FATAL MASSIVE PULMONARY COLLAPSE DURING SPINAL ANESTHESIA *

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ATELECTASIS is still a perplexing problem. For a better understanding of the relationship of cause and effect and a more rational approach to treatment, considerable laboratory and clinical investigation is required. At present, the treatment of obstructive atelectasis is limited either to prophylaxis or to tracheobronchial aspiration by catheter or bronchoscope. Although the latter technics have become effective in the hands of the trained anesthesiologist, there are still those cases, as in the following reported, which present a challenge for explanation and treatment.

Fatal massive pulmonary collapse during spinal anesthesia has not been reported in the literature. It is the purpose of this paper to report three such cases, to suggest a possible explanation, and to offer for consideration some form of treatment for such a potentially fatal condition, as well as to add to those cases of massive collapse already reported during local or general anesthesia.

Pulmonary collapse or atelectasis is defined as a condition of the whole or part of the lung in which there is a return to an airless state. It may be lobular (easily mistaken for bronchopneumonia), lobar, or total, involving both lungs. The term "massive collapse" is used to describe a large deflated area or a subtotal collapse of one or both lungs.

Massive collapse was first described by Pearson-Irvine (1) in 1876. Pasteur (2), in 1890, described 30 cases as a complication of postdiphtheritic paralysis of the diaphragm. Judging from the literature little attention was paid to this condition until Pasteur (1908–1911) (3, 4) first described its occurrence after abdominal operations. In 1914 Pasteur (5) again reported 16 postoperative cases. Since that time massive collapse has been described many times, both as a postoperative complication and as a complication of pneumonia, lung abscess, trauma to the chest, and other types of pulmonary disease.

Lobular atelectasis is a frequent postoperative complication, but massive collapse is unusual and its incidence is reported as representing less than 0.2 per cent of all postoperative pulmonary complications. In a series of 29,648 operations reported by Waters (6) the incidence was 0.142 per cent. Scrimger (7) noted 7 cases in 540 operations.

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Like atelectasis, massive collapse is usually a benign condition occurring hours or days after surgical operation. In one of Scrimger's (7) cases the onset was thirteen days after operation. It has been reported following all types of anesthetic technics, local, spinal, and general, with a minimal immediate mortality, the fatal cases terminating hours or days later. Not until 1927 (8) was a fatal case described as occurring during operation. Since then other fatalities (to be described below) during operation have been reported. In these instances, either local or general anesthesia was used. Deaths during spinal anesthesia have been reported (9, 10, 11, 12), but none in which nonobstructive massive collapse occurred as a fatal complication. The following 3 cases demonstrate that this complication may occur during spinal anesthesia, and can cause a fatal result.

Case 1.—A 59-year-old, white, laundry worker was admitted to the hospital on November 30, 1946, with a history of sudden onset of lower abdominal pain of eight hours' duration. In 1944 an umbilical hernia had been repaired. He had been symptomless until the morning of admission; his condition became progressively worse during the next few hours. There was no nausea, vomiting or melena.

Physical examination revealed that the patient was lethargic; he could be aroused for questioning, at which time he was oriented and cooperative, but then promptly went back to "sleep." There was no history of medication. The other significant findings were marked kyphosis and emphysema, some lower abdominal distention, tenderness, and muscular spasm. The temperature was 100.6, pulse 96, respirations 24, and the blood pressure 130 mm. systolic and 78 mm. diastolic.

The impression on admission was a partial intestinal obstruction with strangulation of bowel secondary to postoperative adhesions. A roentgenogram of the abdomen did not disclose significant findings. The blood urea was 23.9 mg. per 100 cc., hemoglobin 85 per cent, erythrocytes numbered 4,500,000 and leukocytes 16,400 with 60 polymorphonuclear leukocytes, 22 band forms, 16 lymphocytes, and 2 monocytes. The urine showed 4 or 5 leukocytes per high power field. A repeat film of the abdomen taken sixteen hours after admission revealed the presence of gas-filled loops of small bowel and a few isolated loops of large bowel. The patient was brought to the operating room at 3 p.m. on December 1, 1946 with a preoperative diagnosis of acute appendicitis and possible intestinal obstruction.

The patient was inadvertently premedicated with morphine sulfate, 1/6 grain (11 mg.) and scopolamine, 1/150 grain (0.4 mg.), at 2:30 p.m. The anesthetist first saw him on the operating-room table at 3 p.m. at which time the blood pressure was 140 mm. systolic and 80 mm. diastolic and the pulse 92. Because a long procedure was anticipated, 1 cc. of 1 per cent pontocaine solution (10 mg.), 1 cc. of 5 per cent ephedrine solution (50 mg.), and 1 cc. of 10 per cent glucose were slowly injected into the third lumbar interspace at 3:10 p.m.with the patient in the left lateral position and the table horizontal. Ephedrine (50 mg.) was given intramuscularly immediately before the spinal anesthetic agent was administered.

