left upper. Died 1/24/47 four days after first-stage thoracoplasty. Patient had right hemiplegia after reacting from anesthesia. Anesthesia: sodium pentothal + N₂O + O₂ + C₂H₄. Postmortem diagnosis: apical pneumonitis from pulmonary involvement (tube).

15. S. P. Male, age 48. Vital capacity: 3,100/4,300. Three stages of thoracoplasty, last on 5/2/47. Died 6/2/47, amyloid disease. Anesthesia: cyclopropane (tube).

Excision Cases

16. R. D. P. Male, age 26. Cavity, right apex. Unsuccessful collapse after six-rib thoracoplasty. Lobectomy, right, 3/14/46. Fever, second day after operation, which continued until fifteenth day when sudden dyspnea with pulmonary edema caused death in a few hours. Patient was doing well up to this time. Anesthesia: ether (tube).

17. H. F. S. Male, age 52. Exudative disease throughout right side. Scattered calcified nodules throughout entire left side. Patient died on operating table from uncontrolled hemorrhage from pulmonary vein in an attempted pneumonectomy, right 8/20/47. Anesthesia: cyclopropane (tube).

18. J. G. N. Male, age 51. Generalized fibrosis, right; semi-opacity with contraction of hemithorax, left. Pneumonectomy, left, 11/28/47. Profuse hemorrhage from pulmonary artery on table. Calcified hilar glands. Cardiac arrest for three minutes. Convulsion and mental confusion postoperatively. Death on tenth postoperative day. Anesthesia: cyclopropane (tube).

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