THE CLINICAL ASPECTS OF PULMONARY ATELECTASIS * (WITH CASE REPORT OF A DEATH UNDER ANESTHESIA)

SEYMOUR SCHOTZ, M.D.†

Chicago, Ill.

THE literature is voluminous on the subject of pulmonary atelectasis. Most of the articles do little more than review what has been said many years before and consequently serve little purpose. The entire subject is not thoroughly understood and there is much room for careful research, both laboratory and clinical. Our interest in this subject was stimulated recently by the unfortunate occurrence of a death under anesthesia in which the postmortem findings revealed a complete collapse of both lungs. We shall refer to this case in more detail later.

Etiologically atelectases may be classified into two main groups: congenital and acquired.

CONGENITAL ATELECTASIS

Congenital atelectasis consists first of that group in which collapse of one or more lobes is present at birth and in which the lung tissue never becomes inflated. The usual course of events is somewhat as follows: The newborn fails to breathe properly from the time of delivery: suction of the trachea, plus inflation with oxygen, fails to relieve the cyanosis; the child lives from a few minutes to a few days. Postmortem examination often reveals not more than one-quarter of the available lung tissue expanded. The remainder presents the picture of the fetal lung with a generalized reddish-brown color, a marked vascularity and abundant fibrous connective tissue. While it is stated by some that these areas can be inflated with little force, other pathologists have noted that this atelectatic portion of the lung could not be inflated without rupturing the lung tissue. It seems that on the basis of the existing disease there is probably little that can be done for these cases. Maldevelopment of the lung tissue offers a complete barrier to its successful inflation.

There is a small group in which one lobe only is affected, and in these cases the subjects may develop quite normally to adulthood, the abnormality being discovered later during roentgen examination or at autopsy as an incidental finding.

* Read before a meeting of the Chicago Society of Anesthetists, May 28, 1942.
From the Department of Anesthesia, Mount Sinai Hospital, Chicago, Ill.

† Now Captain, M.C., U. S. Army Air Corps.

There is another group of cases in which bouts of cyanosis and convulsions appear, starting at any time from a few days to several weeks after birth. After several such bouts the infant usually dies from asphyxia or exhaustion. Autopsy reveals rather extensive atelectasis of the lung tissue. Here, as in the first group described, the incidence is higher in the premature or generally undeveloped newborn. The diagnosis is often difficult, being most commonly confused with hypostatic pneumonia, roentgen examination often offering little value. It is suggested that this complication can be prevented by changing the position of the premature or otherwise feeble infant from time to time, and that gentle friction and massage might help to stimulate respiration and prevent this form of atelectasis. Possibly these cases are related to another group which we will refer to later under the heading of "Acute Massive Collapse."

There is a fourth group in which atelectasis is incident to the inhalation of foreign material, such as amniotic fluid into the lower respiratory tract with subsequent obstruction and atelectasis. These cases belong to a group which we will refer to under the heading of Obstruction Atelectasis.

ACQUIRED ATELECTASIS

Acquired atelectasis may be subdivided into three classes: 1) obstruction atelectasis; 2) acute massive collapse; 3) compression atelectasis.

Obstruction Atelectasis.—After operation occasionally the following train of symptoms occurs: There is a relatively rapid elevation of temperature, moderate to marked increase in pulse and respiratory rates, dyspnea, and cyanosis to a greater or lesser degree. Commonly there is a desire to cough, but the patient withholds the cough for fear of pain in the operative area. In some cases an accumulation of secretions in the trachea takes place. In others, in which the patient fears to take a deep breath because of pain, these tracheal sounds are not apparent. Copious secretions may be present, but because of suppression of the cough reflex, these are not expectorated. Most frequently the onset is within the first twenty-four to forty-eight hours; pain may occur on one or both sides of the chest. In general the patient presents a picture not unlike that seen in the early stages of lobar pneumonia. Physical examination usually reveals one of two sets of findings. In one, palpation of the chest gives negative results; percussion demonstrates no significant changes, but on auscultation many fine to coarse râles are distributed widely over one or more lobes. Evidence of shift of the mediastinal structure cannot be elicited either by physical or roentgen examination. In the other, the findings are more remarkable. The intercostal spaces are retracted; there is dullness to percussion; markedly diminished to absent breath-sounds; a few fine crepitant râles; the roentgenogram shows a haziness over one or more lobes with

a shift in mediastinal structures toward the involved lung. Prompt and efficient treatment usually will restore either of these types to a normal status. These are two types of obstruction atelectasis. In one instance the obstruction is present simultaneously in a large number of scattered bronchioles. In the other the obstruction is in the main bronchus. The end result is the same—the absorption by the blood stream of the entrapped atmosphere with a collapse of the involved alveoli. The work of Coryllos and Birnbaum (1) proves that atelectasis can be produced in this manner, and the great majority of cases of atelectasis falls into this group.