It was thought that spinal anesthesia was indicated because the level required was not unduly high (about the seventh dorsal) and because spinal

anesthesia at that level tends to disturb the body chemistry less than the inhalation agents.

The patient was able to cooperate with the orderly in assuming the position as described and he was able to resume the supine position unaided after the completion of the injection. At this time his color was good. At 3:22 p.m., twelve minutes after administration of the spinal agent, the level of anesthesia

FIG. 1. Respiratory system

The right lung weighed 140 Gm., the left 110 Gm. The external surfaces were smooth and glistening. The right lung was completely atelectatic and the left was crepitant only in its upper lobe. The trachea and bronchi were normal, their lumen clear.

was determined by pinprick to be at the eighth dorsal segment. Operation was started immediately because this level was considered adequate for the contemplated McBurney incision.

The blood pressure dropped at 3:25 p.m. to 85 mm. systolic and 55 mm. diastolic and the pulse to 64. Neosynephrine, 2 minims (0.06 cc.), was injected into the intravenous tubing. At 3:30 p.m. the blood pressure was 90 mm. systolic and 60 mm. diastolic and the pulse 80. At 3:35 p.m. the blood pressure

rose to 100 mm. systolic and 60 mm. diastolic; the pulse remained at 80. During this time the appendix was found to be normal. Further exploration disclosed a knuckle of small bowel caught in a pocket in the previous hernial scar. Considerable difficulty was encountered in separating the bowel from its attachment to the hernial sac, and a walled-off perforation was found. At several points during the high abdominal exploration the patient evinced discomfort by facial contortions and groaning. At. 3:40 p.m. the blood pressure fell again to 90 mm. systolic and 60 mm. diastolic and the pulse rose to 92. Oxygen by mask was administered although no change in either color or respirations had occurred.

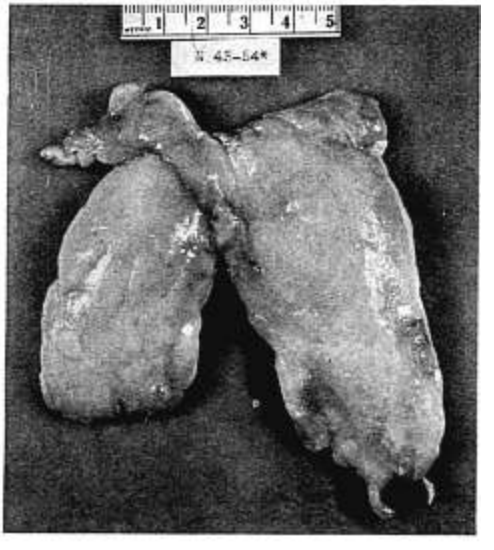

FIG. 2. Thymus

The thymus weighed 44 Gm. (normal about 30 Gm.). Its capsule was delicate and the gland was soft in consistency. The external and cut surfaces were grey.

About 3:42 p.m. the respirations suddenly became irregular and gasping; neither pulse nor blood pressure could be obtained, and then spontaneous respirations ceased. In spite of oxygen by manual compression on the bag, cardiac massage through the diaphragm, and administration of coramine into the intravenous tubing, the patient became deeply cyanotic and was pronounced dead at 4:10 p.m.

The autopsy findings were: (1) Massive pulmonary atelectasis, bilateral. The airway was not obstructed, the pharynx was clear. No vomitus, mucus, or aspirated material was present in the bronchial passages. (2) Coronary sclerosis was most marked in the anterior descending branch; there was no occlusion.

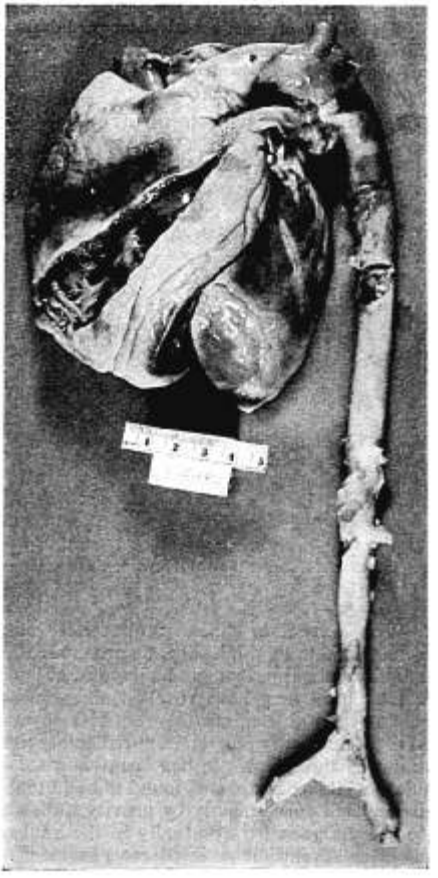

FIG. 3. Heart and great vessels

The heart weighed 250 Gm. and there were no unusual findings. The aortic orifice measured 5 cm. in circumference. The aorta measured 3 cm. in circumference in its descending portion and was hypoplastic.

(3) Early, diffuse, fatty change was present in the liver. (4) In the kidney the corticomedullary markings were obscured and there was marked vascular congestion. (5) A 6 cm. loop of ileum was edematous and dusky, with a perforation 1 cm. in diameter on the mesenteric border. There was a mesenteric unruptured abscess about 4 cm. in diameter, with thick green pus. A mesenteric thrombosis was not found.

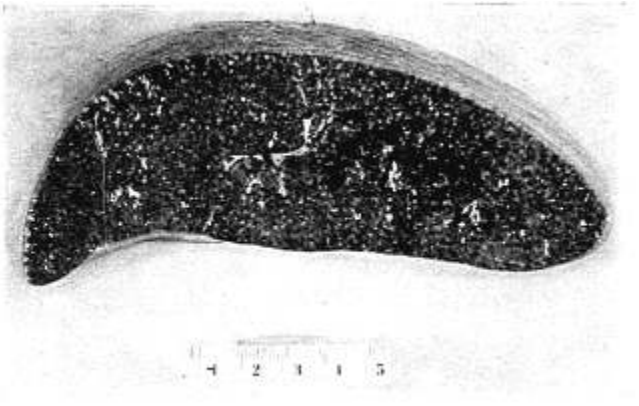

FIG. 4. Spleen

The spleen weighed 450 Gm. (normal 100–150 Gm.). It was firm and purple blue in color. The cut surfaces were purple red and the malpighian corpuscles were prominent.

Comment.—The immediate clinical impression was that the level of anesthesia had risen to the high thoracic or cervical levels thus producing death, with minimal pathologic findings at autopsy. It was believed that a high level may have resulted since, in our experience, pontocaine-glucose-ephedrine mixtures often rise to undesirably high levels because of an unusually long and unpredictable time required for complete fixation of the agent. It has been shown, however, that a hyperbaric solution (this mixture was found to be 1.0188) moves up and down the subarachnoid space mainly by gravity, following the natural curvatures, to become pocketed eventually in the lumbar and thoracic curvatures (13), the seventh or sixth thoracic segment normally being the most dependent portion. Since this patient had a marked kyphosis, and since the injection was performed slowly at the third lumbar interspace in the lateral position, and since the patient was kept flat on the table and the table horizontal, it is hardly conceivable that the level could have risen higher than the sixth thoracic unless other unusual factors were operative. Furthermore, the patient winced

and groaned when the upper abdominal exploration was performed approximately thirty minutes after the start of the anesthesia and only two or three minutes before the signs of extremis became obvious. These points raise justifiable doubt that the level rose higher than the seventh or sixth thoracic segment.

It was suggested at autopsy that the bilateral massive pulmonary collapse could have been the result of pneumothorax resulting from the passage of air through alveolar emphysematous blebs into the pleural space during the positive pressure manipulations used for resuscitating the patient. The pathologist thought that this was impossible inasmuch as no ruptured blebs could be demonstrated.

FIG. 5. Mesenteric lymph nodes

The mesenteric lymph nodes were enlarged up to 2.5 cm. in diameter. They were moderately firm and pink grey in color.

The thought also occurred that the lungs might have collapsed after death. The following statement, "Instead of collapsing as does the normal lung, the emphysematous lung, being in a permanent state of inspiratory inflation, remains distended even after removal from the chest, and may even project" (14), indicates that this state could hardly have occurred after death.

It may be stated summarily that this patient demonstrated signs and symptoms which can contribute to atelectasis, massive or partial.

These signs indicated a profound toxemia as evidenced by the lethargy, general malaise and low temperature reaction, respiratory depression due to the emphysema, the premedication, and the intestinal distention. The spinal anesthetic agent with its paralysis of the lower intercostal muscles together with the complete loss of abdominal muscular support would prohibit any expulsive respiration to remove accumulated secretions. The ciliary activity was depressed by the premedication. All these might conceivably cause an accumulation of bronchial secretion with consequent absorption of air and atelectasis. However, pathologic examination did not reveal the presence of any obstruction in the bronchial tree.