Prevention.—Various factors, other than the anesthetic agent and technic, are responsible for obstruction atelectasis after operation. This fact is underscored by numerous reports in which the incidence of this complication is about the same under regional anesthesia as under general anesthesia. The rate of occurrence is distinctly higher in males and in operations involving the upper part of the abdomen. Too often an anesthetic agent which is irritating to the respiratory mucous membrane, such as ether, is proclaimed the sole cause of a particular case of atelectasis when other factors played a much more important role. In discussing the prevention of postoperative obstruction atelectasis, not only the postoperative but also the preoperative and operative periods must be kept in mind. Not infrequently the stage is set for atelectasis during these early periods.

First, the injudicious use of heavy preanesthetic medication is often the starting point. It should be recalled that: 1) the ciliated epithelium, which lines the lower respiratory tract, aids in removing mucus and foreign matter from the bronchioles; 2) slow peristaltic movements in the bronchioles, together with lengthening and shortening movements in the bronchi and bronchioles during inspiration and expiration (2), also aid in the expulsion of exudates and 3) an efficient cough reflex is one of the best safeguards to the respiratory airway. It is apparent that marked depression of these functions by large doses of nonvolatile preanesthetic drugs offers a serious hazard and may be a contributing factor in the subsequent development of obstruction atelectasis. Second, the failure to use belladonna derivatives to dry the mucous membrane might result in the stimulation of secretions during inhalation anesthesia which might be difficult of removal during the anesthesia. Third, the failure to treat existing respiratory infections properly before operation and anesthesia increases the hazard of all postoperative respiratory complications.

During operation varying conditions arise which, if allowed to go unnoticed and untreated, increase the dangers of postoperative obstruction atelectasis. Under inhalation anesthesia the cough reflex is of necessity abolished; practically all anesthetics in deeper planes of anesthesia relax the bronchial musculature and depress the activity of the cilia. Partial respiratory obstruction leads to the formation and

accumulation of mucus in the bronchi, bronchioles and alveoli. The anesthetic gases are rapidly absorbable (much more so than air) from the alveoli and an inefficient tidal exchange may result in the collapse of scattered alveoli, which if allowed to remain in that state for some time cannot again be easily opened at the end of anesthesia. These factors during inhalation anesthesia aid in producing atelectasis. The maintenance of a perfect airway, including the efficient removal of any accumulating secretions together with an adequate tidal exchange at all times, will circumvent most of these undesirable effects of inhalation anesthesia. It has also been suggested that with potent agents such as cyclopropane or ether, nitrogen or helium be used as a diluent since both are very slowly absorbed from the lungs and that this would aid in preventing the collapse of the alveoli.

The conception of some surgeons that any anesthetic which is not inhaled is safe for the lungs is fallacious. Atelectasis which follows operations under various regional procedures occurs about as frequently as following general anesthesia. Here the factors which operate are different. During operations under local, regional or spinal analgesia it is usually necessary to give large quantities of sedatives to the patient in order that he does not become restless, especially if the operation is time-consuming. As mentioned before, nonvolatile drugs in larger amounts interfere with normal respiratory physiology producing central depression. If to this are added the ineffectiveness of the cough reflex and paralysis of at least one-half the intercostal muscles under spinal analgesia for an effective abdominal block, the conditions for bronchiole stasis with obstruction and atelectasis are ideal. Practically, the anesthetist has less control than under inhalation anesthesia. He can only in part help to prevent atelectasis by the judicious use of sedatives plus frequent instructions to the patient to take deep breaths.