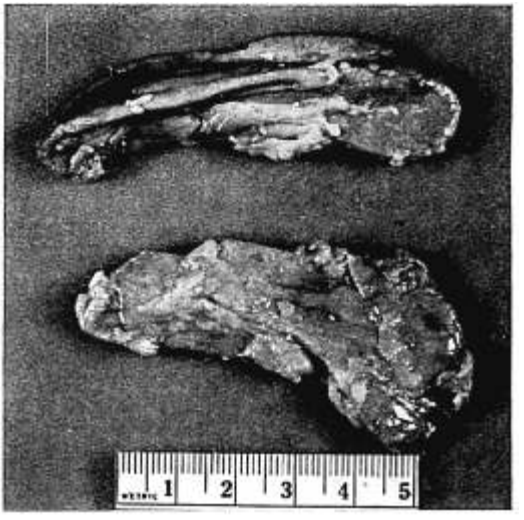

FIG. 6. Adrenals

The adrenals together weighed 9 Gm. (normal about 12 Gm.). On section, the medulla was narrow.

The thought was expressed that the manual compression of the bag during resuscitation may have produced pneumothorax with resultant atelectasis. Macklin (15), working with cats, demonstrated mediastinal emphysema and pneumothorax following the use of positive pressure during intratracheal anesthesia. It was his belief that this technic contributed to tears in the alveolar walls so that the air traveled along the vessels in the interstitial tissue to the root of the lung and into the

mediastinum, and thus the pneumothorax was secondary to the emphysema. However, stained microscopic sections failed to show lacerated alveolar walls, but did show emphysema in the perivascular sheaths. The pressures used varied from 10 to 220 mm. of mercury. Adams (16) and Coryllos (17), working independently, showed that intrabronchial pressures of 52 to 58 mm. of mercury were necessary to produce a rupture of visceral pleura. Marcotte, Phillips, Adams and Livingstone (18), working on dogs, used intrabronchial pressures varying from 16 to 110 mm. of mercury. They found that pressures of 24 mm. or over were routinely accompanied by the development of emphysema, usually only along the great vessels at the hilum. Intrabronchial pressures of 18 mm. or less seldom if ever caused mediastinal emphysema in dogs. It has been the authors' experience that with a patent airway it is impossible by manual compression of a mildly distended bag to obtain pressures higher than 10 mm. of mercury in the human. These laboratory findings mitigate against the possibility that pulmonary emphysema and pneumothorax constitute the cause of the bilateral massive pulmonary collapse either just before or immediately after death.

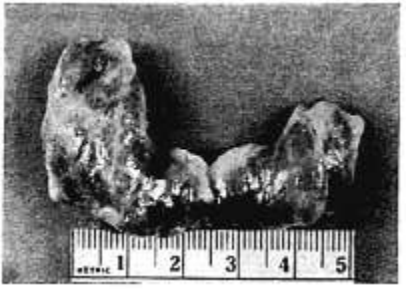

FIG. 7. Thyroid

The thyroid gland weighed 8 Gm. (normal about 30 Gm.). It was enclosed in a delicate capsule. The external and cut surfaces were pink red and homogeneous in appearance.

Case 2.—A 13-year-old boy was admitted to the hospital March 11, 1943, with a history of an upper respiratory infection and a nonproductive cough for one week, nausea and abdominal pain for three days with localization in the right lower quadrant during the past twenty-four hours. There was no vomiting.

Physical examination showed that the patient was obese with the appearance of Fröhlich's syndrome. There was mild pharyngitis, some tenderness in the left upper and right lower quadrants, with muscle spasm and rebound tenderness. The first blood count at 4:30 p.m. showed 8750 leukocytes, 83 polymorphonuclear cells, 13 lymphocytes, and 4 monocytes. The repeat blood count at 10 p.m. showed 7850 leukocytes, with 76 polymorphonuclear cells, 24 monocytes,

4,000,000 erythrocytes and 88 per cent hemoglobin. A preoperative diagnosis of acute appendicitis was made.

Operation was performed March 12, 1943. At noon, morphine sulfate, 1/6 grain (11 mg.), and scopolamine, 1/150 grain (0.4 mg.), were given. The blood pressure was 110 mm. systolic and 60 mm. diastolic, and the pulse 96. He was placed in the left lateral position and 12 mg. of pontocaine in 1.2 cc. and 1.2 cc. of 10 per cent glucose were injected through the third lumbar interspace at 1:05 p.m. Prophylactically, 50 mg. of ephedrine was given intramuscularly before the spinal agent. Just before beginning the operation at 1:10 p.m., the level had risen to the seventh dorsal segment, the blood pressure was 95 mm. systolic and 60 mm. diastolic and the pulse was 80. A McBurney incision was made and a normal appendix was found. The mesenteric lymph nodes were enlarged. The appendix was removed and the stump was being buried when the patient suddenly ceased to breathe at 1:25 p.m. Oxygen by mask under intermittent positive pressure was immediately instituted. His color improved temporarily but soon a mottled cyanosis developed and he was pronounced dead at 2:30 p.m. During the surgical procedure the systolic pressure stayed at 95 mm. while the diastolic pressure rose from 60 to 65, then to 75 just a few minutes before the cessation of respiration; the pulse remained at 80 throughout.

The autopsy findings were obesity; hyperplasia of the thymus; hypoplasia of the pituitary, thyroid, adrenals, aorta, testes, and penis; splenomegaly; enlargement of the mesenteric lymph nodes and fatty changes in the liver. There was massive pulmonary collapse of the entire right lung and of the left lower lobe.

Comment.—In this instance the immediate cause of death was also massive collapse. The improvement of the color of the skin with the administration of oxygen under positive pressure implied that the circulation was probably still slightly active after the pulmonary collapse. The size of the thymus might have played a contributing role if it had caused any tracheal or bronchial obstruction. No sign of obstruction or even indentation could be observed at autopsy.

This patient was unusually large for his age and was therefore treated as an adult in determining the dosage of both the premedicant and the anesthetic agent. The exact level to which the anesthesia finally ascended is not known, but it is still difficult to explain the massive pulmonary collapse solely on the basis of anesthetic level or dosage.

It is significant, however, that this patient showed signs of a generalized infection and a marked endocrine dyscrasia.

Case 3.—A 19-year-old colored primipara was admitted to the hospital 11:30 a.m. on April 18, 1947 in active labor at term. The pregnancy had been completely uneventful except that she had a 4 plus Wassermann; adequate treatment was given.

On admission, the fetal head was still over-riding the pelvis after fourteen hours of labor. A roentgenogram showed a brow presentation; the pelvis was small and flattened with android tendencies. There were no other abnormal findings. Conservative treatment was decided upon and the patient was observed for the next thirty-one hours during which time mild ineffectual pains every five to seven minutes dilated the cervix to 3 cm. At 7 p.m. on April 19 the patient was found to be hysterically incoherent, thrashing about in bed.

The temperature was 102.4° F. and the pulse 128. The fetal heart was regular at 142. A diagnosis was made of fatigue, dehydration and possible infection and the patient was given parenteral fluids and 50,000 units of penicillin intramuscularly every three hours. At 12:30 a.m. on April 20 the temperature was 103, the pulse 112, and the blood pressure 155 mm. systolic and 90 mm. diastolic. The urine showed both albumin 1 + and acetone 1 +. At 2 a.m. the temperature had decreased to 101, and the pulse to 88, and at 3 a.m. the temperature was 99.8 and the pulse 84. The fetal heart was still normal and the membranes were intact. Cesarean section was then proposed and fractional spinal anesthesia was chosen for the anesthetic technic. The solution used was 500 mg. of procaine in 10 cc. solution plus 2 cc. of 10 per cent glucose, each cubic centimeter of the resultant solution containing 42 mg. of procaine. With the table level and the patient in the left lateral position a Lemmon malleable needle was inserted into the third lumbar interspace. At 5 a.m., 1.5 cc. of solution, containing 62 mg. of procaine, was slowly injected. Immediately thereafter the patient was returned to the supine position. She complained of weakness and nausea and the level of anesthesia was now found to be at the fourth thoracic segment. Three cubic centimeters of spinal fluid was withdrawn and intermittent positive oxygen pressure by mask was started. At this time there was complete absence of spontaneous respiration, pulse, and blood pressure. In spite of artificial respiration as described, and intravenous and intracardiac epinephrine, neither respiration nor circulation could be re-established and the patient was pronounced dead at 5:30 a.m.

The autopsy findings were: (1) Heart—minimal hypertrophy. (2) Brain —weight 1665 Gm., above the maximum limit of normal. There was a slight increase in pericellular and perivascular spaces. Normal contact between nucleus and cytoplasm was obscured, and the cell and nucleus had lost their sharp contour. (3) Uterus—inflammatory reaction in the endometrium and myometrium. (4) Respiratory tract—the trachea and bronchi were normal. (5) There was massive collapse of both lungs except for a small area of the right upper lobe.