Most patients believe that if they lie perfectly quiet after operation they will get better more quickly. Unfortunately this idea is shared by some surgeons who routinely prescribe large doses of morphine at the first sign of pain. If anything, this type of therapy actually delays convalescence. Much that already has been said applies here again. The main strategy is to prevent bronchiole stasis. The tactic may have to differ from patient to patient. Since atelectasis more commonly follows upper abdominal operations it is wise to keep in mind the following facts: First, incisions of the upper abdomen tend reflexly to bring about a splinting of the corresponding side of the diaphragm. The diaphragm assumes a high position. The patient tends to restrict his respiratory activity inasmuch as deep breathing is painful. As with any abdominal operation the patient tends to lie in one position, for moving about increases pain. If secretions are present, he refrains from coughing, for again this act is painful. The dependent portions of the lung remain relatively immobile and secretions tend to accumu-

late in the lower respiratory tract. These facts explain the higher incidence of atelectasis in the lower lobes. The successful prevention of atelectasis therefore depends upon just how adequately this undesirable state of affairs is overcome. One of the most satisfactory methods has been to institute a systematic regimen of what has been called "stir-up." This is a simple procedure. It consists of three features: 1) that the patient have a full change of position at least every hour, that is from side to side; side to back, or back to side; 2) that he be made to take deep breathing exercises for a few minutes at least every hour while he is awake; 3) that he be encouraged to cough and clear the respiratory tract of any accumulating secretions. The latter may appear to be difficult for one who has recently undergone an abdominal surgical procedure, but a little encouragement and explanation on the part of the anesthetist will usually bring about the desired degree of cooperation. In patients who will not take deep breaths either because they will not cooperate or because they are drugged into semiconsciousness, the use of carbon dioxide is recommended, allowing some pure carbon dioxide to spray over the face until hyperpnea develops. The procedure is repeated about every hour. It is also suggested that the paravertebral injection of the lower six intercostal segments with some oily anesthetic agent will aid in relieving the discomforts of a high abdominal incision and will allow the patient to take deep breaths and thus better ventilate the lungs. The judicious use of postoperative sedatives should be emphasized. Morphine and the allied opiates depress respiratory activity. It has been our experience that relatively small doses of morphine produce satisfactory analgesia but beyond these small doses all that is accomplished is central medullary depression. Actually, it is quite easy, especially in the old and debilitated subject, to administer enough morphine to produce a certain amount of cerebral anoxia and consequent restlessness. The nursing and resident staffs should be trained to recognize the difference between restlessness from pain and that which is due to too much sedation. Oftentimes a patient is given too much morphine, becomes restless, more morphine is given to relieve the restlessness and if restlessness is not relieved within fifteen minutes, another dose is given. About a half hour later an emergency call comes that the patient is not breathing properly. We have found that satisfactory analgesia can be achieved with doses of morphine of from $\frac{1}{10}$ grain to $\frac{1}{6}$ grain, repeated when needed, without producing central medullary depression. It is far better to give $\frac{1}{6}$ grain of morphine every two hours if needed than to give $\frac{1}{4}$ grain every four hours. This point on morphine cannot be overemphasized.

Treatment.—Once the diagnosis of postoperative atelectasis has been made, prompt action is imperative. The sooner the bronchial tree is rid of accumulated secretions, allowing for better expansion of lung tissue, the better are the chances of avoiding any further complication

such as pneumonia. The most efficient treatment for postoperative atelectasis is suction bronchoscopy. In a few institutions the department of anesthesiology has taken over this treatment. They recommend the careful topical anesthetization of the pharynx and larynx, the introduction of a large caliber endotracheal catheter under vision and sucking out the bronchial tree as far down as it may be approached with a small suction catheter. They report a high degree of success in the management of postoperative atelectasis. Personally we practice a somewhat simpler technic in which a No. 23 Magill catheter is passed through the nose into the trachea without any anesthetization. This is not difficult to accomplish with a little cooperation. It will invariably stimulate a vigorous cough reflex and the tracheobronchial tree is sucked free of any secretions through this catheter. Following this, the above-mentioned "stir-up" should be even more vigorously applied. It has been suggested that the use of carbon dioxide by helping to liquefy secretions is of aid in the treatment of postoperative obstruction atelectasis. The technic for administering carbon dioxide in this instance may be the same as mentioned earlier. It has an added advantage in that pure carbon dioxide is somewhat irritant and will stimulate a cough reflex. During the acute phase when cyanosis may be present oxygen therapy, either by nasal catheter or oxygen tent, should be instituted. It is fortunate that well over 90 per cent of patients with postoperative atelectasis of this nature will recover without serious sequelae, even if suction bronchoscopy has not been used. It is true however that what has been termed adequate "tracheobronchial toilet" is of distinct aid in hastening the convalescence.

ACUTE MASSIVE COLLAPSE

CASE REPORT

A middle-aged woman appeared for vaginal hysterectomy. Upon physical examination she appeared to be normal in all respects except that she was very obese, and she had had a temperature of 101 with a leukocyte count of 12,000 the evening before operation. She received two doses of morphine $\frac{1}{6}$ grain and scopolamine $\frac{1}{150}$ grain as ordered by her physician ninety minutes and forty-five minutes, respectively, before operation. Cyclopropane was employed. Anesthesia was started by filling the bag about half full with oxygen and then allowing the cyclopropane and oxygen to flow each at 500 cc. per minute. After about two minutes the flow of cyclopropane was reduced to 300 cc. and was maintained at that rate. The anesthesia seemed to progress uneventfully. The tidal exchange was adequate although the respiratory rate was between 10 and 12 per minute. About ten minutes after the start of anesthesia the surgeon snared off a cervical polyp and then injected 1.5 cc. of pituitrin into the uterus. This is a common practice among some gynecologists to obtain a dry field. About three to five minutes after the injection of the pituitrin the anesthetist noticed that the patient's color became distinctly blue. She reached over to flush the bag with oxygen and when she turned back the patient had ceased breathing. Arti-

ficial respiration was begun immediately. An endotracheal catheter was introduced and artificial respiration by intermittent bag pressure was carried on for about a half hour, together with attempts at stimulating cardiac action with cardiac puncture. No heartbeat could be heard and after a half hour of resuscitative attempts in which, toward the end various stimulative drugs were used, the patient was pronounced dead. We had seen several cases of pituitrin shock and we were certain that this was the case in which recovery did not take place. However, necropsy revealed massive collapse of both lungs except for a small portion of the right middle lobe. Grossly each lung had shrunk to about one-third its normal size and lay up against the hilum. The lung was bluish-brown in color. There was no evidence of any tracheobronchial obstruction. Large circumscribed hemorrhagic patches were present over the surface of the lung. Histologically the lung showed a combination of atelectasis, edema and hemorrhages into the alveoli, the latter being a marked feature. Edema was found between the heart muscle fibers and, in addition, the right heart showed marked fatty infiltration.

Here was a case of what has been termed acute massive collapse. We have purposely used this term because we believe there is a distinction at least pathogenetically between this condition and atelectasis of the obstructive type. Jones and Burford (3) in reporting 4 cases state that this complication arises through a rapid absorption of anesthetic gases from the lung without their being efficiently replenished through adequate respiratory exchange. We are not in agreement with this conception, for one of the commonest features of all cases reported similar to this one has been the sudden onset and rapid termination of events. Lemmer and Rovenstine (4) reported accurate data on the rapid absorption of anesthetic gases in the lung lobule of the dog when the corresponding bronchus was occluded. However, in the absence of a plugged bronchus and with an adequate tidal exchange, we cannot conceive of the absorption of oxygen taking place from the lung so rapidly that the onset of anoxia could not be detected earlier. In this instance there was no evidence of any obstruction and the collapse was exceedingly rapid. We believe, with certain British writers, notably Rose-Bradford (5) and Morrison (6), that acute massive collapse can take place through some neurogenic mechanism, that the lung is a muscular organ capable of undergoing active contraction. This is well borne out in the many cases reported by Bradford in which relatively minor injuries to one side of the chest resulted in an acute collapse of one or more lobes of the lung on the opposite side of the chest. Morrison believes it is some type of vagal reflex. Certainly in this case three potent vagal drugs had been used, that is, morphine, cyclopropane and pituitrin. Subsequently we discovered that this patient had hay fever and had had some attacks of bronchial asthma.

COMPRESSION ATELECTASIS

It is well to keep in mind one other form of collapse which has been referred to as compression atelectasis. The mechanism is the simple

compression of lung tissue from without by the pressure of fluid, tumors or the presence of a very high diaphragm. Peritoneal infections or the accumulation of a pocket of air below the diaphragm may produce many of the signs which are usually associated with atelectasis of the obstructive nature. One case occurred quite recently in which the surgeon was convinced that we had given his patient a complete atelectasis of the right lower lobe. At necropsy generalized peritonitis with a very high diaphragm and compression of the lung tissue of both lower lobes was found. Before the diagnosis of obstructive atelectasis is established, it is wise to make a careful examination of the abdomen.

SUMMARY AND CONCLUSIONS

The clinical features of the various types of atelectasis have been presented with a view toward establishing a clearer picture of the etiology, pathogenesis, prevention and treatment. Through careful attention to details of normal physiology the dangers of this complication, at least postoperatively, may to a great measure be obviated. Prompt and efficient therapy will hasten convalescence and prevent sequelae.

A report of a case is given in which sudden death under anesthesia was caused by bilateral massive pulmonary collapse. The etiology was discussed.

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