Comment.—In this case there was a definite indication of a very high level of spinal anesthesia with a small dose of intrathecal procaine. It is believed that the high level in this instance was due to several factors. In the supine position, the exaggerated lordosis of the parturient tends to change the most dependent spine of the thoracic curve from the sixth or seventh thoracic to the fourth or fifth thoracic so that hyperbaric solutions tend to rise to the higher levels. Secondly, a uterine contraction occurring simultaneously with the subarachnoid injection would increase the intracranial pressure so that the cerebrospinal fluid would be forced down into the spinal subarachnoid space. Termination of the pain should cause a reversal of the pressure gradients and the fluid dynamics with a consequent reflux of cerebrospinal fluid into the cranium. This kinesia would cause a greater dispersion of the injected procaine than is usual. Thirdly, it was thought that the higher levels of anesthesia could obtain during the engorgement of the rich venous plexus in the epidural space which might compress the spinal subarachnoid space sufficiently to decrease markedly its capacity.

In our clinic fractional spinal anesthesia has been induced for over 700 cesarean sections of all types. With this technic an adequate level, the tenth or eleventh thoracic, can be obtained very slowly. This allows the compensatory mechanisms to sustain blood pressure levels, obviating to a large extent the severe, temporary hypotensive states obtained by the single injection method. The mechanisms causing more marked blood pressure falls during spinal anesthesia in the parturient than in the same patient when she is not pregnant are not clear but are probably related to a sudden reduction in circulating blood volume. The placenta represents a large, low resistance shunt which may contain up to one-sixth of the blood volume (19). The interruption of the vasoconstrictor fibers to the uterus, placenta, abdominal and pelvic viscera, and the lower extremities, could cause a sudden large loss of circulating blood volume by pooling to result in severe hypotension.

Another factor that requires consideration is that in a parturient, the supine position and the paralyzed abdominal and intercostal muscles reduce the tidal exchange markedly, interfering with the venous return and with respiratory excursions. The latter is conducive to retention of secretions with bronchial obstruction and consequent atelectasis. Here again there was nothing to indicate bronchial obstruction. All passages were clear. In this case it is noteworthy that the elements of severe toxemia, fever, exhaustion (fifty-five hour labor), hyperreflex irritability with emotional distress and consequent high oxygen were all operative. The overweight of the brain brings in another factor which requires consideration but is not clear in its import. However, King (20) and others have shown that a patient who is such a poor surgical risk is more prone to develop pulmonary complications.

DISCUSSION

A summary of the outstanding features of these cases is necessary in order to reduce the various factors to their common denominator. The significant findings are temperature with generalized toxic manifestations, spinal anesthesia with a known high level in one, cessation of respiration and cardiac action almost simultaneously, intraperitoneal manipulation in 2, severe apprehension and fear in one, and autopsy findings of almost complete bilateral pulmonary atelectasis in all 3 cases. Case 2 presented, in addition, an imbalance of the endocrine system which is probably an immediate contributing factor to an imbalance of the autonomic nervous system.

A high level of spinal anesthesia cannot satisfactorily explain the pulmonary collapse even if the level were high enough to paralyze all the intercostal muscles and the diaphragm. Koster and others (21, 22, 23) have administered spinal anesthetic agents to sufficient height to permit mastoidectomy, thyroidectomy, and radical mastectomy without loss in consciousness, although it is not denied that severe, precipitous drops in blood pressure have occurred. Such levels do not seem to

paralyze the medulla. Even if the medulla had become anesthetized and a fatality occurred, it would be difficult to explain how a bilateral pulmonary collapse could result. The oxygen could be completely absorbed from the alveoli within a few minutes during the static stage of respiration with a functioning circulation, but the nitrogen would require hours for its complete absorption. If these deaths were attributed to a high level of anesthesia alone, the lungs should have remained inflated.

Paralysis of the respiratory musculature could produce an asphyxial death, but it would not explain the pulmonary collapse or the coincident cardiac arrest. In the acute asphyxia of hanging, drowning or strangulation the circulation is usually maintained for a short time before cardiac cessation. To explain simultaneous cardiac and respiratory cessation a derangement common to both must be present, based on the same pathophysiologic basis.

King (20) studied 2646 surgical cases and found that atelectasis occurred more frequently after laparotomy and herniorrhaphy (13 per cent) than after other operations (1 per cent). The incidence was higher in the presence of a pre-existing perforation, and increased directly in proportion to the amount of sepsis present. Over a two-year period (1931–1932) atelectasis occurred after ether anesthesia in 12.4 per cent, after spinal anesthesia in 16.7 per cent, and after local anesthesia in 18.4 per cent. A careful analysis of the cases showed that "the preoperative condition is a more definite factor in the development of pulmonary complications than the length of operation or the type of anesthesia."

A search of the literature revealed that in most cases of massive atelectasis reported there were coincidental findings of some obstruction in the bronchial tree. In many cases, as in the following, however, obstruction within the bronchial tree was not demonstrable.

During World War I Bradford (24) collected a large series of cases of massive collapse following gunshot wounds in which there were no histories of previous lung disease. The collapse was usually on the wounded side but sometimes on the contralateral side. Many of the wounds were not incapacitating and trivial, either penetrating or nonpenetrating. He also found collapse with nonpenetrating wounds of the abdominal wall, buttocks, pelvis and thighs. There were no such cases following wounds of the head or arms. All diagnoses were made by the roentgenogram. On the basis of this experience, Bradford defined massive collapse of the lungs as "an unusual condition in which the lung, without the presence of any gross lesion such as bronchial obstruction, pleural effusion, etc. interfering with the free entry of air becomes airless to a greater or lesser degree and, therefore, useless for respiratory purposes." There were no fatalities. He concluded that "it is thus clear that the essential factor in the etiology is the wound, and that the condition may ensue even in its most marked form where

other supposed factors such as anesthesia, posture, injury to the diaphragm or its nerves, exudation, secretion, or blood in the bronchial tubes can be excluded. . . . The mechanism is obscure but is possibly of reflex nervous origin."

Santee (25) reported 2 fatal cases of massive collapse, one a laparotomy with death on the table, the other a tonsillectomy under local anesthesia. In both cases there was no demonstrable obstruction. In discussing the laparotomy he said, "The cause is not known but it seems most probable that some infection or insult to the region of the vagus supply produces a reflex action on the small bronchioles permitting their temporary collapse."

Bergamini and Shepard (8) reported 2 cases with sudden death during operation. One case was strikingly similar to Case 1. "The chest was perfectly normal. There was no indication of any respiratory disturbance. The patient was nearly awake when she stopped breathing. This extreme rapidity of onset in itself seems to rule out the theory of obstruction of the bronchi with subsequent absorption of the alveolar air as the etiological factor in this case." In discussing the pathologic findings in the lungs they commented that "the uniform dilatation and engorgement of the capillaries, arterioles, and venules strongly suggest the probability of a vasomotor disturbance. At the same time the swollen, hydropic appearance of the epithelial cells lining the alveoli and the bronchioles gives the impression of an interstitial edema." They believe that this may be closely related to angioneurotic edema. Scott (26), Gwyn (27) and others also subscribe to the vasomotor theory.

Ball (28, 29) reported 4 cases of massive collapse diagnosed at autopsies following thyroidectomy, and advanced the theory that "atelectasis is due to the lack of expansion of the lung and interference with normal intra-alveolar pressure. There is a strong unresisted expiration and a shallow inspiration resulting in a lowering of the intra-alveolar pressure. This causes edema or diapedesis resulting in atelectasis."

Lilienthal (30), Janick (31) and others reported cases of massive collapse without obstruction. Schotz (32) thinks "the lung is a muscular organ capable of undergoing active contractions." Morison (33) believes the cause to be some type of vagal reflex.

There is some experimental support for the neurogenic mechanism. Carlson and Luckhardt (34), working on lizards, found the lungs to possess in part a crossed innervation, the vagus exercising not only a homolateral but also a contralateral control over the lungs. Stimulation of the visceral vagus resulted in contraction and tetany of the lungs. Working on turtles and frogs they showed that reflex lung contractions can be induced from stimulation of the sensory nerves of the respiratory tract and abdominal organs. In the dog and rabbit, typical bronchiolar constriction immediately follows vagal stimulation. The work of Dixon and Brodie (35) also supports the vagal reflex

theory. They showed that bronchoconstrictor fibers run only in the vagus, and reflex contractions of the bronchioles result in collapse.

De Takets, Fenn, and Jenkinson (36), primarily interested in the atelectasis associated with pulmonary embolism, demonstrated reflex bronchoconstriction in dogs. By serial roentgenologic studies they showed that the bronchial tree, visualized by the intratracheal instillation of a radiopaque medium, invariably went into spasm when pulmonary embolism was mechanically produced. This change in bronchial pattern was not the result of asphyxia since a clamp applied to the trachea failed to produce it. However, bilateral vagal section inhibited this change in all experiments, while large doses of atropine intravenously prevented its occurrence in 60 per cent of the experiments. They then showed that other stimuli constitute a sufficient stimulus for bronchoconstriction. Of various intra-abdominal manipulations, traction of the cystic duct and pulling on the mesentery gave an equally convincing bronchial pattern. This change was also inhibited by intravenous atropine, ⅙ grain (11 mg.). Blunt injury to the chest wall, with or without rib fracture, also produced bronchospasm, again prevented by the intravenous injection of atropine, ⅙ grain (11 mg.), in half the cases. "If the embolic atelectasis is originated by a nervous reflex, why could not the surgical or traumatic atelectasis be initiated by the same mechanism? Our animal experiments indicate that this is at least a possibility."

Certainly, the topographic locations of the operations that are followed by massive collapse suggest the vagus as the conduction path of the reflex in Cases 1 and 2.

Reflex vagal stimulation may also produce any of the following cardiac changes: any degree of slowing of the rate to complete stoppage with ventricular escape usually occurring after a time, dislocation of the pacemaker from the SA node downward to the AV node, partial to complete heart block, and idioventricular rhythms. Some observers believe that these cardiac changes are the direct result of a reflex vagal coronary constriction which in itself could produce cardiac changes either reflexly through the vagus, or mechanically by a decreased return to the left heart and coronary arteries, resulting in sudden peripheral vascular collapse. The acute anoxia following collapse could produce fatal ventricular fibrillation. The modus operandi of the vagal reflex in Cases 1 and 2 may have been mediated through one of two routes. The afferent impulses, the result of visceral manipulation, may have reached the central nervous system through the vagus, or by way of the spinal cord through the sympathetic afferent fibers anastomosing through the ganglions to enter the cord above the spinal anesthesic level. The impulses cannot enter the spinal cord through the somatic segment involved because the anesthetic is operative at that level. These impulses may then reflexly stimulate the vagus to produce pulmonary collapse, ventricular fibrillation and cardiac arrest, and peripheral vascular collapse. "Under ordinary conditions, the activities

of the cardiac centers are dependent to a very large extent upon the reception of impulses by afferent paths. The impulses which stream into the nervous centers arise in all parts of the body. For example, Goltz showed many years ago that reflex inhibition of the frog's heart could be induced by repeated gentle taps upon the intestines. Reflex slowing of the pulse can usually be demonstrated in the human subject by pressure upon the eyeball at the outer canthus (oculocardiac reflex), or by the stimulation of nasal branches of the fifth nerve. Stimulation of afferent fibers in the respiratory passages as by the inhalation of irritating vapors, e.g., anesthetics, is particularly likely to cause reflex inhibition of the heart. Extrasystoles and bradycardia have been demonstrated electrocardiographically in man during abdominal operations, the irregularities being the consequence, apparently, of visceral stimulation" (37). It is thought that the toxemia and infection are the precursors but that the paralysis of the lower thoracic and lumbar sympathetics contributes to an imbalance of the autonomic system, resulting in domination and hyperirritability of the parasympathetic division. The slow pulse frequently noted during spinal anesthesia demonstrates that this same imbalance occurs often, yet produces no untoward reactions unless other factors accentuate the imbalance by stimulating the parasympathetics. In Case 3 it is believed that the apprehension and emotional overactivity produced an exhaustion of the sympathetic system with considerable waste of the circulating epinephrine. The further diminution in sympathetic activity caused by the paralysis of the sympathetics by the spinal anesthetic agent produced the consequent overactivity of the parasympathetic system.

CONCLUSION

Fatal massive pulmonary atelectasis can occur as a complication of spinal anesthesia without the escape of air into the pleural space. The accentuating factors of infection, toxemia, high oxygen demand, severe emotional duress and paralysis of the lower thoracic and lumbar sympathetic nerves by spinal anesthesia produce an over-activity of the parasympathetic system particularly accentuated by vagal stimulating manipulations with consequent reflex collapse of the lungs and the circulation.

It is thought that patients exhibiting these symptoms and signs should receive a parasympathetic depressant, atropine 1 mg. to 0.6 mg. (1/60 grain to 1/100 grain), intravenously just before the administration of a spinal anesthetic agent in addition to the usual sympathomimetic drug.

We prefer to avoid spinal anesthesia in patients manifesting these signs and symptoms because the work of De Takats, Fenn, and Jenkinson (36) indicated experimentally that atropine in very high dosage cannot protect the animal, and probably man, against massive pulmonary atelectasis in more than 60 per cent of cases.

SUMMARY

Three cases of fatal massive pulmonary collapse are reported as a complication of spinal anesthesia. Experimental evidence is presented to justify the theory that an unbalanced autonomic nervous system with a hyperactivity of the parasympathetic division can cause a reflex "contraction" of the lung with consequent atelectasis and cessation of cardiac and circulatory activity. Although the use of atropine in addition to the usual parasympathomimetic drug is advocated in spinal anesthesia for patients manifesting preoperative signs and symptoms as described, it is thought that spinal anesthesia is contraindicated whenever it is suspected that a strongly unbalanced antonomic nervous system is operative.

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